20TH EVECC CONGRESS
1-3 JUNE 2023
PORTO PORTUGAL
PROCEEDINGSBOOK

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EUROPEAN VETERINARY EMERGENCY AND CRITICAL CARE CONGRESS

20TH EVECC CONGRESS
1-3 JUNE 2023
PORTO PORTUGAL

Welcome dear Delegates, Colleagues and Friends,

It is with great pleasure that we welcome you to the magnificent city of Porto for another fantastic EVECC Congress. We hope you have the opportunity to get to know the city, from the fantastic Douro River and its vineyards where the famous Port wine is produced to the beaches of the nearby city of Matosinhos, passing through fantastic Portuguese cuisine and the tiny streets full of history. If you have the opportunity, take your time to visit the famous Serralves Museum or the amazing Casa da Música or, if you are feeling strong enough, climb the Clérigos Tower and enjoy the magnificent view over the city.

In this years' edition we have some of the best speakers in the field of ECC and a variety of topics. From transfusion medicine to ventilation or CPR, this year´s EVECC has topics that appeal to everyone. With streams dedicated to nursing and research, as well as a main stream and an advanced one, the Congress aims to provide to a diverse audience an option of interest.We will have again VECCUS. A full day entirely dedicated to POCUS where an enthusiastic group of colleagues will share what is being done in this field that is evolving so rapidly and bringing so much contribution to ECC.

We hope you had the opportunity to enroll in one of our labs, we try to be more accessible each year, to more and more participants. EVECC congress would not be possible without our sponsors, please visit them in the exhibition hall and compete in the competition to win some great prizes! We also hope to see you at one of the many social events that we have at your disposal, from our welcome reception to our gala dinner, followed by an after party in a wonderful Port Wine Cellar on the bank of the Douro River. On behalf of all the members of the EVECCS Board, ECVECC Board and the Local Oganising Committee, we welcome you and hope you will have fun. If you see us walking around, come to talk to us, ask for tips about the city and, we hope that and the end of the week you can go home with great memories of the congress, the city and above all, the people.

Kind regards,

João Pedro Pereira de Araujo
Chair of the Local Organising Committee

Céline Pouzot-Nevoret
Chair of the Congress Organising Committee

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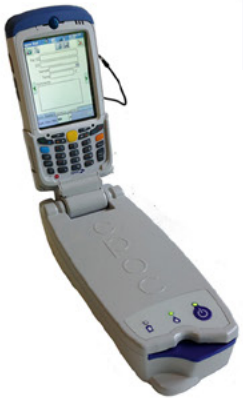

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- NT-proBNP
- Troponin I

Feline Tests:

- fPL
- T4
- TSH
- Cortisol
- Cystatin C (SDMA Alternative)
- D-Dimer
- fSAA
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- Troponin I

Equine Tests:

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VECCUS SYMPOSIUM
WEDNESDAY 31 MAY 2023

Noble hall

- **09:05-09:35 Pocus & trauma** | *Maxime Cambournac*
- **09:35-09:05 Behind the lines- to B or not to B?** | *Alexandra Nectoux*
- **10:05-10:35 GI POCUS: top tips from a diagnostic imager** | *Laura Cole & Thom Watton*
- **11:00-11:30 Point-of-care ultrasound & fluid resuscitation** | *Kris Gommeren & Søren Boysen*
- **11:30-12:00 Cardio-vascular point-of-care ultrasound in cats** | *Ivayla Yozova & Tove Hultman*
- **12:00-12:30 Point-of-care ultrasound in the pulmonary hypertension patient** | *Kris Gommeren & Dave Beeston*
- **13:30-14:30 Key-note lecture: Neurocritical POCUS - the role of transcranial doppler** | *Francisco De Carvalho Guerra Abecasis*
- **14:30-15:00 Neurocritical POCUS in veterinary patients** | *Laura Cole*
- **15:30-16:15 Strength & weakness of veterinary POCUS** | *Chris Kennedy & Joris Robben*
- **16:15-17:00 What role does social media have in POCUS education** | *Hugo Swanstein*

EVECC CONGRESS
THURSDAY 1 JUNE 2023

Main stream — Archive hall

- 09:20-10.05 **Coagulation 101** | Tom Greensmith
- 10:10-10:55 **Say no to clots: understanding antithrombotic therapy** | *Corrin Boyd*
- 11:25-12:10 **Clinical approach to immune-mediated hemolytic anemia: the role of transfusion medicine** | *Marie-Claude Blais*
- 12:15-13:00 **What a bloody mess: treating hemoabdomens** | *Meredith 't Hoen*
- 14:10-14:55 **Thoracic trauma in the ER** | *Anusha Balakrishnan*
- 15:00-15:45 **Take my breath away- a review on oxygen therapy** | *Jo-Annie Letendre*
- 16:25-17:10 **Disorders of sodium and water balance- basic pathophysiology and practical case examples** | *Marcel Aumann*
- 17:15-18:00 **Endocrine emergencies** | *Anusha Balakrishnan*

Advanced stream — Despachantes hall

- 09:20-10:05 **When sniffing around sends you to the ER- hypersensitivity pneumonitis** | *Jo-Annie Letendre*
- 10:10-10:55 **Heart lung interactions during the acute respiratory distress syndrome** | *Anusha Balakrishnan*
- 11:25-12:10 **From children to dogs and cats: what veterinary medicine can learn from assisted ventilation in pediatric patients** | *Nuno Felix*
- 12:15-13:00 **Gary stamp memorial lecture: mechanical ventilation- complex cases** | *Kate Hopper*
- 14:10-14:55 **What's new in canine blood types and pre-transfusion compatibility testing** | *Marie-Claude Blais*
- 15:00-15:45 **Update on feline blood typing and its clinical impact** | *Marie-Claude Blais*
- 16:25-17:10 **Therapeutic plasma exchange** | *Meredith 't Hoen*
- 17:15-18:00 **Cell salvage applications in emergency and critical care** | *Charlotte Russo & Tom Greensmith*

Nurse & Tech stream — Noble hall

- 09:20-10:05 **Forward thinking nursing skills** | *Amy Newfield*
- 10:10-10:55 **The septic patient; an interactive case discussion** | *Chloe Fay*
- 11:25-12:10 **Ischemia and reperfusion injury** | *Amy Newfield*
- 12:15-13:00 **Nursing the Acute Haemorrhagic Diarrhoea Syndrome (AHDS) case** | *Lindsey Ashburner*
- 14:10-14:55 **Non-technical skills for safer emergency care** | *Helen Silver-MacMahon*
- 15:00-15:45 **Low flow- provision of oxygen for patients in respiratory distress** | *Emily Gorman*
- 16:25-17:10 **Acute respiratory distress syndrome/acute lung injury** | *Amy Newfield*
- 17:15-18:00 **Gary stamp memorial lecture: mechanical ventilation 101** | *Kate Hopper*

EVECC CONGRESS
FRIDAY 2 JUNE 2023

Main stream — Archive hall

- 09:00-09:45 **Recover CPR: preparedness and Basic Life Support (BLS)** | *Daniel Fletcher*
- 09:50-10:35 **Recover CPR Advanced Life Support (ALS)** | *Daniel Fletcher*
- 11:15-12:00 **Recover CPR: newborn resuscitation** | *Daniel Fletcher*
- 12:05-12:50 **Recover CPR: post-cardiac arrest care** | *Daniel Fletcher*
- 14:00-14:45 **Blood transfusion in my practice: is it realistic?** | *Marie-Claude Blais*
- 14:50-15:35 **How to fit a mansion into a tiny house- the pop-up emergency area** | *Ivayla Yozova*
- 16:20-17:05 **Let's chit-chat about cats- five pechakucha style presentations** | *Ivayla Yozova*
- 17:10-17:55 **Hepatic lipidosis in cats: emphasis on nutritional support** | *Marie-Claude Blais*

Advanced stream — Despachantes hall

- 09:00-09:45 **Management of Canine and Feline GI motility disorders in the intensive care unit** | *Frederic Gaschen*
- 09:50-10:35 **Digestive tract assessment with POCUS: what's new in ICU?** | *Alexandra Nectoux*
- 11:15-12:00 **Gastroprotectants in critical patients** | *Frederic Gaschen*
- 12:05-12:50 **The dark side of pancreatitis- internal medicine and critical care views on the management of complications associated with acute pancreatitis** | *Marie-Claude Blais & Jo-Annie Letendre*
- 14:00-14:45 **Cardiac trauma** | *Chris Kennedy*
- 14:50-15:35 **Diastology and left atrial filling pressure** | *Chris Kennedy*
- 16:20-17:05 **Haemodynamic optimisation: asking the right questions** | *Corrin Boyd*
- 17:10-17:55 **Fluid therapy in 2023: where are we and where do we go from here?** | *Corrin Boyd*

Nurse & Tech stream — Noble hall

- 09:00-09:45 **Feline transfusion medicine: from conscious collection to xenotransfusions** | *Charlotte Russo*
- 09:50-10:35 **Death is coming; disseminated intravascular coagulation** | *Chloe Fay*
- 11:15-12:00 **Running a patient-friendly ICU** | *Corrin Boyd*
- 12:05-12:50 **Civility saves lives: why behaviour matters and how to thrive as a team** | *Helen Silver-MacMahon*
- 14:00-14:15 **Case Report: Nursing of a dog with generalised tetanus: Care of the neurologicial patient** | *Alexandra Fougeray*
- 14:15-14:30 **Case Report: Locoregional versus systemic analgesia in a dog with a severe wound: Nursing inputs in pain assessment pros-tate** | *Maria Gil Bayarri*
- 14:30-14:45 **Case Report: Nuts! The curious case of the missing prostate** | *Amanda Baldwin*
- 14:50-15:35 **I only shout at the TV when they shock asystole! Getting to grips with how and when to use your defibrillator** | *Emily Gorman*
- 16:20-17:05 **Urinary catheters made easy** | *Amy Newfield*
- 17:10-17:55 **The nurses role in the placement and management of enteral feeding tubes** | *Lindsey Ashburner*

EVECC CONGRESS
SATURDAY 3 JUNE 2023

Main stream — Archive hall

- 09:00-09:45 **Taking the septic patient to theatre (and out of it)** | *Aurora Zolf*
- 09:50-10:35 **The septic patient: a criticalist's view** | *Tom Greensmith*
- 11:15-12:00 **Plenary Session: What the future holds for Critical Care** | *Rui Paulo Jinó Moreno*
- 12:00-12:30 **Abstract award Ceremony**
- 14:10-14:55 **Pneumonia and pneumonitis** | *Simon Cook*
- 15:00-15:45 **Gary stamp memorial lecture: pyothorax: medical or surgical management** | *Kate Hopper*
- 15:50-16:35 **Upper respiratory emergencies** | *Adam Lancaster*

Advanced stream — Despachantes hall

- 09:00-09:45 **It takes a team: evidence-based communication tools for the veterinary team** | *Daniel Fletcher*
- 09:50-10:35 **Looking at medical excellence with a dual process lens** | *Rita Hanel*
- 11:15-12:00 **Plennary session: what the future holds for critical care** | *Rui Paulo Jinó Moreno*
- 12:00-12:30 **Abstract award ceremony**
- 12:30-12:45 **Research grant winner presentation & award session**
- 14:10-14:55 **Life-threatening endocrine emergencies- internal medicine and critical care views on the management of complications associated with hypoadrenocorticism** | *Marie-Claude Blais & Jo-Annie Letendre*
- 15:00-15:45 **Panel discussion: imaging in the trauma patient: why? What? When?** | *Laura Cole, Thom Watton, Anna Frykfors von Hekkel & Anusha Balakrishnan*
- 15:50-16:35 **Panel discussion: carpe diem: seizure management in the er and icu, a case-based discussion** | *Chris Mariani, Daniel FLetcher & Rita Hanel*

Nurse & Tech stream — Noble hall

- 09:00-09:45 **Multimodal analgesia** | *Ana Costa*
- 09:50-10:35 **Pain scoring the critical patient** | *Ana Costa*
- 14:10-14:55 **Invasive blood pressure measurement: curve interpretation and trouble shooting** | *Maarten Makkee*
- 15:00-15:45 **The colloid controversy** | *Amy Newfield*
- 15:50-16:35 **POCUS for haemodynamic assessment for nurses** | *Andrea Armenise*

Resident day — Infante hall

- 09:00-09:45 **Year-in-review** | *Simon Cook*
- 09:50-10:35 **Top reasons for manuscript rejection** | *Dan Chan*
- 14:10-14:55 **Inside the shocked cell** | *Corrin Boyd*
- 15:00-15:45 **Lactate: is it all hypoxia?** | *Tom Greensmith*
- 15:50-16:35 **The "Business end" of capillaries and cellular energy production- assessment of microcirculation, tissue perfusion and oxygen consumption** | *Ivayla Yozova*

ABSTRACTS

Oral abstracts, Original study | Friday 2 June — Infante hall

- 09:00-09:15 Effect of hemodialysis and hemoperfusion using cytosorb adsorber on bilirubin in dogs | *Hannah Ohrem*
- 09:20-09:35 Ionized magnesium in critically ill dogs and cats: a retrospective study | *Francesca Perini*
- 09:40-09:55 Body temperature predicts outcome in cats presenting to an emergency service: a retrospective study on 1539 cases (january 2018-december 2021) | *Alessandra Pontiero*
- 10:00-10:15 Effects of high flow oxygen therapy on oxygenation in dogs undergoing diagnostic bronchoscopy | *Julia Ortlieb*
- 10:20-10:35 The ability of oxygen reserve index to detect mild hyperoxemia in mechanically ventilated dogs: a preliminary study | *Francesca Zanusso*
- 11:15-11:30 A comparison of traditional, Fencl-Stewart approach and quantitative analysis of acid-base imbalances in dogs with dia-betic ketoacidosis | *Josep Cervera*
- 11:35-11:50 A multimodal tissue perfusion measurement approach for the evaluation of the effect of pimobendan, an inodilator, in a porcine sepsis model | *Mathieu Magnin*
- 11:55-12:10 Evaluation of serum and urinary uric acid in dogs with sepsis | *Elena Ciuffoli*
- 12:15-12:30 Clinical manifestations, laboratory findings, treatment and outcome of acute aortic thromboembolism in 202 cats: a retro-spective study | *Sarah Hefer*
- 12:35-12:50 Evaluation of modified qSOFA and news scores in emergency feline patient | *Chiara Di Franco*
- 14:20-14:35 Small animal emergency medicine practice and education in Portugal- an online survey | *Marisa Lourenço*
- 14:40-14:55 A preliminary study to investigate the prevalence and risk factors for the development of chronic kidney disease post acute kidney injury | *Laura Cole*
- 15:00-15:15 Prevalence and risk factors for malignancy and hemangiosarcoma in non-traumatic hemoabdomen in dogs | *Efrat Kelmer*
- 15:20-15:35 Retrospective observational study of acute post-operative complications occurring in the ICU following canine mitral valve repair surgery under cardiopulmonary bypass | *Christopher Ray*

Nurse and Technician Case reports | Friday 2 June 2023 — Noble hall

- 14:00-14:15 Case Report: Nursing of a dog with generalised tetanus: Care of the neurologicial patient | *Alexandra Fougeray*
- 14:15-14:30 Case Report: Locoregional versus systemic analgesia in a dog with a severe wound: Nursing inputs in pain assessment pros-tate | *Maria Gil Bayarri*
- 14:30-14:45 Case Report: Nuts! The curious case of the missing prostate | *Amanda Baldwin*

Poster Abstracts Original Study

Poster Abstracts Case Reports

POSTER ABSTRACTS

Original Study

- **Ultrasonographic signs of idiopathic lung fibrosis in west highland white terriers** | *Michal Gajewski*
- **Acid-base and electrolyte changes in dogs after storaged packed red blood cell transfusion** | *Patricia Bou*
- **A retrospective analysis of venom induced consumptive coagulopathy following eastern brown snake (pseudonaja textilis) envenomation in dogs and cats in South-East Queensland, Australia** | *Samantha Day*
- **Retrospective evaluation of autotransfusion versus allotransfusion in the perioperative management of acute hemoperitoneum in 43 dogs (2017-2021)** | *Fabienne Blunschi*
- **Retrospective study of perioperative parameters in dogs undergoing splenectomy** | *Maria Angeles Daza*
- **Retrospective review of pericardiocentesis in dogs and cats (2016-21): disease prevalence and long-term outcome** | *Martina Zarotti*
- **Viscoelastic test results in dogs with adrenal tumors** | *Ronald Gonçalves*
- **Effects of room temperature on stored canine and feline packed red blood cells quality control analysis** | *Inês Cardoso*
- **The effect of leukodepletion on canine prbc contaminated with infectious agents** | *Inês Cardoso*
- **Retrospective evaluation on the outcomes of dogs and cats with tick paralysis requiring mechanical ventilation in new South Wales, Australia** | *Morgan Woodforde*
- **Heterologous plasma segments for ocular use produced in a commercial animal blood bank - a pilot clinical study** | *Marta Flaminio*
- **Validation of a new qualitative color scale to determine the percentage of hemolysis in feline and canine packed red blood cells** | *Marta Flaminio*
- **Modeling the relationship between arterial blood pressure and sublingual microcirculation in anesthetized piglets** | *Mathieu Magnin*
- **Association between the risk of hemoparasites infection ant the blood type, gender, and breed in canine blood donors** | *Helena Ferreira*

POSTER ABSTRACTS

Case Reports

- **Pain related syndrome of inappropriate antidiuretic hormone secretion in a kitten** | *Patricia Prat*
- **Cardiac defibrillation as emergency treatment of ventricular fibrillation in a sphynx cat with hypertrophic cardiomyopathy and acute congestive heart failure** | *Ilaria Spalla*
- **Case report: percutaneous insertion of small-bore central line to treat an hepatic abscess in a dog** | *Marta Hita Rubio*
- **Pericardial effusion in two dogs with suspect eastern brown snake (pseudonaja textilis) envenomation in Queensland, Australia** | *Samantha Day*
- **Reduction of serum ibuprofen concentration in a dog with hemoadsorption using the cytosorb adsorber and therapeutic plasma exchange** | *Bettina Giani*
- **A case of hypoventilation after placement of an external splint in a cat with severe pectus excavatum** | *Ilaria Piccolo*
- **Human recombinant factor viii treatment for severe gingival hemorrhage and dental surgery in a dog with Hemophilia A** | *Efrat Kelmer*
- **Xenotransfusion of canine fresh frozen plasma and canine fresh whole blood for the treatment of feline coagulopathy** | *Iris Green*
- **Ozonized saline solution lavages in a dog with multiresistant bacterial peritonitis due to a gun shot** | *Miriam Portero*
- **Multiple organ dysfunction syndrome induced by a rupture of an unclassified large hepatobiliary cyst in a 10-month-old dog** | *Ronald Paiva Moreno Goncalves*
- **Case report: severe hemorrhage due to erosion of the inferior alveolar artery caused by severe periodontitis in 2 dogs** | *Harriet Enevoldsen*
- **Acute abdomen secondary to intestinal leiomyositis in a dog** | *Patricia Prat*
- **Thromboelastography trace in a cat with dic secondary to hemangiosarcoma** | *Carles Mengual Riera*
- **Application of fluorescent light energy (fle) therapy in a septic dog with an extensive necrotic skin lesion** | *Maria Anna Gregori*
- **Development and treatment of pyothorax after esophagostomy tube placement in a dog** | *Ana Smajlović*
- **Short-term severe polyuria responsive to vasopressin following hypoglycaemia and hypotension in a cat** | *Federica Porcarelli*
- **Emergency case of dog intoxication by the fire salamander** | *Lénio Ribeiro*
- **Broken tail syndrome' in a cat trapped in dryer machine** | *Gianila Ceccherini*

POCUS & Trauma

Maxime Cambournac
CHV Fregis, Emergency and Critical Care, Arcueil, France

Learning objectives

- Know the "normal" image
- Identify the "abnormal" image
- Understand the purpose of the POCUS in the context of trauma
- Discover the possibility of further exploration in case of trauma, with clinical-guided exam

Lecture Summary

Fast and accurate triage is crucial in the admission of critically ill patients. As there is only one health, improving the speed and accuracy of diagnostic testing represent the core of veterinary and human medicines research efforts. Point-of-care ultrasound (POCUS), defined as a goal-directed ultrasonography evaluation performed by a non-specialist to answer specific diagnostic questions, guide management, or help to perform technical procedures has been shown to achieve these goals. Often regarded as adversaries, the key difference between diagnostic imaging specialists and intensivists is that the first aim to define a diagnostic, if possible, while the second is more focused to (1) answering a specific clinical question or (2) assess a (dys)function (rather than a disease). In veterinary emergency and critical care medicine, admission of unstable patient is frequent, while concise or absent anamnestic information fairly common, and will represent a diagnostic challenge. After the initial primary survey of the patient (the well-known "ABCDE" protocol), proceed to the secondary survey, which would include a physical exam to look for signs of trauma related to the head, neck, chest, abdomen, and extremities. However, detecting internal injury or bleeding can be challenging in an unconscious or uncooperative patient. Among the question every clinician needs to answer, the most important one at this stage : "Is there any life-threatening condition?." In ECC, anticipation is the corner stone of resuscitation, as appropriate measures and therapeutics need to be set rapidly. Using ultrasound-based evaluation protocols enhance the speed of initial assessment, is available at the cageside, achievable during the resuscitation phase and reduce the exposure of ionizing radiation. POCUS in the trauma patient should aim to evaluate both cavities, thorax and abdomen. The trauma POCUS exam should initially focus on effusion, fluid or gas, around the heart (ruling out cardiac tamponade) and free fluid in cavities (internal hemorrhage) or pneumothorax. It is important to remember that the blood inside a cavity may have a variable appearance on ultrasound: from anechoic to hyperechoic. The last one being challenging to detect, as it is easily confused with the spleen.
Abdominal evaluation with POCUS in trauma requires the assessment of at least 4 views: the diaphragmaticohepatic (DH), splenorenal (SR), cystocolic (CC), and hepatorenal (HR). AFAST is performed with the patient in standing or sternal position. The umbilical view is now common in the initial assessment, as this represents a perfect spot for -centesis. Dorsal recumbency should be avoided, as it increases the risk of cardiovascular decompensation. Originally, each respective view was scored as 0 (negative all 4 views) to 4 (positive all 4 views). POCUS can detect up to 10 ml free intraperitoneal fluid (IPF) by experienced operator. In veterinary medicine, recent data suggest a sensitivity and specificity around 80 to 90%.

Thoracic POCUS

The clinical use of POCUS on thoracic assessment is recent, both in human and veterinary medicine, historically related to the idea that air was an insurmountable obstacle to obtaining usable images. Initially, the main objective of this method was the detection of pneumothorax, a significant cause of morbidity and mortality in humans. Now, the use of thoracic ultrasound is becoming a tool to assist in the diagnosis of pulmonary contusions, cardiogenic and non-cardiogenic pulmonary edema, acute respiratory distress syndromes, pneumonia, or pulmonary thromboembolism, with sensitivities and specificities close to those of CT.

Other

Many other evaluations can be done with POCUS on the trauma patient, but too long to be detail extensively in this conference. Briefly, volemic status evaluation, either by ventricular assessment on cardiac view, or vena cava assessment from different views, airway management to confirm endotracheal intubation, as well as vascular access in difficult patient are all different option for the probably medium trained clinician, in the POCUS evaluation in trauma patient. Recently, the increased use of intracranial pressure monitoring, by optic nerve sheath diameter

Conclusion

POCUS has become an integral part of emergency medicine, widely considered as the emergency clinician stethoscope. While POCUS applications are permanently expanding, veterinary clinician should remember that utilization and interpretation of such techniques requires a very little of training and experience, not to overinterpret results.

References

- *Boysen SR, Lisciandro GR. The Use of Ultrasound for Dogs and Cats in the Emergency Room AFAST and TFAST. Vet Clin North Am Small Animal Pract. 2013;43(4):773–797. doi:10.1016/j.cvsm.2013.03.011*
- *Smallwood N, Dachsel M. Point-of-care ultrasound (POCUS): unnecessary gadgetry or evidence-based medicine? Clin Med. 2018;18(3):219–224. doi:10.7861/clinmedicine.18-3-219*

Behind the lines - to B or not to B?

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Learning objectives

- Understand the physical theory behind ultrasound artifacts
- Define criteria that differentiate A, B, Z, T, C and E lines
- List pulmonary disease that can induce ultrasonographic artifacts

Lecture Summary

Lung ultrasound is widely used in emergency and critical care medicine and has become a useful tool to diagnose respiratory disorders and improve their management. Interpretation of lung ultrasound images should always be made in the light of medical context, physical examination or other biological analysis. A B-line is define as a vertical ultrasonographic artefact originating from pleural line, hyperechoic, extending to the depth of the image without decreasing in intensity, moving synchronously with lung sliding and erasing A-lines. This comet tail are thought to be the reflection of the ultrasound beam from a lung with increased density back to the transducer. Increased lung density is caused by replacement of alveolar air by liquid (blood, transudate, exudate or collagen). Comparison between ultrasound and computed tomography images showed that B-lines were correlated to the thickening of subpleural interlobular septa in pulmonary interstitial edema and to the fibrotic thickening in pulmonary fibrosis. Normal lung ultrasound examination may showe no to very few B-lines. Three or more B-lines seen on two or more regions of the thorax is named lung interstitial syndrome. In 2012, a human consensus statement formulated definitions and clinical interpretation of B-lines. In people, several others identified factors affecting B-lines visualization and quantification such as transducer type, depth penetration, the use of spatial compound imaging, gain, time of examination, focal position, extra-pulmonary disease or patient's position. Kameda et al. developed an acoustic model and showed that vertical artifacts as B-lines length and intensity could varied with composition of the sources of the artifact.

Other lines

More than B-lines, other artifacts shorter, brighter, narrow or wide can be seen. Z-lines are also vertical artifacts originating from pleural line but they do not extend to the bottom of the image, do not erase A lines and are not perfectly synchronized with respiration. Change of ultrasound frequency could convert some B-lines to Z-lines. Physical mechanism behind those lines is not fully understand but the theory of acoustic trap of ultrasound beams in channels is suspected. This conversion often coexisted with pleural line abnormalities, suggesting micro-consolidations. T-lines are vertical non-echoic lines extending from the pleural line to the bottom of the image due to small movement of the pleural layers synchronously with cardiac pulsations. E-lines are vertical hyperechoic lines that do not arise from pleural line but from the subcutaneous tissue and do not move synchronously with respiration. They are seen in subcutaneous emphysema. C-lines are broad hyperechoic lines originated from a thickened irregular hypoechoic pleural line that could be seen in pneumonia.

Other lung disease

B-lines are defined as arising from the pleural line. Some others suggest that B-line artifact are only seen in cardiogenic pulmonary edema whereas comet-tail artifact (CTA) could be seen in parenchymal lung diseases. Differentiation of these artifacts would be based on analysis of pleural line, regular in B-lines and irregular in CTA. A shredded line between aerated and consolidated lung and also be seen as the origin of artifacts in consolidated lung parenchyma. Interpretation of thoracic ultrasonographic artifacts should be done after optimization of the image via ultrasound settings, knowledge of clinical history and examination findings, quantification of artifacts and analysis of images around the artifact.

References

- *Buda N, Skoczylas A, Demi M, Wojteczek A, Cylwik J, Soldati G. Clinical Impact of Vertical Artifacts Changing with Frequency in Lung Ultrasound. Diagnostics (Basel). 2021 Feb 26;11(3):401.*
- *Dietrich CF, Mathis G, Blaivas M, Volpicelli G, Seibel A, Wastl D, Atkinson NS, Cui XW, Fan M, Yi D. Lung B-line artefacts and their use. J Thorac Dis. 2016 Jun;8(6):1356-65.*
- *Duggan NM, Goldsmith AJ, Saud AAA, Ma IWY, Shokoohi H, Liteplo AS. Optimizing Lung Ultrasound: The Effect of Depth, Gain and Focal Position on Sonographic B-Lines. Ultrasound Med Biol. 2022 Aug;48(8):1509-1517.*
- *Francisco MJ Neto, Rahal A Junior, Vieira FA, Silva PS, Funari MB. Advances in lung ultrasound. Einstein (Sao Paulo). 2016 Jul-Sep;14(3):443-448.*
- *Kameda T, Kamiyama N, Taniguchi N. Simple Experimental Models for Elucidating the Mechanism Underlying Vertical Artifacts in Lung Ultrasound: Tools for Revisiting B-Lines. Ultrasound Med Biol. 2021 Dec;47(12):3543-3555.*
- *Kameda T, Kamiyama N, Taniguchi N. The Mechanisms Underlying Vertical Artifacts in Lung Ultrasound and Their Proper Utilization for the Evaluation of Cardiogenic Pulmonary Edema. Diagnostics (Basel). 2022 Jan 20;12(2):252.*
- *Mathis G, Horn R, Morf S, Prosch H, Rovida S, Soldati G, Hoffmann B, Blaivas M, Dietrich CF. WFUMB position paper on reverberation artefacts in lung ultrasound: B-lines or comet-tails? Med Ultrason. 2021 Feb 18;23(1):70-73.*
- *Soldati G, Demi M, Inchingolo R, Smargiassi A, Demi L. On the Physical Basis of Pulmonary Sonographic Interstitial Syndrome. J Ultrasound Med. 2016 Oct;35(10):2075-86.*
- *Volpicelli G, Elbarbary M, Blaivas M, Lichtenstein DA, Mathis G, Kirkpatrick AW, Melniker L, Gargani L, Noble VE, Via G, Dean A, Tsung JW, Soldati G, Copetti R, Bouhemad B, Reissig A, Agricola E, Rouby JJ, Arbelot C, Liteplo A, Sargsyan A, Silva F, Hoppmann R, Breitkreutz R, Seibel A, Neri L, Storti E, Petrovic T; International Liaison Committee on Lung Ultrasound (ILC-LUS) for International Consensus Conference on Lung Ultrasound (ICC-LUS). International evidence-based recommendations for point-of-care lung ultrasound. Intensive Care Med. 2012 Apr;38(4):577-91.*

I PACUS: top tips from a diagnostic imager

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Learning objectives

- Recognize important gastrointestinal landmarks on abdominal POCUS.
- Identify ultrasonographic findings compatible with an intestinal obstruction.
- Appraise POCUS findings in the context of the clinical history and physical examination.
- Formulate a plan based on clinical history, physical examination and POCUS findings.

Lecture Summary

This lecture is a case-based co-led lecture in the format of an emergency specialist interviewing a specialist diagnostic imager – the focus will be on key tips and tricks for gastrointestinal POCUS, particularly identifying findings supportive of a surgical condition. The lecture will review important anatomical landmarks including the pylorus, duodenum and ileocaecocolic junction, as well as tips for probe placement, differentiating small and large intestine and identifying accompanying abdominal findings such as pneumoperitoneum to increase the clinicians index of suspicion for surgical pathology.

References

- *Garcia D.A., Froes T.R., Vilani R.G., Guérios S.D., Obladen A. Ultrasonography of small intestinal obstructions: a contemporary approach, Journal of Small Animal Practice; 2011,52(9);484-90.*
- *Kim S.Y., Park K T., Yeon S.C., Lee H.C. Accuracy of sonographic diagnosis of pneumoperitoneum using the enhanced peritoneal stripe sign in Beagle dogs, Journal of Veterinary Science; 2014, 15(2);195–198.*
- *Nyland T.G., Mattoon J.S. Small Animal Diagnostic Ultrasound, Elsevier, Saunders; 2020*
- *Penninck D.G., d'Anjou M.,Mellor B. Atlas of small animal ultrasonography, Wiley-Blackwell; 2008.*
- *Sanderson J.J., Boysen S.R., McMurray J.M., Lee A., Stillion J.R. The effect of fasting on gastrointestinal motility in healthy dogs as assessed by sonography, Journal of Veterinary Emergency and Critical Care, 2017; 27(6):645-50.*
- *Boysen S.R., Gambino J.M. POCUS: Gastrointestinal Tract and Pancreas, Point-of-Care Ultrasound Techniques for the Small Animal Practitioner, 2021;1:225-44.*

Point-of-Care ultrasound & fluid resuscitation

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Learning objectives

- Fluid resuscitation is beneficial in many patients that present to the emergency service, yet it is important to avoid hypo-and hypervolemia, as they are associated with significant morbidity and mortality.
- Fluid resuscitation can be guided by Point-of-care ultrasound (POCUS) by rapidly answering different binary response questions of the cardiovascular system, as well as signs of left- and right-sided congestion.
- Cardiac POCUS relative wall and lumen size
- Caudal vena cava (and other vessel) size and (respiratory) variation
- Lung Ultrasound appearance of B lines and/or pleural effusion
- Abdominal ultrasound gall bladder wall edema and/or appearance of ascites

Lecture Summary

The ROSE principle, developed in human medicine, subscribes the importance of rapid and aggressive fluid resuscitation, without omitting the relevance of optimization, stabilization and evacuation, in order to avoid complications secondary to overzealous fluid administration.

Although fixed volumes over specified times are typically recommended in the literature to provide fluid resuscitation to companion animals, the volumes used by clinicians are typically quite variable. This wide variation is explained by regional preferences, personal experience, the clinical scenario (underlying condition), and the patient (individual perfusion characteristics, estimated intravascular volume, comorbidities, species, age, body condition score, hematocrit, total proteins, renal parameters, acid-base and electrolyte analysis).

Point-of-Care Ultrasound (POCUS) can facilitate aggressive fluid resuscitation, allowing initial and serial assessment of specific sonographic findings during therapy. POCUS is typically based on closed ended questions, using limited or fixed views, to assess predetermined parameters. In the scenario of fluid resuscitation these closed ended questions include:

Does the patient have sonographic signs of hypovolemia?

Focal cardiac ultrasound. On both longitudinal 4-chamber and the transverse short-axis views, the clinician assesses the relative thickness of the chamber walls and septum, as well as the left ventricular and atrial lumen sizes. If walls appear to be thick, and the lumen appears small, hypovolemia should be suspected. If the left atrium seems small compared to the diameter of the aorta, again, hypovolemia should be suspected. During resuscitation, these parameters can be tracked. When the walls appear to become thin and stretched, and the lumen appears to be large, particularly when the left ventricular contractility look hyperkinetic, hypervolemia should be considered. Similarly, whenever the left atrial size becomes roughly double the size of the aorta, hypervolemia should be considered.

Caudal vena cava size and collapsibility: The CVC can be assessed at different levels, with the 2 most often cited techniques being the subxiphoid view and the paralumbar views. At the subxiphoid view the collapsibility is more easily assessed, whilst the paralumbar view offers the advantage of comparing the size of the CVC to that of the aorta. A small caudal vena cava is suggestive of hypovolemia. Moreover, if the CVC collapses completely during the respiratory cycle, this once more suggests hypovolemia. This parameter can also be tracked during the resuscitation phase, and whenever the CVC becomes "fat" and hardly changes in size throughout the respiratory cycle, the patient is very likely to be unresponsive to further fluid administration and may already be hypervolemic.

Does the patient display signs of decreased air in the pulmonary parenchyma? For this question, the clinician should perform lung ultrasound, for which different techniques have been described. The following applies as long as a single technique is used consistently: when a patient develops B lines, or the amount of B lines increases during the resuscitation phase, this may be suggestive of left sided congestion and/or

Does the patient have sonographically detectable free cavitary effusion? Abdominal POCUS using the 4 or 5 point technique as described in the original FAST paper remains the recommended technique to look for free abdominal fluid. In order to detect even small amounts of free pleural effusion, the authors recommend the use of the PLUS-protocol to specifically look for small amounts at the gravity dependent pleural spaces.

Whenever a patient develops free pleural or abdominal fluid (or in rare, mostly feline, cases pericardial effusion) during resuscitation, it suggests right sided congestion and/or

Does the patient display gall bladder wall edema? The gallbladder is assessed at the level of the subxiphoid view. Whenever a halo sign of the gall bladder wall is sonographically observed, it suggests right sided congestion and and/or hypervolemia.

All POCUS findings should however be interpreted carefully, integrating all clinically relevant information, such as signalment, history, clinical findings and results of complementary examinations. As examples, septicemia or anaphylaxis may also induce gall bladder wall edema; a road traffic accident may lead to contusions within the first hours after the injury.

In summary, POCUS uses a multimodal approach to help guide fluid therapy, rapidly assessing multiple organs and structures, and is best used in a serial manner to track changes over time.

References

- *Cambournac M, Goy-Thollot I, Violé A, et al: Sonographic assessment of volaemia: development and validation of a new method in dogs. J Small Anim Pract 59(3):174-182, 2018.*
- *Darnis E, Boysen S, Merveille AC, et al: Establishment of reference values of the caudal vena cava by fast-ultrasonography through different views in healthy dogs. J Vet Intern Med 2018. 32(4):1308-1318. 2018.*
- *Herreria-Bustillo VJ, Fitzgerald E, Humm KR: Caval-aortic ratio and caudal vena cava diameter in dogs before and after blood donation. J Vet Emerg Crit Care 29(6):643-646, 2019.*
- *Lichtenstein D: Fluid administration limited by lung sonography: the place of lung ultrasound in assessment of acute circulatory failure (the FALLS-protocol). Expert Rev Respir Med 6(2):155-62, 2012.*
- *Malbrain ML, Marik PE, Witters I, et al: Fluid overload, de-resuscitation, and outcomes in critically ill or injured patients: a systematic review with suggestions for clinical practice. Anaesthesiol Intensive Ther 46(5):361-80, , 2014.*
- *Marshall KA, Thomovsky EJ, Brooks AC. Ultrasound measurements of the caudal vena cava before and after blood donation in 9 greyhound dogs. Can Vet J 59(9):973-980. 2018.*
- *Nelson NC, Drost WT, Lerche P, Bonagura JD, et al: Noninvasive estimation of central venous pressure in anesthetized dogs by measurement of hepatic venous blood flow velocity and abdominal venous diameter. Vet Radiol Ultrasound, 51(3):313-23, 2010.*
- *Oricco S, Rabozzi R, Meneghini C, Franci P: Usefulness of focused cardiac ultrasonography for predicting fluid responsiveness in conscious, spontaneously breathing dogs. Am J Vet Res 80(4): 369-377, 2019.*
- *Rabozzi R, Oricco S, Meneghiniet C, et al: Evaluation of the caudal vena cava diameter to abdominal aortic diameter ratio and the caudal vena cava respiratory collapsibility for predicting fluid responsiveness in a heterogeneous population of hospitalized conscious dogs. J Vet Med Sci, 82(3):337-344, 2020.*
- *Silverstein DC, Aldrich J, Haskins SC, et al: Assessment of changes in blood volume in response to resuscitative fluid administration in dogs. J Vet Emerg Crit Care 15(3):185-192, 2005.*

Cardiovascular point-of-care ultrasound in cats

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Learning objectives

- Revise the POCUS modalities used for cardiovascular assessment in cats
- State the presumed causes for cats being prone to intravascular volume overload and fluid overload
- Understand the advantages and limitations of cardio-vascular POCUS modalities in cats
- Give examples for indications to use cardiovascular POCUS in cats

Lecture Summary

Ultrasonographic cardiovascular assessment has become a necessity in the emergency room and intensive care unit. Main indications for cardiovascular assessment in sick cats include the detection of asymptomatic heart disease, aid in diagnosis of elusive hypovolemia and circulatory shock and monitoring response to treatment and last but not least, diagnosing a monitoring of intravascular volume overload (IVVO) and fluid overload (FO) whether present at admission of developing the course of hospitalization. In the hands of the skilled point-of-care sonographer, these can be further expanded to detection of spontaneous echogenic contrast, aortic thrombi, pulmonary hypertension, and aid during CPR, among others.

Focused cardiac ultrasound

FCU or FOCUS has proven to be a valuable tool in the hands of non-specialist practitioners. A recent study in 343 apparently healthy cats demonstrated that after short-term training, non-specialist practitioners could diagnose 93.1% and 100% of moderate and severe asymptomatic cardiomyopathies in screened cats. The study used a semi-quantitative assessment of common cardiac indices, such as left atrium-to-aorta (LA:Ao) ratio, inter-ventricular septal thickness at diastole, and fractional shortening. The images were recorded from two relatively easily obtainable views - the right parasternal short axis heart base ("Mercedes- Benz") and "mushroom" views. Since prevalence of asymptomatic HCM is relatively high, ~ 15% in various populations, being able to detect it may influence therapeutic plans especially in regards of IV fluids administration and transfusions. Previous studies in dyspnoeic cats have demonstrated high sensitivity and specificity of left atrial (LA) enlargement in diagnosis of congestive heart failure (CHF). Echocardiography has been used to document intravascular volume overload in severely anaemic cats in two studies, including screening for transfusion-associated volume overload. Some evidence suggests that the administration of corticosteroids in otherwise healthy cats may lead to apparent IVVO. "Pseudohypertrophy" of the left ventricle has been demonstrated in experimentally induced hypovolemia in heathy cats. Indeed, the left ventricle can have a thickened appearance during hypovolemic states, associated usually with a normal or decreased LA: Ao and left ventricular underfilling. Whether normal or abnormal, FOCUS findings can guide veterinary practitioners in many common clinical scenarios, ranging from stabilizing hypovolemic cats, dealing with cats in respiratory distress, screening cats undergoing procedures at risk for decompensating in CHF or simply daily monitoring of volume status of hospitalized cats. The reader is referred to a recent review article on use of FCU in cats from Kerry Loughran, VCSA 2021 for further details.

The caudal vena cava

CVC is a major vessel carrying up to 80% of venous return, that can be viewed from multiple windows relevant to point-of-care ultrasound (subxiphoid view, hepatic view, paralumbar views). From these, several parameters relating to its absolute diameter (at inspiration and expiration), diameter relative to the aorta, and collapsibility (percentage change in diameter within the respiratory cycle) can be quantified or subjectively evaluated. The diameter of the CVC is expected to decrease in hypovolemic states and increase, during IVVO from naturally occurring disease (CHF, acute kidney injury) or iatrogenic. These changes can be assessed as absolute compared to reference intervals, or relative to the aorta. The aortic diameter remains unchanged during "dysvolaemias" and the aorta and CVC run in parallel in the paralumbar view, making assessment relatively easy. The CVC diameter is affected by the phase of the respiratory cycle, especially when measurements are obtained in the sites closer to the thorax (such as the subxiphoid view). It decreases with inspiration, owning to the increase in negative intra-thoracic pressure and blood being "pooled into the lungs" and normalizes during expiration. From this difference in diameters, a collapsibility index (CVC-CI) can be obtained as a percentage using the following equation: CVC-CI = $(CVC_{expiration} - CVC_{inspiration})/CVC_{expiration} \times 100$.
Ultrasonographically-derived inferior vena cava (IVC) indices are widely used in people to assess intravascular volume status, including prediction of fluid responsiveness and monitoring effects of haemodialysis. As any other tool, they are not without limitations, owing to unstandardized techniques and location of measurements, operator variability, concomitant diseases affecting measurements. Caudal vena cava parameters, obtained from various views, have been reported in dogs, foals, calves, and cats, with reference intervals established in dogs and, recently, cats (abstract). In a study evaluating healthy cats undergoing blood donation, changes in CVC diameter were noted before and after blood donation and after administration of a fluid bolus. A study with experimentally induced hypovolemia and IVVO, expiratory CVC diameter was more accurate in detecting IVVO, when compared to inspiratory diameter and CVC-CI (abstract). A more recent study evaluating fluid responsiveness in 24 hypovolemic cats demonstrated a difference in both inspiratory and expiratory CVC diameters and CI between responders and non-responders with non-responders having larger CVC diameters and lower CI. Some of these studies report significant inter-rater variability and some difficulty obtaining images. The CVC-CI has a wide reference interval (2 – 59 %) in cats, which challenges its usefulness in this species. This is probably due to the relatively small CVC diameters, especially inspiratory (RI 1.4 – 6.1 mm), which makes measurement errors possible. Despite these technical challenges, the CVC can be used for assessment of volume status in cats, especially combined with cardiac indices and other tools. Rather than accurate measurements, a more semi-quantitative approach might be more appropriate for cats. Ultimately, the expiratory CVC diameter might prove the more reliable parameter to use in this species.

Pleural space and lung ultrasound

PLUS should accompany every cardio-vascular POCUS in cats. Internal hemorrhage or third space losses, manifesting as pleural effusion, can be the cause for hypovolemia, while IVVO and FO can result in development pulmonary oedema (manifesting ultrasonographically as b-lines) and pleural effusions. While a pleural effusion is always and abnormal finding in cats, a discrete number of b-lines can be found in healthy cats. Indeed, depending on the protocol used, 12-51% healthy cats have been found to have < 3 b-lines in at least one site. Development of b-lines and their diagnostic value has been mostly studied in the context of respiratory distress and CHF in cats with the presence of multiple b-lines being ~ 80% sensitive and specific for CHF. In people the association of IVVO and FO with development of b-lines has been well established. Studies in cats are, however lacking. Since cats are relatively prompt to IVVO and FO, due to their relatively small blood volume, slow elimination of IV fluids, asymptomatic HCM and are frequently presented anemic, a baseline PLUS exam at admission, followed by serial exams throughout hospitalization is warranted. As b-lines are not specific for pulmonary oedema and merely a sign of decreased aerated to non-aerated lung ratio (or wet lung), the presence of b-lines should be interpreted in context with the underlying condition. In conclusion POCUS offers additional modalities to help manage "dysvolaemias" in cats, whether by screening for asymptomatic cardiomyopathies in anticipation of complications and assessment of actual intra-vascular volume for diagnostic and monitoring purposes. Both hypovolemia and IVVO can be detrimental, however the latter is easier to miss. Therefore, POCUS has a specific value in being one of the few clinically available diagnostic tools for IVVO. While some of those modalities come with technical challenges and need further validation, their use is becoming wider with more research to improve the robustness of the methodology underway.

References

- *Armenise A, Boysen RS, Rudloff E, et al. Veterinary-focused assessment with sonography for trauma-airway, breathing, circulation, disability and exposure: a prospective observational study in 64 canine trauma patients. J Small Anim Pract 2019;60:173-182.*
- *Cambournac M, Goy-Thollot I, Viole A, et al. Sonographic assessment of volaemia: development and validation of a new method in dogs. J Small Anim Pract 2018;59:174-182.*
- *Casalta H, Busoni V, Eppe J, et al. Ultrasonographical Assessment of Caudal Vena Cava Size through Different Views in Healthy Calves: A Pilot Study. Vet Sci 2022;9.*
- *Darnis E, Boysen S, Merveille AC, et al. Establishment of reference values of the caudal vena cava by fast-ultrasonography through different views in healthy dogs. J Vet Intern Med 2018;32:1308-1318.*
- *Del Prete C, Freccero F, Lanci A, et al. Transabdominal ultrasonographic measurement of caudal vena cava to aorta derived ratios in clinically healthy neonatal foals. Vet Med Sci 2021;7:1451-1459.*
- *Donati PA, Tunesi M, Araos J. Caudal vena cava measurements and fluid responsiveness in hospitalized cats with compromised hemodynamics and tissue hypoperfusion. J Vet Emerg Crit Care (San Antonio) 2023;33:29-37.*
- *Hultman TM, Boysen SR, Owen R, et al. Ultrasonographically derived caudal vena cava parameters acquired in a standing position and lateral recumbency in healthy, lightly sedated cats: a pilot study. J Feline Med Surg 2022;24:1039-1045.*
- *Marshall KA, Thomovsky EJ, Brooks AC, et al. Ultrasound measurements of the caudal vena cava before and after blood donation in 9 greyhound dogs. Can Vet J 2018;59:973-980.*
- *Sänger F, Dorsch R, Hartmann K, et al. Ultrasonographic assessment of the caudal vena cava diameter in cats during blood donation. J Feline Med Surg 2022;24:484-492.*
- *Tuplin MC, Romero AE, Boysen SR. Influence of the Respiratory Cycle on Caudal Vena Cava Diameter Measured by Sonography in Healthy Foals: A Pilot Study. J Vet Intern Med 2017;31:1556-1562.*
- *Jalava S HT, Boysen SR, Yozova ID. Effects of hypovolemia and volume overload on ultrasonographically derived caudal vena cava parameters in healthy lightly sedated cats. In: Abstracts from the International Veterinary Emergency and Critical Care Symposium, the European Veterinary Emergency and Critical Care Annual Congress, and the ACVECC VetCOT Veterinary Trauma & Critical Care Conference 2022 Journal of Veterinary Emergency and Critical Care 2022;32:S27-S28.*
- *Hultman TM JS, Boysen SR, Yozova ID. Establishment of reference intervals for ultrasonographically derived caudal vena cava parameters in healthy lightly sedated cats. In: Abstracts from the International Veterinary Emergency and Critical Care Symposium, the European Veterinary Emergency and Critical Care Annual Congress, and the ACVECC VetCOT Veterinary Trauma & Critical Care Conference 2022. Journal of Veterinary Emergency and Critical Care 2022;32:S34-S35.*
- *Campbell FE, Kittleson MD. The effect of hydration status on the echocardiographic measurements of normal cats. J Vet Intern Med 2007;21:1008-1015.*
- *Duler L, Scollan KF, LeBlanc NL. Left atrial size and volume in cats with primary cardiomyopathy with and without congestive heart failure. J Vet Cardiol 2019;24:36-47.*
- *Goutal CM, Keir I, Kenney S, et al. Evaluation of acute congestive heart failure in dogs and cats: 145 cases (2007-2008). J Vet Emerg Crit Care (San Antonio) 2010;20:330-337.*
- *Groom AC, Rowlands S. The cardiac output and blood volume of the anaesthetized cat. Phys Med Biol 1958;3:138-156.*
- *Loughran K. Focused Cardiac Ultrasonography in Cats. Vet Clin North Am Small Anim Pract 2021;51:1183-1202.*
- *Loughran KA, Rush JE, Rozanski EA, et al. The use of focused cardiac ultrasound to screen for occult heart disease in asymptomatic cats. J Vet Intern Med 2019;33:1892-1901.*
- *Payne JR, Brodbelt DC, Luis Fuentes V. Cardiomyopathy prevalence in 780 apparently healthy cats in rehoming centres (the CatScan study). J Vet Cardiol 2015;17 Suppl 1:S244-257.*
- *Ward JL, Lisciandro GR, DeFrancesco TC. Distribution of alveolar-interstitial syndrome in dogs and cats with respiratory distress as assessed by lung ultrasound versus thoracic radiographs. J Vet Emerg Crit Care (San Antonio) 2018;28:415-428.*
- *Ward JL, Lisciandro GR, Ware WA, et al. Evaluation of point-of-care thoracic ultrasound and NT-proBNP for the diagnosis of congestive heart failure in cats with respiratory distress. J Vet Intern Med 2018;32:1530-1540.*
- *Letendre JA RM, Boysen SR, Masseau IM. Evaluation of B-lines with 2 point-of-care lung ultrasound protocols in cats with radiographically normal lungs. In: Abstracts from the International Veterinary Emergency and Critical Care Symposium, the European Veterinary Emergency and Critical Care Annual Congress, and the ACVECC VetCOT Veterinary Trauma & Critical Care Conference 2022. Journal of Veterinary Emergency and Critical Care 2022;32:S28-S29.*
- *Lisciandro GR, Fosgate GT, Fulton RM. Frequency and number of ultrasound lung rockets (B-lines) using a regionally based lung ultrasound examination named vet BLUE (veterinary bedside lung ultrasound exam) in dogs with radiographically normal lung findings. Vet Radiol Ultrasound 2014;55:315-322.*
- *Lisciandro GR, Lisciandro SC. Lung Ultrasound Fundamentals, "Wet Versus Dry" Lung, Signs of Consolidation in Dogs and Cats. Vet Clin North Am Small Anim Pract 2021;51:1125-1140.*
- *Szymczak J, Kiełbowicz Z, Kinda W, et al. Transthoracic lung and pleura ultrasonography as a diagnostic tool of pulmonary edema in dogs and cats. Pol J Vet Sci 2018;21:475-481.*

Point-of-care ultrasound in the pulmonary hypertension patient

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Learning objectives

- Recall the underlying pathophysiology of pulmonary hypertension
- Understand the techniques for assessment of pulmonary hypertension and their limitations
- Review the point-of-care ultrasound findings associated with moderate to severe pulmonary hypertension
- Apply the above knowledge to a real-life clinical case

Lecture Summary

Pathophysiology of Pulmonary Hypertension

Pulmonary hypertension (PH) is defined by abnormally increased pressure within the pulmonary vasculature caused by a variety of respiratory, cardiovascular and systemic diseases.[1] Patients can present with signs of syncope, cyanosis or respiratory distress at rest, exercise intolerance and signs of right-sided congestive heart failure (R-CHF).[1] As patients can present with severe respiratory distress, ruling out severe PH in respiratory compromise is an important step in emergency room evaluation and stabilization. PH can loosely be categorized as pre- or post-capillary based on absence or presence of increased pulmonary arterial wedge pressure (PAWP) and elevated LA pressures, respectively.[1] The ACVIM consensus statement guidelines on canine pulmonary hypertension classifies patients into 6 discrete categories:

-Primary pulmonary arterial hypertension
-PH secondary to left heart disease
-PH secondary to respiratory disease and/or hypoxia
-PH secondary to thromboembolic disease
-PH secondary to parasitic disease
-Multifactorial PH

The gold standard for PH diagnosis (right-heart catheterization) is rarely available or practiced in veterinary medicine.[1] Assessment of PH often relies on estimation of pulmonary arterial pressure (PAP) and right ventricular changes via Doppler echocardiographic assessment.[2,3] PH is categorized as mild, moderate or severe. Prompt treatment of pulmonary hypertension is indicated in moderate or severe cases. For all cases, identification of the underlying cause is recommended.

Use of point-of-care ultrasound in Pulmonary Hypertension

Point-of-care ultrasound (POCUS) can be immensely helpful in approaching the patient with suspected pulmonary hypertension. Whilst POCUS signs of R-CHF alone have failed to differentiate patients with and without moderate to severe PH,[4] a recently published 10-point scoring system for pre-capillary PH adopted a grey-zone approach to rule in (scores >= 5) or rule out (scores < 2) severe PH with >90% accuracy.[5] The scoring system assessed right atrial (RA) and right ventricular (RV) enlargement, RV hypertrophy, flattening of the interventricular septum (IVS), PA enlargement and signs of R-CHF (presence of abdominal effusion and a distended, non-compliant CaVC).[5] Combining the 10-point scoring system with traditional thoracic ultrasound may aid the clinician in choosing the appropriate diagnostic pathway for the classification of pulmonary hypertension.

Future research

Additional simple measurements such as the eccentricity index (EI), assessed in the right parasternal short axis view, may help the clinician rapidly recognize the PH patient. The EI evaluates the shape of the left ventricle and IVS in either systole or diastole, providing rapid quantitative assessment of PH, as it has been shown to correlate with severity of PH.[7] Future studies should aim to validate both the EI and the 10-point scoring system using images acquired by emergency and critical care clinicians.

References

- *Reinero C, Visser LC, Kellihan HB, et al. ACVIM consensus statement guidelines for the diagnosis, classification, treatment, and monitoring of pulmonary hypertension in dogs. J Vet Intern Med 2020; 34: 549–573.*
- *Vezzosi T, Domenech O, Costa G, et al. Echocardiographic evaluation of the right ventricular dimension and systolic function in dogs with pulmonary hypertension. J Vet Intern Med 2018; 32: 1541–1548.*
- *Visser LC, Wood JE, Johnson LR. Survival characteristics and prognostic importance of echocardiographic measurements of right heart size and function in dogs with pulmonary hypertension. J Vet Intern Med 2020; 34: 1379–1388.*
- *Vientós-Plotts AI, Wiggen KE, Lisciandro GR, et al. The utility of point-of-care ultrasound right-sided cardiac markers as a screening test for moderate to severe pulmonary hypertension in dogs. Vet J 2019; 250: 6–13.*
- *Lyssens A, Lekane M, Gommeren K, et al. Focused Cardiac Ultrasound to Detect Pre-capillary Pulmonary Hypertension. Front Vet Sci 2022; 9: 80.*
- *Klabunde RE. Cardiovascular Physiology Concepts. 3rd ed. Wolters Kluwer Health, https://www.cvphysiology.com/textbook-3 (2020, accessed 31 December 2022).*
- *Lekane M, Merveille AC, Gommeren K, et al. Left Ventricular Eccentricity Index for Asessment of Precapillary Pulmonary Hypertension in Dogs. In: ECVIM-CA Online Congress. 2021.*

Neurocritical POCUS - the role of transcranial doppler

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Learning objectives

- To understand the basic principles of TCD
- To learn how TCD can be used in the context of paediatric neurocritical care
- Relationship between human and animal TCD use

Lecture Summary

Transcranial Doppler (TCD) has been used for more than 30 years in clinical practice. Although adult intensive care is relatively well covered, pediatric cases are still underrepresented. In this presentation I intend to review the basic principles of TCD and how it can be used in pediatric neurocritical care.
To better accomplish this objective a series of pediatric cases where TCD was determinant in clinical decisions will be analyzed.
I will describe cases with different pathologies where TCD had an important role in clinical management of the patients and discuss TCD utility and potential role both in the emergency department and the intensive care unit.
Five patients with different neurologic insults will be presented. TCD was useful in the identification of intracranial hypertension in traumatic brain injury, hydrocephalus and central nervous system infection; identification of decreased cerebral perfusion pressure in hypovolemic shock and the diagnosis of impending cerebral circulatory arrest in a child with meningococcal septicemia.
A review of the literature relevant for this topic will be briefly presented.
I will then talk about the relationship between animal and human experiments and how they can help one another.
I believe non-invasive testing using TCD can aid clinical decisions and that more widespread use of this technique will allow for better care of patients with neurologic insults. I hope this technique will also improve neurocritical care in the veterinary setting.

Neurocritical POCUS in veterinary patients

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Learning objectives

- Recall different POCUS techniques for the assessment of intracranial pressure in small animal patients
- Describe the technique for measurement of optic nerve sheath diameter
- Understand the limitations of measuring the optic nerve sheath diameter in small animal patients
- Interpret the optic nerve sheath diameter in the context of the clinical history and physical examination

Lecture summary

This lecture is an overview of the current available veterinary literature of POCUS techniques for assessment of intracranial hypertension. Published techniques including measurement of the optic nerve sheath diameter and transcranial doppler will be summarized. Clinical application of the techniques will be discussed, discussing top tips and common pitfalls before considering how to interpret the measurement in the context of the clinical history and physical examination findings. This lecture will be interactive. It will encourage audience participation in sharing their experiences, comments, and question.

References

- *Drolet C., Pinard C., Gaitero L., Monteith G., Bateman S., Study of the effect of anaesthesia on the canine ultrasonographic optic nerve sheath diameter, Journal of Small Animal Practice; 2021,62(12);1070-1078.*
- *Sasaoka K., Nakamura K., Osuga T., Morita T., Yokoyama N., Morishita K., Sasaki N., Ohta H., Takiguchi M. Transcranial Doppler ultrasound examination in dogs with suspected intracranial hypertension caused by neurologic diseases, Journal of Veterinary Internal Medicine; 2018, 32(1);314-23.*
- *Smith J.J., Fletcher D.J., Cooley S.D., Thompson M.S. Transpalpebral ultrasonographic measurement of the optic nerve sheath diameter in healthy dogs, Journal of Veterinary Emergency and Critical Care; 2018, 28(1);31-8.*
- *Lee H.C., Choi H.J., Choi M.C., Yoon J.H. Ultrasonographic measurement of optic nerve sheath diameter in normal dogs, Journal of Veterinary Science; 2003,4(3);265-8.*
- *Cho J. Ocular Ultrasound Abnormalities and Optic Nerve Sheath Diameter in Dogs and Cats, Veterinary Clinics: Small Animal Practice; 2021, 51(6):1295-314.*
- *Vitiello L., De Bernardo M., Capasso L., Cornetta P., Rosa N. Optic Nerve Ultrasound Evaluation in Animals and Normal Subjects, Frontiers in Medicine (Lausanne); 2022, 8;797018.*
- *Evangelisti M.A., Carta G., Burrai G.P. Repeatability of ultrasound examination of the optic nerve sheath diameter in the adult cat: comparison between healthy cats and cats suffering from presumed intracranial hypertension. Journal of Feline Medicine and Surgery; 2020, 22(10);959-965.*

Strength & weakness of veterinary POCUS

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Learning objectives

• To discuss POCUS and its use and abuse in veterinary medicine

Lecture Summary

Since its introduction in small animal emergency medicine almost two decades ago by S. Boysen et al.[1], point-of-care ultrasonography, POCUS, has become an important and integrated part of the diagnostic work-up process. As with every new asset to our extensive diagnostic toolbox, a critical appraisal is now-and-again necessary to evaluate its use and suggested advantages. This helps us determine how it can be used now and to develop its use in the future. Does POCUS, as it is performed now, deserve its reputation? Has it become an indispensable diagnostic tool to be used in almost every emergency patient? Or is its reputation inflated beyond its ability to deliver results? Worse, is POCUS potentially misguiding, leading to false affirmations, false paradigms and inappropriate treatments? In this session, Joris and Chris will highlight pros and cons of POCUS.

References

• *Boysen SR, Rozanski EA, Tidwell ES, Holm JL, Shaw SP, Rush JE. Evaluation of a focused assessment with sonography for trauma protocol to detect free abdominal fluid in dogs involved in motor vehicle accidents. J Am Vet Med Assoc. 2004;225(8):1198-204. doi: 10.2460/javma.2004.225.1198*

Coagulation 101

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Learning objectives

- Understand the different clinical presentations of disturbances in primary and secondary haemostatic disorders, and those of the fibrinolytic cascade
- Recognize common diseases that may affect the three haemostatic systems, and outline a rational approach to their treatment
- Discuss clinical 'in-clinic' testing of primary and secondary haemostasis
- Understand why the clotting cascade fails to fully explain clinical haemorrhage (or the lack thereof) depending on the factors involved
- Understand that 'true' in vivo coagulation system is significantly more complex, but that a more basic approach still provides an extremely useful scaffold for case-based clinical decision making

Lecture summary

Introduction: Derangements in different areas of the haemostatic system may present with different clinical signs. These can help the clinician to narrow down the affected aspect of coagulation, which further aids in appropriate diagnostic testing, differentials and treatments. The three main aspects of haemostasis are primary and secondary haemostasis followed by fibrinolysis. Disorders may affect one or multiple areas, and patients can be in both pro-thrombotic and haemorrhagic states simultaneously (such as in disseminated intravascular coagulation). The coagulation system is a fine balancing act of pro- and anti-thrombotic factors, as well as pro- and anti-fibrinolytic factors.

Haemostatic systems: Primary haemostasis involves the interactions of platelets with each other and the vessel wall, along with a milieux of other molecules including von Willebrands factor. Disorders in this system often lead to multiple sites of small haemorrhage, often at mucosal surfaces. Secondary haemostasis involves the sequential activation of factors within the clotting cascade culminating in a stable fibrin plug at the site of primary haemostatic activity, with disorders of this system often represented by large bleeds from a single site into potential spaces (such as body cavities and joints). Despite their name, both primary and secondary haemostasis occur simultaneously. The clotting cascade as we know it was devised 'in vitro' and fails to account for the contribution of any molecules other than those found in plasma, and for this reason fails to explain why diseases such as factor XII deficiency is not typically associated with clinical bleeding. A newer model (the cell-based model) is described and explains the interplay between cells (such as platelets, white blood cells and endothelial cells) and their contribution to primary and secondary haemostasis, providing a more thorough (although significantly more complex) understanding of the in vivo physiology of haemostasis. Fibrinolysis is the process by which clots are broken down and the patency restored to vessels, insufficient fibrinolysis may lead to endothelial obstruction and perfusion defects, while excessive fibrinolysis can result in cleavage of some soluble clotting factors as well as insoluble clots resulting in excessive bleeding.

Clinical signs: As noted previously, primary haemostatic disorders often involve bleeding from mucosal surfaces such as gingival haemorrhage, haematuria and hyphema. Bleeding into the skin from primary haemostatic disorders occurs as petechiae, ecchymoses or the combination of both (purpura). Secondary haemostastic dysfunction may commonly present with conditions such as haemothorax, haemoabdomen, haemopericardium and haemarthrosis. Epistaxis can occur with either primary or secondary haemostatic dysfunction, as can melena (although the latter is most common in primary disturbances). Dermal bleeding for secondary haemostatic disorders commonly present as haematomas. Fibrinolytic disorders can be more varied in their presentation but should be considered empirically in sighthounds, the critically ill and severely traumatised patients. Excessive fibrinolysis may present as sudden worsening of haemorrhage after initial stabilisation following trauma or surgery.

Testing: In clinic testing of coagulation is often limited to tests such as platelet count, buccal mucosal bleeding time (both used to assess primary haemostasis), prothrombin time (PT) and activated partial thromboplastin time ((a)PTT) (used to assess secondary haemostasis). Thus it can be incredibly difficult to assess platelet function, von Willebrand factor contribution and the subtle nuances of fibrinolytic disturbances without external laboratory testing. However, these tests still provide an extremely useful first line assessment when combined with understanding of the physiology involved and the test's limitations.

References

- *Hackner, S.G., Rousseau, A., Chapter 105 Bleeding Disorders, Small animal critical care medicine 2nd Ed., St. Louis, USA, Elsevier Saunders; 2015;554-566.*
- *Smith, S.A., The cell-based model of coagulation, J Vet Emer Crit Care, 2009,19(1);3-10.*

Say no to clots: understanding antithrombotic therapy

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Learning objectives

- Discuss the advantages and disadvantages of common antiplatelet and anticoagulant treatments
- Prescribe rational evidence-based antithrombotic therapy for common conditions

Lecture summary

The physiology of coagulation is complex. However, it can be simplified as a delicate balance between procoagulant and anticoagulant forces. The competing risks of imbalance are haemorrhage and thrombosis. Thrombosis risk increases with any of the three elements of Virchow's triad: static or turbulent blood flow, endothelial injury or activation, and hypercoagulability of the blood.

Thrombosis can cause devastating clinical consequences due to obstruction to blood flow, with resultant tissue ischaemia, congestion, or even obstructive shock. The clinical manifestations of thrombosis are dependent of the location of the thrombus. Broadly, this can be divided into arterial and venous thrombosis. Clinically relevant arterial thrombosis most commonly occurs at the aortic trifurcation, but can also manifest in other limb arteries, as a cerebrovascular accident, a myocardial infarction, or infarction of another organ. The manifestations of venous thrombosis likewise depend upon the location. The most acutely devastating is embolism of a systemic venous thrombus to the pulmonary circulation: a pulmonary thromboembolism. Other common manifestations of venous thrombosis include vena cava thrombosis and thrombosis of the portal system.

Medications aimed at preventing thrombosis are broadly termed antithrombotic agents. These are two broad categories: antiplatelet agents and anticoagulants. Historically, the major antiplatelet agent employed in small animal medicine was aspirin. However, due to unpredictable pharmacokinetics and pharmacodynamics, it is no longer recommended as a first line. Inhibitors of the ADP receptor $P2Y_{12}$, such as clopidogrel, are now considered standard of care. Anticoagulants inhibit the function of coagulation factors. They include the heparins (unfractionated and low molecular weight), vitamin K antagonists such as warfarin, and a newer class of drugs called direct oral anticoagulants (DOACs). Heparins are heavily utilized in hospitalised small animals. However, the need for frequent injection limits their utility after hospital discharge. Historically, the only oral anticoagulant option was vitamin K antagonists such as warfarin. Due to highly unpredictable pharmacokinetics, the need for frequent monitoring, and the high risk of haemorrhage, these agents are not recommended. The newer DOACs such as rivaroxaban are thought to be more predictable in their dose-response relationship and carry a lower risk of haemorrhage. Thus, their usage in veterinary medicine is greatly expanding.

Choosing whether to administer an antiplatelet agent, an anticoagulant, or both is challenging. A broad simplification is that arterial thrombi are platelet-rich, while venous thrombi are platelet-poor. Thus, if the primary risk is of arterial thrombosis, an antiplatelet agent is commonly the first line, whilst anticoagulants are first line for preventing venous thrombosis. However, in conditions where there is risk of both arterial and venous thrombosis, or the overall thrombosis risk is considered very high, it is reasonable to administer an anticoagulant and antiplatelet agent together.

It is challenging for the small animal clinician to know when thromboprophylaxis, the administration of antithrombotic agents to prevent thrombosis, is indicated. For this reason, the American College of Veterinary Emergency and Critical Care created the Consensus on the Rational Use of Antithrombotics in Veterinary Critical Care (CURATIVE) guidelines. These guidelines discuss conditions associated with a high risk of thrombosis, such as immune mediated haemolytic anaemia, protein losing nephropathy, and feline cardiomyopathy, as well as those associated with an intermediate thrombosis risk. Many recommendations about antithrombotic choice, dosing, monitoring, and discontinuation are also discussed.

References

- *Blais, M.C., Bianco, D., Goggs, R., Lynch, A.M., Palmer, L., Ralph, A. and Sharp, C.R., 2019. Consensus on the rational use of antithrombotics in veterinary critical care (CURATIVE): domain 3-defining antithrombotic protocols. Journal of Veterinary Emergency and Critical Care, 29(1), pp.60-74.*
- *Brainard, B.M., Buriko, Y., Good, J., Ralph, A.G. and Rozanski, E.A., 2019. Consensus on the Rational Use of Antithrombotics in Veterinary Critical Care (CURATIVE): Domain 5-Discontinuation of anticoagulant therapy in small animals. Journal of Veterinary Emergency and Critical Care, 29(1), pp.88-97.*
- *deLaforcade, A., Bacek, L., Blais, M.C., Goggs, R., Lynch, A. and Rozanski, E., 2019. Consensus on the rational use of antithrombotics in veterinary critical care (CURATIVE): domain 1-defining populations at risk. Journal of Veterinary Emergency and Critical Care, 29(1), pp.37-48.*
- *Goggs, R., Bacek, L., Bianco, D., Koenigshof, A. and Li, R.H., 2019. Consensus on the rational use of antithrombotics in veterinary critical care (CURATIVE): domain 2-defining rational therapeutic usage. Journal of Veterinary Emergency and Critical Care, 29(1), pp.49-59.*
- *Goggs, R., Blais, M.C., Brainard, B.M., Chan, D.L., deLaforcade, A.M., Rozanski, E. and Sharp, C.R., 2019. American College of Veterinary Emergency and Critical Care (ACVECC) consensus on the rational use of antithrombotics in veterinary critical care (CURATIVE) guidelines: small animal. Journal of Veterinary Emergency and Critical Care, 29(1), pp.12-36.*
- *Sharp, C.R., deLaforcade, A.M., Koenigshof, A.M., Lynch, A.M. and Thomason, J.M., 2019. Consensus on the rational use of antithrombotics in veterinary critical care (CURATIVE): domain 4-refining and monitoring antithrombotic therapies. Journal of veterinary emergency and critical care, 29(1), pp.75-87.*
- *Sharp, C.R., Goggs, R., Blais, M.C., Brainard, B.M., Chan, D.L., deLaforcade, A.M. and Rozanski, E., 2019. Clinical application of the American College of Veterinary Emergency and Critical Care (ACVECC) Consensus on the Rational Use of Antithrombotics in Veterinary Critical Care (CURATIVE) guidelines to small animal cases. Journal of Veterinary Emergency and Critical Care, 29(2), pp.121-131.*
- *Sharp, C.R., Goggs, R., Blais, M.C., Brainard, B.M., Chan, D.L., deLaforcade, A.M. and Rozanski, E., 2019. Clinical application of the American College of Veterinary Emergency and Critical Care (ACVECC) Consensus on the Rational Use of Antithrombotics in Veterinary Critical Care (CURATIVE) guidelines to small animal cases. Journal of Veterinary Emergency and Critical Care, 29(2), pp.121-131.*

Clinical approach to immune-mediated hemolytic anemia: the role of transfusion medicine

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Learning objectives

- Recognize an immune-mediated hemolytic anemia (IMHA) in a canine anemic patient and exclude possible diagnostic differentials.
- Understand the limitation of blood compatibility testing in severely anemic patients and/or in dogs with persistent auto-agglutination
- Describe the principle behind treatment and follow-up of canine patients with IMHA, including corticosteroids and other immunosuppressive agents as well as antithrombotic therapy.

Lecture summary

Immune-mediated haemolytic anemia (IMHA) is one of the most common causes of anaemia in dogs, yet treatment regimens remain non-standardized and, in some cases, controversial. IMHA may present as an idiopathic event but may be triggered/associated with a variety of infectious diseases, neoplasia, drugs, vaccines, or inflammatory processes. Recently, a diagnosis algorithm was proposed including criteria such as moderate to severe anaemia, evidence of immune-mediated destruction (e.g. spherocytes, positive saline test and/or Coomb's test or flow cytometry) and haemolysis (hyperbilirubinemia, hemoglobinemia, erythrocyte ghost).[1]

History and clinical presentation

Predisposed breeds include English Spaniels, Poodles, Irish Setters and Collies, with American Cocker Spaniel representing nearly a third of idiopathic IMHA cases. Clinical signs parallel the severity and progression of the anaemia, ranging from lethargy/exercise intolerance to collapse. Physical examination typically reveals pale mucous membranes, tachypnea, systolic heart murmur, splenomegaly, hepatomegaly, icterus, pigmenturia, fever and lymphadenopathy. Petechiae, ecchymosis and melena may be present (if severe thrombocytopenia).

Diagnostic

To evaluate the level of anaemia, a spun PCV is suggested because calculated haematocrit may be unreliable when agglutination is present.[1] Typically, patients with IMHA have a moderate to severe, highly regenerative, anaemia, although a third of dogs may present with poorly regenerative anaemia. Spherocytosis is present in 89-95% of cases of IMHA; although it is not considered pathognomonic for IMHA, marked spherocytosis is certainly very suggestive of the disease. Neutrophilic leukocytosis with a left shift is also frequently encountered. Approximately 50-70% of dogs with IMHA have concomitant thrombocytopenia caused by Evans syndrome and/or DIC. A blood smear may also allow identification of infectious agents. A positive saline test is reported in 50-90% of cases and is associated with a higher mortality rate. The sensitivity of the Coombs test is reported to be between 60-90%; therefore, a negative Coombs test does not exclude a diagnosis of IMHA. Flow cytometry is an interesting alternative (sensitivity 67-100%, specificity of 87.5%).[1] Because of the risk of DIC and thromboembolisms, coagulation times (PT and PTT) and evaluation of fibrin degradation products, d-dimer, thromboelastography can help with diagnosis.

Treatment

1- Blood transfusion

About 70-90% of IMHAs will require one or several blood transfusions. Packed RBC, ideally no older than 7-10 days, are preferred given the normovolemic state of the anaemia.[3] Increasing age of pRBC was associated with increased risk of mortality in dogs with haemolysis (90% had IMHA) and of haemolytic transfusion reaction.[4,5] The decision to transfuse should ultimately be based on the patients clinical signs (e.g. tachypnoea, tachycardia and weakness), but most dogs with a PCV < 15% will require blood transfusion.

2- Immunosuppressive therapy

Glucocorticoids remain the cornerstone of IMHA therapy. Their mode of action is to reduce RBC destruction by inhibiting phagocytosis of antibody-coated RBCs and reducing the production of cytokines and immunoglobulins. Unless the side effects are unacceptable, the glucocorticoid dose should not be decreased until the patient's PCV has stabilized close to normal values, with improvement in disease activity indices (e.g. spherocytosis, agglutination, serum bilirubin concentration and reticulocyte count). Unfortunately, glucocorticoids are associated with numerous side effects that can frustrate the owners and compromise the quality of life of the patient: polyuria / excessive polydipsia, almost obsessive polyphagia, incontinence, and excessive panting. Additional immunosuppressive agents should be administered if glucocorticoids alone fail to induce remission, cause significant side effects, or fail to control IMHA unless given consistently at high doses. At the onset of IMHA, they are usually reserved for severe cases: presence of marked persistent autoagglutination, intravascular haemolysis, or non-regenerative anaemia. The most used immunosuppressive agents in dogs are azathioprine, cyclosporine and mycophenolate molfetil.[6,7] Cyclophosphamide, in combination with prednisone, was associated with decrease therapeutic success. Leflunomide, human intravenous immunoglobulin, splenectomy and plasmapheresis have been used in refractory cases with variable success rates.

3- Antithrombotic drugs

All dogs with IMHA, except those with severe thrombocytopenia, should receive some form of antithrombotic.[3,8] The ideal drug(s) and protocol are unknown: low dose aspirin (30% dogs with "aspirin resistance"), clopidogrel, heparin, low molecular weight heparin or rivaroxaban are all options to consider.[9, 10]

Prognosis: The Canine Haemolytic Anaemia Objective Score (CHAOS), which was recently re-evaluated, was positively associated with mortality during hospitalisation and at 30 days; similarly ASA classification, bilirubin, urea and creatinine were independently associated with death in hospital or by 30-days.[13]

References

- *Garden OA, Kidd L, Mexas AM, Chang YM, Jeffery U, Blois SL, et al. ACVIM consensus statement on the diagnosis of immune-mediated hemolytic anemia in dogs and cats. Journal of veterinary internal medicine / American College of Veterinary Internal Medicine. 2019.*
- *Schaefer DM, Stokol T. Retrospective study of reticulocyte indices as indicators of iron-restricted erythropoiesis in dogs with immune-mediated hemolytic anemia. Journal of veterinary diagnostic investigation : official publication of the American Association of Veterinary Laboratory Diagnosticians, Inc. 2016.*
- *Swann JW, Garden OA, Fellman CL, Glanemann B, Goggs R, LeVine DN, et al. ACVIM consensus statement on the treatment of immune-mediated hemolytic anemia in dogs. Journal of veterinary internal medicine / American College of Veterinary Internal Medicine. 2019.*
- *Maglaras CH, Koenig A, Bedard DL, Brainard BM. Retrospective evaluation of the effect of red blood cell product age on occurrence of acute transfusion-related complications in dogs: 210 cases (2010-2012). Journal of veterinary emergency and critical care (San Antonio, Tex : 2001). 2017;27(1):108-20.*
- *Hann L, Brown DC, King LG, Callan MB. Effect of duration of packed red blood cell storage on morbidity and mortality in dogs after transfusion: 3,095 cases (2001-2010). Journal of veterinary internal medicine / American College of Veterinary Internal Medicine. 2014;28(6):1830-7.*
- *Wang A, Smith JR, Creevy KE. Treatment of canine idiopathic immune-mediated haemolytic anaemia with mycophenolate mofetil and glucocorticoids: 30 cases (2007 to 2011). The Journal of small animal practice. 2013;54(8):399-404.*
- *West LD, Hart JR. Treatment of idiopathic immune-mediated hemolytic anemia with mycophenolate mofetil in five dogs. Journal of veterinary emergency and critical care (San Antonio, Tex : 2001). 2014;24(2):226-31. 8. deLaforcade A, Bacek L, Blais MC, Goggs R, Lynch A, Rozanski E. Consensus on the Rational Use of Antithrombotics in Veterinary Critical Care (CURATIVE): Domain 1-Defining populations at risk. Journal of veterinary emergency and critical care (San Antonio, Tex : 2001). 2019;29(1):37-48.*
- *Goggs R, Blais MC, Brainard BM, Chan DL, deLaforcade AM, Rozanski E, et al. American College of Veterinary Emergency and Critical Care (ACVECC) Consensus on the Rational Use of Antithrombotics in Veterinary Critical Care (CURATIVE) guidelines: Small animal. Journal of veterinary emergency and critical care (San Antonio, Tex : 2001). 2019;29(1):12-36.*
- *Blais MC, Bianco D, Goggs R, Lynch AM, Palmer L, Ralph A, et al. Consensus on the Rational Use of Antithrombotics in Veterinary Critical Care (CURATIVE): Domain 3-Defining antithrombotic protocols. Journal of veterinary emergency and critical care (San Antonio, Tex : 2001). 2019;29(1):60-74.*
- *Weinkle TK, Center SA, Randolph JF, Warner KL, Barr SC, Erb HN. Evaluation of prognostic factors, survival rates, and treatment protocols for immune-mediated hemolytic anemia in dogs: 151 cases (1993-2002). Journal of the American Veterinary Medical Association. 2005;226(11):1869-80.*
- *Swann JW, Skelly BJ. Evaluation of immunosuppressive regimens for immune-mediated haemolytic anaemia: a retrospective study of 42 dogs. The Journal of small animal practice. 2011;52(7):353-8.*
- *Goggs R, Dennis SG, Di Bella A, Humm KR, McLauchlan G, Mooney C, et al. Predicting Outcome in dogs with Primary Immuně-Mediated Hemolytic Anemia: Results of a Multicenter Case Registry. Journal of veterinary internal medicine / American College of Veterinary Internal Medicine. 2015;29(6):1603-10.*

What a bloody mess: treating hemoabdomens

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Learning objectives

- Know the aetiologies of haemoabdomen
- Identify patients with haemoabdomen based on history and clinical signs
- Understand aspects of stabilizing patients with haemoabdomen including blood product administration
- Understand indications for surgical intervention

Lecture summary

Haemoabdomen, also known as hemoperitoneum, occurs when free haemorrhage is present in the peritoneal cavity. Causes of haemoabdomen include traumatic and nontraumatic aetiologies. As the name implies, traumatic haemoabdomen occurs following blunt or penetrating trauma. Nontraumatic haemoabdomen (NTH) occurs secondary to a variety of conditions, including coagulopathy, anaphylaxis, splenic or liver torsion, or masses (benign or neoplastic). The spleen and liver are the most common location of masses, although other organs such as the adrenal gland and kidney can be affected. Hemangiosarcoma is the most common neoplastic cause, but neoplasias such as lymphoma and other sarcomas are also reported.

Historical information provided by the owner may vary by underlying cause, including recent trauma or exposure to anticoagulant rodenticides. Regardless of aetiology, signs related to blood loss such as weakness, collapse, lethargy, pale gums, and abdominal distension may be reported. Physical examination may reveal pale mucous membranes, tachycardia, arrythmias, weak pulses, abdominal masses and a fluid wave. Emphasis should be placed on identifying signs of shock to initiate therapy in a timely manner.

In patients presenting with signs of shock, emergent diagnostic efforts should focus on assessing perfusion and cardiovascular stability, as well as confirming the presence of a haemobadomen and potentially the cause. Point-of-care bloodwork (acid-base status, lactate, electrolytes, PCV/TS, glucose), coagulation testing, ECG, and point-of-care ultrasound should be priorities. Confirmation of peritoneal effusion should prompt abdominocentesis. PCV of the abdominal fluid should be compared to that of peripheral blood; haemorrhagic effusion will have a PCV of at least 10-25% of peripheral PCV.

Resuscitation efforts should be aimed at restoring perfusion regardless of the underlying cause. Many patients (72% in one study) experiencing blunt trauma are polytrauma patients, so emphasis should be placed on global evaluation and therapy. Judicious use of isotonic crystalloids (10-20ml/kg boluses) with frequent reassessment should be initiated. Limited fluid volume resuscitation likely has an important role in resuscitation and includes hypertonic saline administration. For patients with evidence of ongoing haemorrhage or persistent cardiovascular instability, blood product administration is recommended. Recent evidence supports fresh whole blood administration. Component therapy using fresh frozen plasma (FFP) and packed red blood cells (pRBCs) with a ratio of 1:1 may also be considered but fails to replace platelets. Autotransfusion can be lifesaving when financial or blood product resources are limited. Most patients with traumatic haemoabdomen can be managed medically, but surgery should be considered if stabilisation can't be achieved with medical management. For patients with NTH, stabilisation is recommended prior to anaesthesia for exploratory laparotomy. Ultimately, exploratory surgery to remove the source of bleeding and obtain samples for histopathology is necessary. Specific therapy for patients suspected of anticoagulant rodenticide toxicity should include vitamin K, FFP, and potentially pRBC administration.

Due to the implications for prognosis, differentiating between malignant and benign processes early in the course of NTH would be ideal. Unfortunately, due to the limitations of the diagnostic tests available, histopathology remains the gold standard for determining the underlying aetiology. Variable incidences of neoplastic vs. benign lesions are reported with a recent systematic review of dogs with NTH due to a splenic mass reporting 73% of masses as malignant, whereas 27% were benign. The majority of malignant lesions were haemangiosarcoma (87%). The canine hemangiosarcoma likelihood prediction (HeLP) score was recently developed to predict the risk of diagnosis of hemangiosarcoma in dogs with NTH. Four predictors (body weight, total plasma protein, platelet count and thoracic radiograph findings) are used to aid identification of patients at low, medium or high risk for haemangiosarcoma.
In one retrospective study of patients with a traumatic haemoabdomen, 75% of patients managed medically survived, and 67% of those managed surgically survived. In another study of blunt trauma patients, presence of a haemoabdomen was not associated with mortality and the overall survival rate in that population of dogs was 88%.

References

- *Hnatusko AL, Gicking JC, Lisciandro GR. Anaphylaxis-related hemoperitoneum in 11 dogs. J Vet Emerg Crit Care (San Antonio). 2021 Jan;31(1):80-85.*
- *Pintar J, et al. Acute nontraumatic hemoabdomen in the dog: a retrospective analysis of 39 cases (1987-2001). J Am Anim Hosp Assoc. 2003 Nov-Dec;39(6):518-22.*
- *Herold LV, et al. Clinical evaluation and management of hemoperitoneum in dogs. J Vet Emerg Crit Care (San Antonio). 2008. 18 (1): 40-53.*
- *Hammond TN, Holm JL, Sharp CR. A pilot comparison of limited versus large fluid volume resuscitation in canine spontaneous hemoperitoneum. J Am Anim Hosp Assoc. 2014 May-Jun;50(3):159-66.*
- *Schick AR, Grimes JA. Evaluation of the validity of the double two-thirds rule for diagnosing hemangiosarcoma in dogs with nontraumatic hemoperitoneum due to a ruptured splenic mass: a systematic review. J Am Vet Med Assoc. 2023. 261(1): 69-73.*
- *Schick AR, et al. Development and validation of a hemangiosarcoma likelihood prediction model in dogs presenting with spontaneous hemoabdomen: The HeLP score. J Vet Emerg Crit Care (San Antonio). 2019. 29(3):239-245.*

Thoracic trauma

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Learning objectives

- General approach to the traumatised patient in the ER focusing on global triage evaluation
- Focus on emergency stabilization in the ER with recognition of thoracic injuries using point of care testing such as POCUS.
- Specific focus on recognition and stabilization of pneumothorax, hemothorax, rib fractures with flail chest, diaphragmatic hernia and pulmonary contusions.
- Emphasis on owner communication for short term management of patients with these conditions and considerations for definitive management.

Lecture summary

Thoracic trauma in dogs and cats is common and can be a source of morbidity and mortality in these patients. These injuries can range in severity from mild, such as superficial abrasions, to life-threatening, such as tension pneumothorax and damage to the chest wall, vessels and thoracic viscera.

Rib fractures and flail chest: These are a common finding in dogs with thoracic trauma and can be clinical markers for more severe underlying thoracic injury and a source of significant pain. A further complication of traumatic rib fractures is flail chest. In these patients, the intrinsic costal arch support of a section of the thoracic wall is lost due to multiple fractures of at least two adjacent ribs. The section of rib then tends to move asynchronously with the normal motion of the thorax during respiration. The diagnosis of a flail chest is established most readily by observing the paradoxical motion of the affected segment in a spontaneously breathing patient. Upon inspiration, the flail segment is pulled inward by the negative intrathoracic pressure. With exhalation, the positive pressure forces the segment to protrude outward. In one veterinary study evaluating flail chest in 24 canine and feline patients, the median number of fractured ribs was three. Flail chest and multiple rib fractures are often markers for more severe thoracic injuries such as pulmonary contusions.

Pneumothorax & Pneumomediastinum: Traumatic pneumothorax is a common occurrence, and tension pneumothorax, in particular, can be a rapidly life-threatening complication of thoracic trauma. Pneumothorax with blunt force trauma can be caused by laceration of lung parenchyma by a fractured rib segment, or tearing of the lung from shear injury caused by trauma or from rapid changes in airway pressure. With penetrating trauma, pneumothorax can result from penetration of the thoracic wall by foreign objects or teeth (in case of bite wounds) causing an open pneumothorax. A tension pneumothorax may result from leakage of air into the pleural space with no outlet for escape of air, causing a one-way valve effect. This progressive build-up of air increases intra-thoracic pressure, causing collapse of the great vessels within the thorax and ultimately cardiovascular collapse.

Diagnosis of a traumatic pneumothorax is typically made based on clinical signs of respiratory distress, short, shallow breathing, and auscultation of dull, muffled lung sounds in the dorsal aspect of the thorax on physical examination. Other bedside tools that permit rapid diagnosis of a pneumothorax include point-of-care ultrasound examination (POCUS).

Pulmonary contusions: Pulmonary contusions are common with thoracic trauma and should be anticipated in any patient sustaining a high energy blunt chest impact. Lung contusions represent a concussive loss of vessel integrity resulting in intra-parenchymal and alveolar haemorrhage, oedema, decreased pulmonary compliance, and increased shunt fraction, with a resultant decrease in gas exchange and subsequent hypoxemia.

It is important to note that while rib fractures and pulmonary contusions often occur together, the absence of rib fractures does not rule out the possibility of pulmonary contusions. Patients with severe contusions can often present with significant respiratory distress, with airway crackles occasionally heard upon auscultation. The POCUS examination can also be used to detect the presence of pulmonary oedema and haemorrhage. Bright, hyper echoic lines known as B-lines that extend from the pulmonary-pleural interface to the far field and oscillate like a pendulum with inspiration and expiration can be observed, and indicate abnormalities within the pulmonary parenchyma. Most often, pulmonary contusions are often not readily apparent radiographically in the early stages after trauma, and can be delayed for up to 6-12 hours. Management of pulmonary contusions is typically with supportive care including oxygen supplementation and sedation as needed. In patients with severe respiratory compromise and hypoxaemia from pulmonary contusions, mechanical ventilation is sometimes necessary for 1-3 days to improve oxygenation and provide ventilatory support as the contusions resolve.

Diaphragmatic hernia: This is a common sequela to blunt abdominal trauma wherein a sudden increase in intra-abdominal pressure results in disruption of the diaphragm and cranial displacement of abdominal viscera into the thorax. The herniation of abdominal contents and associated haemorrhage and oedema into the pleural space can result in decreased ability of the lungs to inflate, and cause tachypnoea and dyspnoea in these patients. Diagnosis of traumatic diaphragmatic hernia can be made based on a combination of clinical suspicion, auscultation (muffled lung sounds, or occasionally gut sounds heard within the thorax) and thoracic imaging such as radiography or CT scan. Stabilization of these patients involves oxygen supplementation and sedation prior to surgical intervention for diaphragmatic repair.

References

- *Frykfors von Hekkel AK, Pegram C, Halfacree ZJ. Thoracic dog bite wounds in dogs: A retrospective study of 123 cases (2003-2016). Veterinary Surgery. 2020 May;49(4):694-703.*
- *Sigrist NE, Doherr MG, Spreng DE. Clinical findings and diagnostic value of post-traumatic thoracic radiographs in dogs and cats with blunt trauma. J Vet Emerg Crit Care 2004; 14(4): 259-268*
- *Streeter EM, Rozanski EA, deLarforcade A, et al. Evaluation of vehicular trauma in dogs. Am Vet Med Assoc 2009; 235:405-408)*
- *Dicker SA, Lisciandro GR, Newell SM, Johnson JA. Diagnosis of pulmonary contusions with point-of-care lung ultrasonography and thoracic radiography compared to thoracic computed tomography in dogs with motor vehicle trauma: 29 cases (2017-2018). Journal of Veterinary Emergency and Critical Care. 2020 Nov;30(6):638-46.*

Take my breath away - a review on oxygen therapy

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Learning objectives

- Recognize when a patient would benefit from oxygen therapy
- List non-invasive, conventional oxygen delivery methods
- Recognize when non-invasive, conventional oxygen delivery methods are not sufficient and should be escalated to high-flow nasal oxygen therapy or mechanical ventilation
- For each oxygen delivery methods discussed, identify the indications, contra-indications, and possible complications

Lecture summary

Oxygen therapy, a treatment frequently used in veterinary medicine, is indicated before any diagnostic test or examination is attempted in the presence of signs of respiratory distress. Clinical signs and physical examination findings suggesting respiratory distress include increased respiratory rate, crackles, wheezes, harsh or muffled lung sounds, increased efforts, exaggerated/abnormal movement of the chest/abdomen, orthopnea, collapse, cyanosis, and open mouth breathing in cats. When in doubt, it is best to provide supplemental oxygen until information about the patient's respiratory status is available. Pulse oximetry and arterial blood gas can be used to obtain quantitative evaluation of the respiratory function. Oxygen supplementation is indicated if the SpO_2 is <95% and/or if PaO_2 is <80 mmHg. It is important to limit the amount of time the patient receives high concentrations of oxygen, because oxygen toxicity can develop when a FiO_2 of >60% is maintained for >24 hours. Oxygen can be supplied from various sources and in many ways. This will be the focus of this presentation.

Non-invasive, conventional oxygen delivery methods: Flow-by oxygen involves providing oxygen by holding the oxygen tube near the animal's nose/mouth. The achievable FiO_2 is 25-40%. One person should hold the oxygen in front of the animal. This technique is therefore useful mainly during procedures such as intravenous catheterization, X-rays, and ultrasound. This method is easy and accessible. However, it leads to a waste of oxygen, as high flow rates must be used (6-8 L/min) and is not always well tolerated by the patients. A mask can be attached to the oxygen source to provide a higher FiO_2 (35-60%). Since one person usually must hold the mask in front of the patient, it is usually used as a temporary first-line method during a procedure. It is important to ensure that the mask is not too tight to avoid CO_2 rebreathing and hyperthermia.

An oxygen hood can be made by using an oxygen source at a flow rate of 0.5-2 L/min, an Elizabethan collar and plastic wrap. It is important to leave an opening to allow evacuation of CO_2 and heat. With this method, a FiO_2 of 30-40% can be achieved. This method is not tolerated by all patients.

Various commercial cages are available, offering different functions to control O_2, CO_2, humidity, temperature, etc. Home-made oxygen cages can also be created with an oxygen source, an incubator/pet transporter, plastic wrap, etc. Oxygen cages are usually low stress for the patient and can provide FiO_2 levels close to 100% (most are limited to a FiO_2 of 60%). However, whenever we want to access the patient for examination or treatment, oxygen supplementation must be stopped or greatly reduced, thus limiting access and care of the patient while in the oxygen cage. Oxygen supplementation can be provided with nasal prongs or nasal cannulas. When nasal oxygen is chosen, it is important to use a humidifier to avoid drying of the nasal mucosa and associated discomfort. Nasal prongs provide a FiO_2 similar to the flow-by oxygen technique. Nasal cannula can be placed in one or both nostrils. The tip of the cannulas can reach the caudal nasal cavity or the nasopharynx. Suggested oxygen flow vary from from 100 to 200 ml/kg/min, which allows a FiO_2 of between 30 and 80%. Using 2 cannulas allows to reach a higher total FiO_2 by limiting the flow in each nostril. This may also ensure a better tolerance of the patient. Nasal oxygen allows better access to patients, which can facilitate follow-up and monitoring.

Advances oxygen delivery methods: Sometimes, conventional oxygen delivery methods are not sufficient to alleviate the respiratory signs or achieve a satisfactory PaO_2. In these cases, high-flow nasal therapy (HFNT) or mechanical ventilation should be considered. HFNT involves the use of designated prongs and allows for the delivery of a high flow of oxygen at FiO_2 levels up to 100%. These high flow rates, which are permitted because the air is heated and humidified, allow a dead space washout and a certain level of CPAP (mainly when a flow rate of 2 L/kg/min is used). The size of the cannula should not obstruct the nares by more than 50%. Complications associated with HFNT include intolerance, aerophagia, and the development of pneumothorax in rare instances. Mechanical ventilation is necessary in cases of hypoxemia refractory to oxygen supplementation (PaO_2 <60 mmHg), hypoventilation ($PaCO_2$ >60 mmHg), or markedly increased respiratory efforts. Mechanical ventilation involves endotracheal intubation, intravenous anaesthesia, dedicated staff, and associated costs that can be high depending on the duration of treatment.

References

- *Krawec P, Marshall K, Odunayo A. A Review of High Flow Nasal Cannula Oxygen Therapy in Human and Veterinary Medicine. Top Companion Anim Med. 2022;46. doi:10.1016/j.tcam.2021.100596*
- *Hopper K, Powell LL. Advanced Oxygen Therapy for the Small Animal Patient - High-Flow Oxygen Therapy and Mechanical Ventilation. Vet Clin North Am - Small Anim Pract. 2022;52(3):689-705. doi:10.1016/j.cvsm.2022.01.006*
- *Tong CW, Gonzalez AL. Respiratory Emergencies. Vet Clin North Am - Small Anim Pract. 2020;50(6):1237-1259. doi:10.1016/j.cvsm.2020.07.002*
- *Lynch, A. Providing Supplemental Oxygen to Patients. Today's Veterinary Practice. 2018. https://todaysveterinarypractice.com/respiratory-medicine/providing-supplemental-oxygen-to-patients*

Disorders of sodium and water balance - basic pathophysiology and practical case examples

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Learning objectives

- The participants will learn how to manage patients with disorders of sodium and water balance by
- Reviewing basic physiological principles of sodium and water balance
- Discussing the theoretical basis of the management of patients with disorders of sodium and water balance
- Working through practical case examples

Lecture summary

Disorders of sodium and water balance - basic pathophysiology and practical case examples: In the vast majority of patients, disorders of serum sodium concentration result from abnormalities in water handling rather than an increased or decreased number of sodium molecules. Clinical signs associated with disorders of serum sodium and water balance are primarily neurological in nature (ex. obtundation, head pressing, seizures, coma and death) and related to osmotic movement of water out of / into the brain cells and the resultant changes in cell volume. The clinical signs associated with hyper- / hyponatremia and potential complications of management are related to the magnitude, ex. severe hypernatremia (≥170 mEq/L), severe hyponatremia (<120 mEq/L) or rapid changes in serum sodium concentration.

Hypernatremia: Patients which are able to drink, and have access to water rarely develop significant hypernatremia. Patient with hypernatremia have a free water deficit. To correct hypernatremia, free water is administrated in the form of hypotonic fluids, including 5% dextrose in water or 0.45 percent sodium chloride. Correction of hypernatremia caused by hypertonic sodium gain requires the excretion of excess sodium and water through the administration of Furosemide (furosemide-induced diuresis is equivalent to one-half isotonic saline solution and will aggravate the hypernatremia) and the administration of free water. Patients with hypernatremia due to gain of sodium may be hypervolemic and may develop signs of volume overload. Free water may be administrated IV as 5% dextrose in water. Water can also be given orally or via nasoesophageal or nasogastric tube in patients that don't have significant neurological deficits. Ongoing hypotonic fluid losses may outpace the administration of free water and aggravate the hypernatremia. Measuring urine sodium concentration, and urine output may facilitate fluid therapy prescription and allow for appropriate calculation and replacement of ongoing fluid losses. If hypernatremia has developed over a period of hours reducing the serum sodium concentration by 1 mEq/L/h is appropriate. In patients with chronic hypernatremia or in those in which the duration of hypernatremia is unknown serum sodium concentration should not be decreased by more than 0.3-0.5 mEq/L/h. In patients with severe neurological deficits due to hypernatremia serum sodium concentration may be decreased initially by 3-7 mEq/L until neurological signs improve, with the daily decrease ≤10-12 mEq/L (chronic hypernatremia).

Equation to calculate the effect of 1 liter of any infusate on serum sodium concentration: Change in serum Na = infusate Na - plasma Na / (total body water + 1). Total body water = 0.5-0.6 x kg. Using the above equation will give a rough estimate of the anticipated changes in serum sodium concentration, however since the resulting changes in serum sodium concentration may vary significantly between individual patients, serum sodium concentration and neurological status have to be evaluated at least every 4 hours, and fluid therapy adjusted accordingly. Cerebral oedema is the primary complication of therapy to correct hypernatremia. If neurological signs develop during the treatment of hypernatremia the serum sodium concentration should be measured to confirm that it has decreased since signs of worsening hypernatremia may be similar to those seen with cerebral oedema.
Cerebral oedema is treated with mannitol at 0.5-1 gr/kg IV over 20-30 minutes or by administration of hypertonic saline.

Hyponatremia: Hyponatremia usually implies hypoosmolality (hypotonic hyponatremia). Patients with hypotonic hyponatremia typically have free water retention in excess of sodium retention, although they may have sodium loss as well.

Calculation of plasma osmolality: Osmolality (mOsm/kg) = 2 x (Na) + (BUN mg/dL : 2.8) + (glucose mg/dL : 18). BUN and glucose concentration are divided by 2.8 and 18, respectively to convert them from mg/dL to mmol/L. There is no consensus on the optimal treatment for symptomatic hyponatremia. Serum sodium concentration should be increased by restriction of free water intake and / or intravenous administration of hypertonic saline. In patients with severe neurological deficits due to hyponatremia serum sodium concentration may be increased initially by 3-7 mEq/L until neurological signs improve, with the daily increase ≤10-12 mEq/L (chronic hyponatremia). Myelinolysis is the major complication of therapy to correct hyponatremia. Clinical signs include paresis, ataxia, and changes in mentation rarely develop during the initial treatment of hyponatremia. If neurological signs develop during the treatment of hyponatremia serum sodium concentration should be measured since signs of worsening hyponatremia may be similar to those seen with myelinolysis.

References

- *Burkitt Creedon JM: Sodium disorders: In Silverstein DC, Hopper K (Hrsg.), Small Animal Critical Care Medicine, ed 2, Philadelphia , 2008, Saunders Elsevier, pp 263-268.*
- *Rose BD: Introduction to renal function. In Rose BD, editor: Clinical physiology of acid-base and electrolyte disorders, ed 4, New York, 1994, McGraw Hill, pp 2-19.*
- *Verbalis JG. Brain volume regulation in response to changes in osmolality. Neuroscience 2008; 168: 862-870.*
- *DiBartola SP: Disorders of sodium and water: hypernatremia and hyponatremia. In DiBartola SP, editor: Fluid, electrolyte, and acid-base disorders in small animal practice, ed 4, St Louis, 2012, Saunders Elsevier, pp 45-79.*
- *Kahn A, Brachet E, Blum D. Controlled fall in natremia and risk of seizures in hypertonic dehydration. Intensive Care Med 1979; 5: 27-31.*
- *Adrogué HJ, Madias NE. Hypernatremia. N Engl J Med 2000; 342: 1493-1499.*
- *Barr JM, Khan SA, McCullough SM et al. Hypernatremia secondary to homemade play dough ingestion in dogs: a review of 14 cases from 1998 to 2001. J Vet Emerg Crit Care 2004 ; 14: 196-202.*
- *Adrogué HJ, Madias NE. Hyponatremia. N Engl J Med 2000; 342: 1581-1589.*
- *Porzio P, Halberthal M, Bohn D et al. Treatment of acute hyponatremia: Ensuring the excretion of a predictable amount of electrolyte-free water. Crit Care Med 2000; 28: 1905-1910.*
- *Sarnaik AP, Meert K, Hackbarth R et al. Management of hyponatremic seizures in children with hypertonic saline: a safe and effective strategy. Crit Care Med 1991; 19: 758-762.*
- *Worthley LIG, Thomas PD. Treatment of hyponatraemic seizures with intravenous 29.2% saline. BMJ 1986; 292: 168-170.*
- *MacMillan KL. Neurologic complications following treatment of canine hypoadrenocorticism. Can Vet J 2003; 44: 490-492. 13. Plumb DC (Hersg.): Veterinary drug handbook, ed 6, Ames, Iowa, 2008, Blackwell Publishing Professional, pp 555-557.*
- *Oh MS, Kim HJ, Carroll HJ. Recommendations for treatment of symptomatic hyponatremia. Nephron 1995; 70: 143-150.*

Endocrine emergencies

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Learning objectives

General approach to all/most common endocrine emergencies (unstable diabetics/DKA, Addisonian crisis, hyperaldosteronism, thyroid storm and pheochromocytomas).Focus on emergency stabilization and workup in the ER. Emphasis on owner communication for long term and short term management of patients with these diseases and how they are managed. Discuss some of the challenges with in stabilizing/managing these cases to facilitate a clear discussion with the owner at the time of diagnosis. Discuss some of the challenging client communication that surrounds the decision-making in these cases, DKA in particular.

Lecture summary

Endocrine disorders are common presenting complaints in dogs and cats in an emergency setting. Although typically presenting with chronic, insidious, and slowly progressive signs, there are some instances when endocrine disease can present with life-threatening complications.

Complicated Diabetes Mellitus: Diabetic ketoacidosis (DKA) and Hyperglycemic Hyperosmolar Syndrome (HHS) are 2 diabetic crises that require emergency intervention.

DKA is identified by presence of: Hyperglycemia, Glucosuria, Ketonemia, or ketonuria, Metabolic acidosis as evidenced by low pH, low bicarbonate, and large negative base excess on arterial or venous blood gas (VBG) analysis. The diagnostic criteria for HHS include: Severe hyperglycemia, Minimal or absent serum or urine ketones, Severe hyperosmolality (serum osmolality >350 mOsm/L). DKA and HHS share a common yet divergent pathophysiology. In both, an absolute or relative lack of insulin renders most cells unable to use glucose for energy and promotes gluconeogenesis and glycogenolysis. With HHS, the main difference is presence of small amounts of insulin and hepatic glucagon resistance which allows for inhibition of lipolysis and subsequently a lack of ketosis. In these patients, the primary abnormality is hyperglycemia → osmotic diuresis → vicious circle of progressive dehydration and hyperosmolality. Goals of therapy for patients with HHS and DKA are to:: Replace dehydration deficit and vascular volume. Manage electrolyte abnormalities. Initiate insulin therapy to help reduce glucose levels and reverse ketone production in DKA- often a short-acting insulin in the emergency setting with subsequent transition to long-acting insulin for maintenance. Treat underlying diseases that may have precipitated the diabetic crisis.

Hypoadrenocorticism: Classically manifests as a deficiency of cortisol and mineralocorticoids, although isolated cortisol deficiency can also occur. Primary HA occurs secondary to destruction of the adrenal cortex either because of immune-mediated destruction (most common in dogs), neoplasia, infection, hemorrhage, iatrogenic causes (e.g. mitotane, trilostane), or hemorrhage. Secondary HA is the absence of cortisol, which occurs when the pituitary fails to produce ACTH. Tertiary HA is the lack of corticotrophin-releasing hormone secretion and is much rarer. Clinical signs of HA primarily include: Lethargy, Weakness, PU/PD, Gastrointestinal signs, including anorexia, vomiting, diarrhea, or abdominal pain. Tremors/shaking. Collapse. Physical examination findings are dependent on the stage of disease: Dehydration +/- shock. Bradycardia with severe mineralocorticoid deficiency and subsequent hyperkalemia
Therapeutic Goals: Aggressive management of shock and dehydration. Treat hyperkalemia if ECG changes seen. Caution with use of 0.9% NaCl as fluid of choice in severely hyponatremic patients- too fast of a rise in serum sodium → risk of osmotic demyelination syndrome
Glucocorticoid therapy: Dexamethasone: initial "crisis" dose: 0.1-0.5 mg/kg, subsequent doses 0.05-0.1 mg/kg IV q12 → transitioned to physiologic doses of prednisone for long-term management. DOCP: 2.2mg/kg IM q25 days for mineralocorticoid replacement (recent data shows that individualized dosing and lower doses can be considered)

Pheochromocytoma: Uncommon tumor of the catecholamine-secreting chromaffin cells of the adrenal medulla. This neoplasm can be malignant or locally invasive into regional vasculature and can be seen in dogs with concurrent hyperadrenocorticism. Acute presentation is usually associated with severe hypertension, hemorrhage, or arrhythmias, requiring emergent intervention. α-Blockade: typically using phenoxybenzamine or prazosin. This takes several days for full effect so is typically not useful in a hypertensive crisis. Fast-acting vasodilator drugs such as nitroprusside or hydralazine can be used in an acute hypertensive crisis. Nitroprusside: 0.5 mcg/kg/min and titrate up to desired BP Hydralazine: 0.5-2 mg/kg PO q8-12. In general, the use of β-blockers should be avoided until α-blockade is in full effect. Ultimately, surgical removal of the affected adrenal gland +/- associated tumor thrombus is typically indicated.

Thyroid Storm: Rare, acute exacerbation of thyrotoxicosis marked by: Fever. CNS, cardiovascular, and GI or hepatic signs. The levels of total and free T4 in patients with TS are not different from those in patients with hyperthyroidism without crisis. Tachycardia, arrhythmias, gallop rhythm, and murmurs may all be identified in thyrotoxic cats. Other clinical consequences of a thyroid storm include presentation in congestive heart failure, which can be marked by signs of tachypnea and respiratory distress. Severe hypertension can precipitate neurologic signs (acute blindness from retinal hemorrhage or retinal detachment, seizures, depressed mentation or stupor). Weakness and cervical ventroflexion can be seen secondary to severe hypokalemia, and loss of limb function as a result of thromboembolism has also been reported.
Treatment Goals: Manage systemic manifestations, Reduce excess thyroid hormone levels, Symptomatic therapy: Anti-arrhythmic therapy with beta blockade (esmolol, atenolol) - Use with great caution if patient is in active CHF Use with great caution if patient is in active CHF. Use of vasodilators for hypertensive crisis. Potassium supplementation for treatment of hypokalemia. Reduction of thyroid levels: Methimazole (oral, rectal or transdermal). Plasmapheresis or TPE.

References

• *Drobtaz KJ et al. Textbook of Small Animal Emergency Medicine, Hoboken, USA. Wiley Blackwell; 2018*
• *Silverstein D.C., Small animal critical care medicine, St. Louis, USA,: Elsevier, Saunders; 2022*

When sniffing around sends you to the ER - hypersensitivity pneumonitis

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Learning objectives

- Define what hypersensitivity pneumonitis is
- Give examples of causative agents
- Describe the clinical presentation and management of hypersensitivity pneumonitis in dogs

Lecture summary

Definition: Hypersensitivity pneumonitis, historically named "extrinsic allergic alveolitis", is an inflammatory and/or fibrotic lung parenchymal and small airways disease that typically results from an immune-mediated reaction in susceptible individuals. When very small antigenic particles (5-10 microns) reach the alveoli, they can induce a more or less severe inflammatory reaction, depending on the host and the antigenic load inhaled, resulting in radiographic and clinical signs of varying intensity. In people, an extensive number of causative agents have been described, including microorganisms (fungal, bacterial, protozoal), agricultural dusts, bioaerosols, and reactive chemical species. In the past, hypersensitivity pneumonitis (HP) has been categorized as acute, subacute, and chronic, depending on the clinical presentation. More recently, it has been categorized into two subtypes: acute HP and chronic HP, with or without fibrosis (fibrotic or non-fibrotic HP). This syndrome has been reported in humans and in a paucity of cases in dogs.

Pathogenesis: The immunopathological mechanisms that lead to the development of HP and associated signs are not completely understood. An aberrant immune response is suspected, and a two-hit hypothesis has been suggested. The first hit would be a genetic susceptibility in certain individuals. In fact, although the inciting antigens that can lead to HP are distributed throughout the world and in many environments, only a small number of people develop the disease after being exposed to the inhaled particles (the second hit). An increase in individual susceptibility to HP is thought to be related to polymorphism in genes related to molecules involved in antigen processing and presentation. Class II major histocompatibility complex (MHC-II) molecules appear to be mainly involved. Polymorphism in genes implicated in lung homeostasis and wound repair could also be linked to an increased susceptibility. HP is mostly believed to be characterized by a type IV hypersensitivity reaction (T-cell mediated immunity, principally Th1 cells) and a type III hypersensitivity reaction (immune complex-mediated injury with IgG antibodies).

Human literature: clinical signs, diagnostics, and treatments: In people, clinical signs of HP include cough, breathlessness, chest pain and/or tightness, dyspnea, chills, malaise, fatigue, fever, headache. Signs can be acute or chronic. In the acute form, the symptoms (usually flu-like) appear 4-8 hours after exposure to the antigen and resolve gradually within 12 hours to several days following antigen exposure removal. Acute HP can be self-limiting. HP is suspected in patients with known exposure to agent, and in patients with clinical signs and imaging evidence of interstitial lung disease. Although chest radiographs can demonstrate micronodular or reticular opacities, they may be normal. High-resolution computed tomography (HRCT) is therefore the preferred imaging modality. Other tests and findings, such as serum IgG testing for the presence of antigen exposure, bronchoalveolar lavage lymphocytosis, and lung biopsy demonstrating granulomas, inflammation, and fibrosis can also be performed to strengthen the diagnosis. If pulmonary function is evaluated, a restrictive pattern, particularly a reduced forced vital capacity, is commonly found, as well as a reduction in diffusing capacity. As previously mentioned, acute HP can resolve spontaneously without specific treatment once the exposure to the antigen has ceased. In more severe cases, corticosteroid therapy is necessary. Other immunosuppressives agents, such as azathioprine and mycophenolate, might be necessary in fibrotic HP. Anti-fibrotic agents such as pirfenidone and nintedanib may become promising treatments in progressive fibrotic form of HP. Depending on the severity of the respiratory signs and hypoxemia, invasive or non-invasive oxygen therapy is provided. The prognosis depends on the presence or absence of fibrosis and the ability to identify and eliminate the offending antigen.

What is known in veterinary medicine: Hypersensitivity pneumonitis is very rare in veterinary medicine. Only five case reports are described in the literature, and all concern dogs (no feline cases have been reported to the author's knowledge). All cases of HP reported in the veterinary literature are lycoperdonosis or lycoperdonosis-like. In this presentation, a case of acute respiratory failure caused by lycoperdonosis will be described. Lycoperdonosis is the term used to describe an acute form of hypersensitivity pneumonitis resulting from the inhalation of large amounts of spores from mature puffball mushrooms. In this lecture, we will give a detailed presentation of our case, and we will describe other canine cases reported in the literature, highlighting the similarities and differences with human cases. We will focus on clinical presentation, diagnostics, treatments, and prognosis. Because HP can be severe and life-threatening, it is important to be aware of the existence of this syndrome to be able to suspect and consider it as a differential diagnosis when a dog presents with acute respiratory signs.

References

- *Alenghat T, Pillitteri CA, Bemis DA, et al. Lycoperdonosis in two dogs. J Vet Diagn Invest. 2010;22(6):1002-1005. doi:10.1177/104063871002200629*
- *Whitney J, Beijerink N, Martin P, Talbot J, Barrs V. Hypersensitivity pneumonitis in a dog associated with Geastrum triplex spores. Med Mycol Case Rep. 2013;2(1):122-124. doi:10.1016/j.mmcr.2013.05.002*
- *Buckeridge D, Torrance A, Daly M. Puffball mushroom toxicosis (lycoperdonosis) in a two-year-old dachshund. Vet Rec. 2011;168(11). doi:10.1136/VR.C6353*
- *Churg A. Hypersensitivity pneumonitis: new concepts and classifications. Mod Pathol. 2022;35(Suppl 1):15-27. doi:10.1038/S41379-021-00866-Y*
- *Barnes H, Troy L, Lee CT, Sperling A, Strek M, Glaspole I. Hypersensitivity pneumonitis: Current concepts in pathogenesis, diagnosis, and treatment. Allergy. 2022;77(2):442-453. doi:10.1111/ALL.15017*
- *Moua T, Petnak T, Charokopos A, Baqir M, Ryu JH. Challenges in the Diagnosis and Management of Fibrotic Hypersensitivity Pneumonitis: A Practical Review of Current Approaches. J Clin Med. 2022;11(6). doi:10.3390/jcm11061473*
- *Dabiri M, Jehangir M, Khoshpouri P, Chalian H. Hypersensitivity Pneumonitis: A Pictorial Review Based on the New ATS/JRS/ALAT Clinical Practice Guideline for Radiologists and Pulmonologists. Diagnostics (Basel, Switzerland). 2022;12(11):2874. doi:10.3390/diagnostics12112874*
- *Huntley CC, Walters GI. An update on hypersensitivity pneumonitis: what a clinician wants to know. Curr Opin Pulm Med. 2021;27(2):95-104. doi:10.1097/MCP.0000000000000763*
- *Selman M, Buendía-Roldán I. Immunopathology, diagnosis, and management of hypersensitivity pneumonitis. Semin Respir Crit Care Med. 2012;33(5):543-554. doi:10.1055/s-0032-1325163*
- *Rubensohn M. Practitioners' Corner Le Coin Des Praticiens Inhalation Pneumonitis in a Dog from Spores of Puffball Mushrooms. Vol 50.; 2009.*

Heart lung interactions during the acute respiratory distress syndrome

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Learning objectives

Brief review of general pulmonary vascular mechanics and right ventricular physiology. Clinical consequences of, and evaluation of right ventricular failure. Fluid therapy management in ventilated ARDS patients with signs of RV failure. Potential approaches to mechanical ventilation including the open lung approach in patients with clinical signs of RV failure.

Lecture summary

Normal Right Ventricular Physiology: One of the hallmarks of pulmonary circulation when compared to systemic circulation is that it remains a low-pressure circuit, both at baseline resting states and during exertional states. In systemic circulation, exertional increases in cardiac output cause a significant rise in systemic blood pressure while in pulmonary circulation, the pulmonary arterial pressure only has minimal increases, and the pulmonary vascular resistance may decrease. This difference exists due to the ability of the lungs to recruit collapsed capacitance vessels in the face of increased cardiac output and the overall low vascular motor tone of the proximal pulmonary vascular bed. This leads to a minimal pressure drop between mean arterial and venous pressure in the pulmonary circulation (<10 mmHg), where the greatest pressure gradient occurs across the pulmonary microcirculation and alveolar capillaries. Given the low vascular tone in the pulmonary circulation, it is difficult to change the pulmonary arterial pressure acutely markedly. Even during states of acute hypoxia, with hypoxic pulmonary vasoconstriction, the overall pulmonary vascular resistance tends to be lower than baseline systemic vascular resistance.

What happens with changes in vascular resistance?
Because of the above physiological differences, the RV is poorly suited to handling acute large increases in afterload (while the more muscular LV is better equipped to handle this). Conversely, the more compliant RV can handle preload increases much better than the LV can. As RV afterload increases, the RV begins to dilate limiting its ability to increase contractility.
Interventricular dependence: This is an important concept impacting how the failure of one ventricle can impact the other due to the shared interventricular septum (IVS). As the RV dilates in response to increases in afterload, it can impede LV filling by moving the IVS toward the LV in a process known as interventricular dependence. This change can cause a "D-shape" to appear in cross section due to the flattened IVS (which normally bows into the RV in diastole). This reduced LV filling in this setting is often refractory to intravascular volume expansion, as raising central venous pressure and RV preload only increases the difference between RV end-diastolic pressure and LV end-diastolic pressure, and further compromises LV filling.
RV dysfunction vs RV failure: RV dysfunction represents any abnormalities typically detected in RV function with echocardiography, while RV failure represents a clinical syndrome characterized by low cardiac output and high RV end-diastolic pressure.

What Causes RV failure?
Causes of RV failure have not been thoroughly documented in veterinary patients but have been studied extensively in people. Causes of RV failure documented in people include cardiomyopathies, valvular heart disease, sepsis or other states of systemic inflammation, infiltrative disease, pressure overload of the RV (such as with pulmonary hypertension). In general, the progression of RV dysfunction to failure arises from increased wall stress → myocyte hypertrophy and chamber dilation → reduced ventricular function → increased filling pressures → increasing RV workload and microcirculatory dysfunction → oxygen supply-demand mismatch → RV ischemia

What specifically happens in patients with ARDS or other acute lung injury?
Potential factors leading to RV dysfunction in these patients include: Increased pulmonary vascular resistance due to a complex interplay of inflammatory mediators causing both vasoconstriction and vasodilation, hypoxic pulmonary vasoconstriction and pulmonary vascular remodelling. Hypercapnia and respiratory acidosis. Mechanical ventilation. Sepsis.
Detection of RV failure: Invasive hemodynamic monitoring: Evaluating for pulse pressure variation (PPV) could potentially indicate a fluid-responsive state or increased RV afterload. In these patients, a small fluid challenge can be attempted, and if the PPV does not improve, potential RV dysfunction should be investigated with echocardiography. Echocardiographic evaluation: Evidence of RV dilation as well as systolic and diastolic septal dyskinesia can be evaluated with 2-d echocardiography. Other methods, such as cardiac MRI or CT, or invasive monitoring with the use of pulmonary arterial catheters has not been well studied and is unlikely to have practical implications for use in veterinary patients due to high cost and lack of availability widely.
Management Strategies: Preload Optimization: Improvement of RV inotropy, Reducing RV afterload, Protective ventilation strategies

In conclusion, right ventricular failure is a potentially life-threatening condition and can result from a variety of underlying causes, including ARDS. Further study of the incidence and clinical course of right ventricular failure in veterinary patients is needed to better understand this condition and ultimately determine appropriate treatment options for these patients.

References

• *Sipmann FS, Santos A, Tusman G. Heart-lung interactions in acute respiratory distress syndrome: pathophysiology, detection and management strategies. Ann Transl Med. 2018 Jan;6(2):27*
• *Visser LC, Scansen BA, Schober KE, Bonagura JD. Echocardiographic assessment of right ventricular systolic function in conscious healthy dogs: repeatability and reference intervals. Journal of Veterinary Cardiology. 2015 Jun 1;17(2):83-96*

From children to dogs and cats: what veterinary medicine can learn from assisted ventilation in pediatric patients

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Learning objectives

- To learn about major etiologies which require assisted mechanical ventilation in children
- To learn about different modalities of invasive mechanical ventilation in children and how this can be applicable to dogs and cats
- To learn about different modalities of non-invasive mechanical ventilation in children and how these can be applicable to dogs and cats
- To present clinical cases of typical pediatric diseases and how its management can be applicable to canine and feline diseases

Lecture summary

Lecture context: Critically ill pediatric patients frequently require some form of ventilatory assistance during their disease process. The average pediatric intensive care unit (PICU) has about 30-63% of its patients mechanically ventilated for a mean duration of 5-7 days. In addition, the most common cause of PICU admission is acute or impending respiratory failure requiring mechanical ventilation (MV). Until recently, invasive MV (IMV) was the main form of ventilatory support in critically ill children. In recent years, technological improvements, increased clinical experience and recognition that it is associated to less complications have led to an increased use of non-invasive IMV (NIMV) in PICUs. Performing MV in children is challenging for several reasons which are similar to those found in veterinary medicine. First, children are very heterogeneous in terms of age, weight and physiological conditions such as lung maturity. In addition, there is a large spectrum of acute and chronic conditions which can be treated with MV. Some of these diseases are specific of this age group, and the ones which are not, behave differently from its counterpart in adults (e.g., Pediatric ARDS-PARDS). To complicate further, there is a paucity of large-scale randomized controlled trials which can generate data to guide clinical practice. In fact, in many PICUs, in another similarity to what is common practice in veterinary medicine, MV is instituted based on personal experiences, local PICU policy and protocols (which can vary within and between countries) and what pediatric critical care practitioners have adapted from adult and neonatal experience. Despite these factors, much progress has been made in the last decades.
NIMV: Assisted MV can be divided in three phases: initiation, escalation and resolution. Nowadays, for most clinical conditions, NIMV is the initial mode used in the initiation phase of management of pediatric acute respiratory failure. NIMV comprises High-flow Nasal Oxygen (HFNO) therapy, Continuous Positive Airway Pressure (CPAP) and bilevel positive airway pressure (BiPAP) ventilation. In recent years, one has seen the widespread use of HFNO for many clinical conditions and its transfer from PICU to emergency rooms and pediatric wards. When HFNO fails, escalation proceeds to CPAP or BiPAP or, if the condition does not permit it, to intubation and IMV. Despite the lack of clear guidelines, NIMV has been used to treat pneumonia, upper airway obstruction, post-extubation respiratory failure, acute cardiogenic pulmonary edema and acute chest syndrome. It is also considered the first line of treatment in mild PARDS.
IMV: Recent advancements in IMV were centered on optimizing ventilator settings and customizing monitoring with the overarching goal of reducing complications of IMV, such as ventilator-induced lung injury. One such example includes the choice of volume-guaranteed controlled modes which permit, as the name suggests, a controlled tidal volume in all breaths. There is no data available in pediatric patients to dictate the best ventilation mode and parameters for most clinical conditions with its choice being dictated by clinical experience and theoretical arguments considering disease´s condition.
Other developments: In recent years, several developments have been reported including new mechanical ventilator strategies integrating esophageal pressure monitoring, transpulmonary pressure, volumetric capnography, and neurally adjusted ventilator assist (NAVA) to optimize conventional ventilator support. Studies are ongoing to clarify the role of these promising developments in MV of pediatric critically ill children. Nonconventional modes of ventilation in PICU include high-frequency modes and airway pressure release ventilation. Extracorporeal pulmonary support via extracorporeal membrane oxygenation or paracorporeal lung assist devices provides rescue options when conventional and nonconventional methods fail.
Clinical cases: The use of MV for PARDS, bronchiolitis and acute exacerbation of asthma will be discussed through the presentation of real clinical cases, including its clinical course and final outcome.

Conclusions

The use of MV in pediatric patients is evolving. Areas of ongoing research include timing of tracheostomy, risk reduction in ventilator-induced lung injury, and decreased sedation requirements. In many aspects MV in veterinary species shares many of the challenges of pediatric MV. This suggests than a multidisciplinary collaboration can be performed, with benefits to both human and veterinary critically ill patients.

References

- *Conti G., Piastra M. Mechanical ventilation for children. Curr Opin Crit Care. 2016; 22(1):60-66.*
- *Kneyber M.C.J. et al. Recommendations for mechanical ventilation of critically ill children from the Pediatric Mechanical Ventilation Consensus Conference (PEMVECC). Intensive Care Med. 2017;43(12):1764-1780.*
- *Kollisch-Singule M., Ramcharran H., Satalin J., Blair S., Gatto L.A., Andrews P.L., Habashi N.M., Nieman G.F., Bougatef A. Mechanical Ventilation in Pediatric and Neonatal Patients. Front Physiol. 2022; 17; 12: 805620. doi: 10.3389/fphys.2021.805620*
- *Miller A.G., Scott B.L. 2021 Year in Review: Pediatric Mechanical Ventilation. Respir Care. 2022;67(11):1476-1488. doi: 10.4187/respcare.10311*
- *Nardi N., Mortamet G., Ducharme-Crevier L., Emeriaud G., Jouvet P. Recent advances in Pediatric Ventilatory Assistance. 2017; F1000Res, 17(6):290-300.*
- *Pediatric Acute Lung Injury Consensus Conference Group. Pediatric Acute Respiratory Distress Syndrome: Consensus recommendations from the Pediatric Acute Lung Injury Consensus Conference. Pediatr Crit Care Med. 2015; 16 (5): 428-39.*
- *Weiss S.L., et al. Surviving sepsis campaign international guidelines for the management of septic shock and sepsis-associated organ dysfunction in children. Intensive Care Med. 2020; 46(1):10-67.*

Gary stamp memorial lecture: mechanical ventilation - complex cases

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Learning objectives

- To review options for the management of refractory hypoxemia in the ventilator patient
- To understand assessment and management of severe hypercapnia in the ventilator patient
- To consider patient management challenges such as airway management and anesthetic choices

Lecture summary

Refractory Hypoxemia: The most challenging mechanical ventilator cases are those with severe lung disease. These patients have severe hypoxemia that does not always improve with the initiation of mechanical ventilation. A lung protective approach to ventilator settings is recommended for all animals with lung disease, including low tidal volume ventilation, moderate to high positive end expiratory pressure (PEEP) and permissive hypercapnia. One of the most frequently asked questions in these patients is how to select the best PEEP for a patient. The aim of PEEP is to prevent alveolar collapse and reopen already collapsed alveoli. Ultimately the goal of PEEP is to improve ventilation-perfusion matching in the lung which will improve oxygenation. PEEP may also reduce ventilator induced lung injury (VILI) by keeping alveoli from cyclic reopening on inspiration and collapse on exhalation, otherwise known as atelectrauma. There are a lot of studies on PEEP selection in adult human acute respiratory distress syndrome (ARDS). The relevance of these studies on canine patients with pneumonia is difficult to determine. It is important to recognize that many lung diseases do not improve with PEEP as they are not 'recruitable', meaning the alveoli cannot be reopened with increased airway pressure. PEEP has the potential to create harm as it can cause alveolar overdistension, a well-recognized mechanism of VILI, it can create alveolar dead space and can have adverse cardiovascular effects. Further, human studies are yet to define the true benefits of PEEP. A 2021 meta-analysis identified 10 studies with a total of 3851 adult ARDS participants and found little to no difference in the in-hospital mortality between high (mean of 13 to 16 cmH_2O) and low levels of PEEP (mean of 5 to 11 cmH_2O). Ultimately, as a clinician managing a case with severe lung disease, some level of PEEP needs to be determined for the individual case. There are many approaches including the use of human ARDS protocols where PEEP is increased until oxygenation improves. This could be harmful for patients that do not respond to PEEP. PEEP trials where multiple levels of PEEP are evaluated in a patient and the clinician chooses the PEEP that provides the most benefits and the least concerns is labor intensive, but may be beneficial in animals with refractory hypoxemia.

Refractory Hypercapnia: Hypercapnia can be a common frustration when managing severe lung disease on the ventilator. This is often a byproduct of a low tidal volume, high respiratory rate approach. Permissive hypercapnia, where a higher-than-normal level of carbon dioxide is tolerated rather than increasing the ventilator settings is generally in the range of a $PaCO_2$ of 50-65 mmHg. When $PaCO_2$ is elevated beyond this level, it requires re-evaluation of the ventilator settings. Normal human tidal volumes are lower than normal dogs and cats that have far great anatomic dead space. It may not be possible to effectively ventilate dogs and cats with extremely low tidal volumes. Another consideration is if PEEP is creating too much alveolar dead space which would further limit excretion of carbon dioxide.

Troubleshooting and Complications: Ventilator patients are some of the most compromised patients in the ICU and can experience many different concerns that maybe associated with the primary disease, acquired new disease, anesthesia, nursing care and/or machine settings. When lung function declines gradually while the animal is on the ventilator concerns include progression of the underlying disease, VILI, ventilator associated pneumonia and aspiration pneumonia. The incidence of ventilator associated pneumonia and aspiration pneumonia can be reduced by meticulous nursing care protocols. Acute changes in lung function requires immediate attention and possible cause include pneumothorax, machine malfunction and loss or problem with the artificial airway. Hemodynamic challenges are also common in the ventilator patient, this can be associated with the anesthetic protocol, volume status, primary disease process (e.g. sepsis) or the ventilator settings used. Continuous arterial blood pressure measurement is important in the ventilator patient. Many complications in the ventilator patient are preventable and/or require rapid recognition and resolution to prevent patient mortality. The benefits of a trained and experienced nursing care team cannot be overstated.

References

- *Hopper K. Mechanical Ventilation - Core Concepts. In: Silverstein DC, Hopper K, editors. Small Animal Critical Care Medicine, ed 3. St Louis, USA, Elsevier, 2023. pp. 101-112*
- *Hopper K. Mechanical Ventilation - Advanced Concepts. In: Silverstein DC, Hopper K, editors. Small Animal Critical Care Medicine, ed 3. St Louis, USA, Elsevier, 2023. pp. 193-197*
- *Shelledy DC, Peters JI. Mechanical Ventilation, ed 3. Burlington, USA, Jones and Bartlett Learning, Burlington, 2020. pp. 95-366*
- *Hess D, Kacmarek R. Essentials of Mechanical Ventilation, ed 4. New York, USA, McGraw-Hill, 2019. pp50-72*
- *Santa Cruz R, Villarejo F, Irrazabal C, Ciapponi A. High versus low positive end-expiratory pressure (PEEP) levels in mechanically ventilated adult patients with acute lung injury and acute respiratory distress syndrome.*
- *Cochrane Database of Systematic Reviews 2021;3:CD009098 Sahetya SK, Golgher EC, Brower RG. Setting positive end-expiratory pressure in acute respiratory distress syndrome. Am J Respir Crit Care Med 2017;11:1429-1438*
- *Shao S, Kang H, Qian Z, et. al. Effect of different levels of PEEP on mortality on mortality in ICU patients without acute respiratory distress syndrome: systematic review and meta-analysis with trial sequential analysis. J Crit Care 2021;65:246-258*

What's new in canine blood types and pre-transfusion compatibility testing

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Learning objectives

- Describe blood types in dog, focusing on newer blood types, and understand their clinical significance
- Understand the principal of blood compatibility in dogs
- Recognize an immune-mediated hemolytic transfusion reaction

Lecture summary

DEA 1: the most important canine blood group system: DEA 1 antigen analysis by immunochromatography and flow cytometry using a monoclonal antibody showed that the expression of DEA 1 is a continuum of reactions ranging from negative to strongly positive. Previously typed DEA 1.2+ are rather DEA 1+ with lower antigenic expression. DEA 1 is considered the most important canine blood group system, both because of its well-documented immunogenicity and of its overall prevalence. A DEA 1+ blood transfusion in a DEA 1- dog will invariably elicits a strong alloantibody production, and lead to hemolytic transfusion reaction if subsequent DEA 1+ blood is transfused. The risk of sensitization, and thereafter transfusion reactions, is high if untyped blood transfusions are used as roughly half of the canine population is DEA 1+ (47-65%, with geographical and breed variation),while the other half is DEA 1-. Thus, DEA 1 blood typing has been recommended before any blood transfusion in dogs for decades, which is facilitated nowadays by standardized typing kits using monoclonal antibodies based on agglutination reactions (DMS/RapidVet-H) or on chromatographic techniques (Alvedia).

Particularities of the "other DEAs": Until recently, extended blood typing was limited even in research context, but the commercialization of bedside assay for DEA 4 and 5 may improve accessibility. The clinical importance of DEA 4 comes from its high prevalence (>97% of dogs are DEA 4+), which places DEA 4- dogs at high risk of transfusion incompatibility and hemolytic reaction as previously documented in a clinical patient. As for DEA 3, 5 and 7, their clinical importance is less with only delayed hemolytic reactions reported in previously sensitized dogs.

Novel Canine Blood Types: Dal and Kai ½: In 2007, blood incompatibility in a previously transfused anemic Dalmatian led to the identification of a newly recognized blood type: Dal. Dal+ phenotype is dominantly inherited over Dal-. In addition to Dalmatian (11.7%), Doberman Pinschers (42.4%) and Shih Tzus (57.1%) were later found to have a high prevalence of Dal-negative individuals. Considering that all non-Doberman Pinschers blood donors tested to date are Dal+, Dal- dogs are at high risk of transfusion incompatibility if they are to receive multiple blood transfusions. The strong immunogenicity of Dal has been documented both in clinical patients and in research settings. Therefore, Dal blood typing, in addition to standard DEA 1 typing, is recommended in these breeds especially in previously transfused dogs which may be facilitated by a recently commercialized Dal typing cards (DMS/RapidVet-H). Most recently, a murine anti-Dal monoclonal antibody was produced and will likely facilitate reliable Dal blood typing soon. Following the production of monoclonal antibodies by mouse hybridoma technique, two new canine blood types Kai 1 and Kai 2 (meaning "dog" in Korean) have recently been identified. To date, Kai 1 and Kai 2 antigens have not been shown to coexist in any dogs tested. Their prevalence varies significantly between South Korea and the USA (South Korea: 42% Kai 1+, 37% Kai 2+, 20% Kai -; USA: 94% are Kai 1+). No naturally occurring anti-Kai alloantibodies have been documented but presumed anti-Kai alloantibodies were documented in 4 dogs 21 post-transfusion.

Naturally Occurring Alloantibodies in Dogs: First-time transfusions to dogs are considered safe without prior cross-matching, as dogs do not possess clinically significant naturally occurring antibodies. Similarly, pregnancy does not sensitize dogs to RBC antigens. Although mild immunologic incompatibilities have been documented in first-time transfusion recipients their clinical importance needs further investigation. Indeed, despite several recent publications documenting the presence of naturally occurring anti-DEA 7 antibodies in up to 50% of all DEA 7-negative dogs, the clinically significance of such antibodies has not been documented.

Assessing Blood Compatibility Beyond DEA 1: Transfusion reactions may also occur after a sensitized dog receives blood that is mismatched for a RBC antigen other than DEA 1 and hemolytic transfusion reaction have been described against DEA 4, Dal, and another unspecified common RBC antigen. A crossmatch is therefore essential in any dogs that have received transfusion > 4 days previously or have an unknown transfusion history. Many veterinary hospitals rely on a standardized tube crossmatching procedure which requires very little material, but some expertise given its subjectivity. Gel column techniques (DiaMed and Ortho Clinical) have been evaluated and found to be simple, sensitive, and standardized methods to crossmatch dogs: the objectivity of the results makes it an ideal research tool. Gel-based and immunochromatographic in-house crossmatching kits are now available, which facilitates the procedure and the interpretation of results, but have variable reported sensitivity and specificity (DMS Laboratories and Alvedia).

References

- *Blais MC et al. Canine Dal blood type: A red cell antigen lacking in some Dalmatians. J Vet Intern Med 2007; 21: 281-286.*
- *Blais MC et al. Lack of evidence of pregnancy-induced alloantibodies in dogs. J Vet Intern Med 2009; 23: 462-465.*
- *Corrales Mesa CL et al. Production and characterization of a murine anti-Dal monoclonal antibody for blood typing in dogs. Vet Immunol Immunopathol 2022;254:110516.*
- *Euler CC et al. Survey of Two New (Kai 1 and Kai 2) and Other Blood Groups in Dogs of North America. J Vet Intern Med 2016; 30: 1642-1647.*
- *Giger U et al. An acute hemolytic transfusion reaction caused by dog erythrocyte antigen 1.1 incompatibility in a previously sensitized dog. J Am Vet Med Assoc 1995; 206: 1358-1362.*
- *Goulet S & Blais MC. Characterization of Anti- Dal Alloantibodies Following Sensitization of Two Dal-Negative Dogs. Vet Pathol 2018; 55: 108-115.*
- *Goulet S et al. Prevalence and Mode of Inheritance of the Dal Blood Group in Dogs in North America. J Vet Intern Med 2017; 31: 751-758.*
- *Goy-Thollot I et al. Pre- and Post-Transfusion Alloimmunization in Dogs Characterized by 2 Antiglobulin-Enhanced Cross-match Tests. J Vet Intern Med 2017; 31: 1420-1429.*
- *Hale AS et al: An evaluation of 9570 dogs by breed and dog erythrocyte antigen typing. J Vet Intern Med 2008 [abstract]; 22: 740.*
- *Kessler RJ et al. Dog erythrocyte antigens 1.1, 1.2, 3, 4, 7, and Dal blood typing and cross-matching by gel column technique. Vet Clin Pathol 39: 306-316, 2010*
- *Lee JH et al. Kai 1 and Kai 2: Characterization of these dog erythrocyte antigens by monoclonal antibodies. PLoS One 2017; 12: e0179932.*
- *Melzer KJ et al. A hemolytic transfusion reaction due to DEA 4 alloantibodies in a dog. Journal of Veterinary Internal Medicine 2003; 17: 931-933.*
- *Polak K et al. Dog erythrocyte antigen 1: mode of inheritance and initial characterization. Vet Clin Pathol 2015; 44: 369-379.*
- *Spada E et al. Activity, specificity, and titer of naturally occurring canine anti-DEA 7 antibodies. J Vet Diagn Invest 2016; 28: 705-708.*
- *Spada E et al. Dog erythrocyte antigens (DEA) 1, 4, 7 and suspected naturally occurring anti-DEA 7 antibodies in Italian Corso dogs. Vet J 2017; 222: 17-21.*
- *Spada E et al. Prevalence of naturally occurring antibodies against dog erythrocyte antigen 7 in a population of dog erythrocyte antigen 7-negative dogs from Spain and Italy. Am J Vet Res 2016; 77: 877-881.*
- *Swisher SN & Young LE: The blood grouping systems of dogs. Physiol Rev 1961; 41: 495-520.*
- *Swisher SN, Young LE, Trabold N: In vitro and in vivo studies of the behavior of canine erythrocyte-isoantibody systems. Ann NY Acad Sci 1962; 10-15.*
- *Veran E and Blais MC. Validation of a cage-side agglutination card for Dal blood typing in dogs. Journal of Veterinary Internal Medicine. Accepted Jan 2023*
- *Vriesendorp HM et al. Joint report of the Second International Workshop on Canine Immunogenetics. Transplant Proc 1976; 8: 289-314.*

Update on feline blood typing and its clinical impact

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Learning objectives

- Describe the AB system and understand its clinical significance
- Understand the prevalence of naturally occurring alloantibodies in cats and the current knowledge of their clinical significance
- Recognize the existence of Feline Erythrocyte Antigens beyond the AB system

Lecture summary

In cats, significant knowledge gaps concerning blood banking and blood compatibility in cats have been filled, notably concerning the molecular characterization and molecular genetics of the AB system. However, much remains to be investigated especially regarding naturally occurring alloantibodies (NoAb) and corresponding red blood cell antigens (RBC) outside of the AB system.

Cat AB Blood Group System: The AB blood group system is composed of 3 phenotypes (type A, type B, and type AB) defined by NoAb against the antigen they lack. Most significant is the presence of strong anti-A alloantibodies in type B cats that may be responsible for acute hemolytic transfusion reactions and neonatal isoerythrolysis.

Incidence of the AB Antigens: Type A is the most common blood type. Type B is less common, and type AB is very rare. The percentage distribution of types A and B in domestic cats can vary markedly with geographic location worldwide. Type B domestic cats are uncommon in most countries; however their prevalence can reach up to 36% in Australia, Greece and Turkey. Similarly, the variation per breed is significant, ranging from 0% (ex: Siamese) to up to 60% (ex: Turkish Van and Angora cats) of Type B among different purebred cats. Type AB is exceedingly rare, except in Italian Ragdolls (reported frequency: 18-24%).

Molecular Characterization of the Antigens and Molecular Genetics: The A and B blood types of cats are caused by differences in the neuraminic acid residues present on a ceramide dihexose backbone on the RBC membrane. Types A RBCs have mainly N-glycolylneuraminic acid (NeuGc), whereas N-acetylneuraminic acid (NeuAc) is the determinant of the B antigen. Type AB cats express both NeuAc and NeuGc, in similar quantities. Cytidine monophospho-*N*-acetylneuraminic acid hydroxylase (CMAH) is the enzyme that catalyzes the conversion of NeuAc (type B antigen) to NeuGc (type A antigen). Molecular genetic studies, including a study in type AB Radgoll cats, have identified several variants (mutations) in the CMAH gene believed to disrupt the enzyme's function. Combined, these variants represent a promising diagnostic scheme to genotype all cats capable of differentiating type A, type B and type B.

The *Mik* Red Blood Cell Antigen: Based on the presence of a NoAb in three blood donor cats, the Mik blood group system was described in 2007. The clinical relevance of anti-Mik alloantibodies was documented after an acute hemolytic transfusion reaction following inadvertent transfusion of Mik-positive blood to a thereafter confirmed Mik-negative renal transplant recipient upon its first blood transfusion. Unfortunately, anti-Mik antibodies are no longer available preventing future investigations.

Naturally Occurring Alloantibodies and Corresponding Feline Erythrocyte Antigen Outside of the AB System: In recent literature, there is some evidence for the presence of other naturally NoAb outside the AB blood group system, like anti-Mik, but their clinical importance is poorly defined and controversial. As such, McClosky et al documented major crossmatch incompatibilities outside of the AB system in 23 of 154 transfusion-naive cats (14.9%). Similarly, Sylvane et al identified 10 of 52 major crossmacthes performed in transfusion-naïve cats to be incompatible (19%). However, the crossmatch screening of 112 cats in the United Kingdom failed to detect any non-AB incompatibilities. Similarly, the prevalence of non-AB RBC incompatibilities in previously transfused cats has been reported to be approximately 25-27%. While in some studies, the presence of NoAb do not appear to impact the safety and efficacy of transfusions, in other they are associated with less increase in post-transfusion PCV and increase number of transfusion reactions (mostly febrile). Based on the presence of NoAb, our research group at the Université de Montréal has recently begin mapping the corresponding feline erythrocyte antigens (FEA) behind these incompatibilities and identified five different putative FEA. FEA 1, 4 and 5 were most frequent with a prevalence of 84%, 65% and 96%, respectively. Only FEA 1 was significantly associated with NoAb ($P = 0.005$), which were observed in 8 of 43 FEA 1-negative cats (19%). Because of its prevalence and association with NoAb, FEA 1 may correspond to the lost Mik antigen. The immunogenicity of FEA 1 both in clinical and research settings were recently documented. Most recently, following experimental sensitization of a cat, a 6th FEA was identified.

Pre-Transfusion Compatibility Testing: Both AB-blood typing and crossmatching are recommended in all cats, even with the first transfusion as up to 19% of cats have naturally occurring antibodies against RBC antigens outside of the AB system. Point of care card agglutination and immunochromatographic tests are available for feline AB blood typing and crossmatching (DMS Laboratories and Alvedia).

References

- *Andrews GA et al. Blood. 1992;79(9):2485-91.*
- *Auer L, Bell K. Transfusion reactions in cats due to AB blood group incompatibility. Research in veterinary science. 1983;35(2):145-52.*
- *Bell K. The blood groups of domestic animals. In: Agar NS, Board PG, editors. Red Blood Cells of Domestic Mammals: Elsevier; 1983. p. 133-64.*
- *Bighignoli B et al. Cytidine monophospho-N-acetylneuraminic acid hydroxylase (CMAH) mutations associated with the domestic cat AB blood group. BMC Genet. 2007;8:27.*
- *Binvel M et al. Identification of five novel erythrocyte antigens based on the presence of naturally occurring alloantibodies. Journal of Veterinary Medicine, 2021; 35(1): 234-244.*
- *Gandolfi B et al. A Novel Variant in CMAH Is Associated with Blood Type AB in Ragdoll Cats. PloS one. 2016;11(5):e0154973.*
- *Gandolfi B et al. Correction: A Novel Variant in CMAH Is Associated with Blood Type AB in Ragdoll Cats. PloS one. 2018;13(3):e0194471.*
- *Giger U, et al. Frequencies of feline blood groups in the United States. Journal of the American Veterinary Medical Association. 1989;195(9):1230-2.*
- *Giger U, Akol KG. Acute hemolytic transfusion reaction in an Abyssinian cat with blood type B. Journal of veterinary internal medicine 1990;4(6):315-6.*
- *Goy-Thollot I et al. Detection of naturally occurring alloantibody by an in-clinic antiglobulin-enhanced and standard crossmatch gel column test in non-transfused domestic shorthair cats. Journal of veterinary internal medicine 2018.*
- *Hourani L et al. Evaluation of a novel feline AB blood typing device. Journal of feline medicine and surgery. 2014;16(10):826-31.*
- *Kehl A et al.Molecular characterization of blood type A, B, and C (AB) in domestic cats and a CMAH genotyping scheme. PloS one. 2018;13(9):e0204287.*
- *McClosky et al. Prevalence of naturally occurring non-AB blood type incompatibilities in cats and influence of crossmatch on transfusion outcomes. Journal of veterinary internal medicine 2018;32(6):1934-42.*
- *Omi T et al. Molecular Characterization of the Cytidine Monophosphate-N-Acetylneuraminic Acid Hydroxylase (CMAH) Gene Associated with the Feline AB Blood Group System. PloS one. 2016;11(10):e0165000.*
- *Sylvane B et al. Effect of cross-match on packed cell volume after transfusion of packed red blood cells in transfusion-naive anemic cats. Journal of veterinary internal medicine 2018;32(3):1077-83.*
- *Tasker S et al. Feline blood genotyping versus phenotyping, and detection of non-AB blood type incompatibilities in UK cats. The Journal of small animal practice. 2014;55(4):185-9.*
- *Weinstein NM, Blais MC, et al. A newly recognized blood group in domestic shorthair cats: the Mik red cell antigen. Journal of veterinary internal medicine 2007;21(2):287-92.*
- *Weltman JG et al. Influence of cross-match on posttransfusion packed cell volume in feline packed red blood cell transfusion. Journal of veterinary emergency and critical care (San Antonio, Tex : 2001). 2014;24(4):429-36.*

Therapeutic plasma exchange

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Learning objectives

- Understand what therapeutic plasma exchange is
- Understand how automated therapeutic plasma exchange is performed
- Know the indications for therapeutic plasma exchange in small animal patients
- Review the recent veterinary literature

Lecture summary

Apheresis is the process by which blood is removed from circulation and separated into components, one or more of which are removed or processed prior to returning the remaining blood to the patient. Plasmapheresis is specifically the selective removal of plasma that is not replaced. Therapeutic plasma exchange (TPE) is a type of apheresis in which the plasma is removed and replaced with other fluids. TPE can be performed using either manual or automated methods, with specialized machines performing automated TPE. Automated TPE can be performed using centrifugation or filtration methods. During the centrifugation method, blood components are separated in a rotating chamber based on density, with plasma being less dense than red blood cells. The blood in the extracorporeal circuit must be anticoagulated, usually with citrate. After separation, plasma is not returned to the patient while red blood cells, white blood cells, and platelets are. Filtration techniques vary from centrifugal in that a membrane with varying pore sizes separates plasma (or plasma components) for removal and leaves behind blood components and systemic anticoagulation is typically performed using unfractionated heparin. The lost plasma volume must be replaced to the patient and is typically done so using a combination of crystalloids and colloids. Both synthetic and natural colloids (canine albumin, human albumin, and fresh frozen plasma) can be used. At the author's institution, patients typically receive 1/3 of their replacement volume as isotonic crystalloid, 1/3 as synthetic colloid, and 1/3 as natural colloid (typically FFP). However, specific patient conditions (i.e. preexisting hypoalbuminemia) should be considered. In order to undergo TPE, patients must have a large bore multi-lumen jugular catheter placed. The patient must be instrumented to closely monitor heart rate, blood pressure and temperature. Additionally, acid base status, hemoglobin, and electrolytes (specifically ionized calcium) should be monitored frequently during the procedure. At the author's institution, calcium gluconate and magnesium sulfate are administered at set timepoints, or as needed based on patient parameters. Some patients require mild sedation, as the procedure can last up to several hours. The driving therapeutic principle behind TPE is that a pathologic or toxic substances in the plasma can be removed by removing the plasma from circulation. When deciding if TPE is appropriate, the volume of distribution (Vd) of the substance in question must be considered; substances with a low Vd (<0.5L/kg) are ideal. Additionally, only toxins that are highly protein bound (>80%) can be removed utilizing TPE. After steady state is reached, 97% of a toxin is eliminated after 5 half-lives. Therefore, time from ingestion to presentation must be considered for toxins with a short half-life. Typically, 1-2 plasma volumes are exchanged in a single treatment. Depending on the underlying disease, multiple sessions may be necessary with sessions being performed every other day to allow for redistribution of the substance into the intravascular space (such as immunoglobulins). The most commonly reported indications for TPE in veterinary medicine include adjunctive treatment of immune mediated hemolytic anemia (IMHA), myasthenia gravis (MG) and intoxications, although there are also reports of treating immune mediated thrombocytopenia, immune glomerulonephritis, polyradiculoneuritis, and hepatic encephalopathy among other conditions. In patients with non-associative IMHA, TPE can remove immunoglobulins in the intravascular space. TPE should never be utilized as the sole treatment and is generally considered when patients have failed traditional management or are severely affected. Both the literature and clinical experience support the use of TPE in these select patients, with TPE not being recommended for patients responding well to traditional therapy. Similarly, patients with MG may benefit from TPE when they fail traditional management or experience rapidly progressive disease or life-threatening complications such as respiratory depression. Multiple sessions may be required to see clinical improvement for patients with IMHA or MG. Intoxications treated by TPE reported in the veterinary literature include ibuprofen, carprofen, naproxen and meloxicam. By removing plasma, highly protein bound toxins are also removed, preventing or diminishing effects of the toxin. The body of literature on the use of TPE in veterinary patients is growing and supports its use in patients with severe overdoses of drugs that can be removed using TPE, if implemented in a timely manner. Complications reported to occur during TPE include clotting of the circuit, hypotension, hypocalcemia (with and without associated clinical signs), hemorrhagic complications, allergic reactions to FFP (urticaria, chemosis) and difficulties achieving adequate flow rates in very small patients. Approximately one third of patients undergoing TPE are reported to experience complications, although the vast majority of these tend to be mild and self-limiting.

References

- *Butty EM, et al. Outcomes of nonsteroidal anti-inflammatory drug toxicosis treated with therapeutic plasma exchange in 62 dogs. J Vet Intern Med. 2022 Sep;36(5):1641-1647.*
- *Chalifoux NV, et al. Outcomes of 434 dogs with non-steroidal anti-inflammatory drug toxicosis treated with fluid therapy, lipid emulsion, or therapeutic plasma exchange. J Vet Intern Med. 2023 Jan;37(1):161-172.*
- *Culler CA, Vigani A, Ripoll AZ, Gareau A, Suter SE. Centrifugal therapeutic plasma exchange in dogs with immune-mediated hemolytic anemia (2016-2018): 7 cases. J Vet Emerg Crit Care (San Antonio). 2022 Sep;32(5):645-652.*
- *Foster JD. Extracorporeal Therapies in the Emergency Room and Intensive Care Unit. Vet Clin North Am Small Anim Pract. 2020 Nov;50(6):1215-1236.*
- *Francey T, Schweighauser A. Membrane-based therapeutic plasma exchange in dogs: Prescription, anticoagulation, and metabolic response. J Vet Intern Med. 2019 Jul;33(4):1635-1645.*
- *Palm C. Small Animal critical Care Medicine, St. Louis, USA,: Elsevier, Saunders; 2015, 22; 1062-1064.*

Cell salvage applications in emergency and critical care

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Learning objectives

- Understand the different forms of cell salvage machines, and possible indications for their use
- Outline how cell salvage can affect blood resource management and implications on cost of care
- Review possible contraindications, sequelae and controversies related to cell salvage
- Recognise the technical aspects of administering cell salvaged products

Lecture summary

Introduction: Cell salvage techniques involve collection of shed blood, such as cavitary haemorrhage, before processing and reinfusion into the patient and can be used as part of a blood resource management solution, or in situations where no ready access to blood products exists. While cell salvage can be very low tech (such as autotransfusion without processing of the fluid, using only syringes as equipment) technology now exists which aims to improve the safety of this procedure. In human medicine cell salvage has become a vital part of managing patients undergoing major surgery with expected blood loss who decline red blood cell transfusions due to their faith. Although commonly used during surgical procedures where there is a broad recommendation to use such technology if it can reduce allogenic blood transfusion requirements (Klein et al., 2018) this technology has its place in the ER and ICU either in the perioperative setting, or as a standalone therapeutic intervention depending in the situation. Use of cell salvage technology is reported in the veterinary literature as part of intraoperative blood conservation (Hirst & Adamantos, 2012; Cole & Humm, 2019) and is commonly used at the authors' institution both in and outside of theatre.

Technology: There are two major technologies employed in cell salvage processing: centrifugal and membrane-based separation of shed blood into its component parts, with the former being the most common technology available. Centrifugal technology may also allow such machines to be utilised for therapeutic plasma exchange procedures further increasing their clinical utility for the critically ill patient population. Membrane based modalities are touted to retain clotting proteins, platelets and other molecules within the processed blood, while centrifugal modalities typically attempt to discard most components other than red blood cells, although some contamination with other molecules still occurs and this reinfusion via filters is recommended in all cases.

Contraindications and controversies: Contraindications in veterinary medicine are not well established, and instead guidelines from human medicine are often used. Any agent known to directly damage red blood cells or that may affect cardiovascular function on reinfusion should be avoided in the shed fluid, including dermal antiseptics, topical clotting agents, orthopaedic cement or fibrin based glues, and absolute contraindications are extremely sparse including lack of trained personnel or (for human medicine) refusal of the patient (Schmidbauer & Seyfriend, 2022). While some authors list bacterial contamination of the shed fluid as a relative contraindication numerous studies exist indicating this is less of a concern due to bacterial removal from both processing and filters during reinfusion (with up to 99% removal depending on assessment methodology). One study evaluated the ex vivo safety of shed vaginal blood and its suitability for re-infusion, determining it should be used (Tear et al., 2016) and bacterial contamination of shed blood even during sterile surgery has been noted in human medicine without concern. Even frank faecal contamination of the shed fluid does not appear to increase rates of sepsis in humans who receive such a product (Esper & Waters, 2012). Malignancy in the shed fluid has also been considered a concern historically, however data, including from meta-analyses, show no increased rates of adverse outcomes and in fact commonly show either benefit or no difference for cell salvage of malignant effusions. In the UK the body responsible for evidence-based treatment recommendations (NICE) recommend cell salvage for several oncologic surgeries (Carroll & Young, 2021), and there is currently no evidence to suggest diffuse metastases after reinfusion of such blood, although precautions (such as either leukoreduction filters or irradiation depending on the country) are recommended.

References

- *Klein, A.A., Bailey, C.R., Charlton, A.J., et al. Association of Anaesthetists guidelines: cell salvage for peri-operative blood conservation 2018, Anaesthesia, 2018, 73(9);1141-1150*
- *Hirst, C., Adamantos, S., Autologous blood transfusion following red blood cell salvage for the management of blood loss in 3 dogs with hemoperitoneum, J Vet Emerg Crit Care, 2012, 22(3);355-360*
- *Cole, L.P., Humm, K., Twelve autologous blood transfusions in eight cats with haemoperitoneum, J Feline Med Surg, 2019, 21(6);481-487*
- *Schmidbauer, S.L., Seyfried, T.F., Cell Salvage in the ICU, J Clin Medicine, 2022, 11(13);3848*
- *Esper, S.A., Waters, J.H., Intra-operative cell salvage: a fresh look at the indications and contraindications, Blood Transfus., 2010, 9(2);139-147*
- *Teare, K.M., Sullivan, I.J., Ralph, C.J., Is Cell Salvaged Vaginal Blood Loss Suitable for Reinfusion?, Obstetric Anesthesia Dig., 2016, 36(2);58-59 Carroll, C., Young, F., Intraoperative cell salvage, BJA Educ., 2021, 21(3);95-101*

Forward thinking nursing skills

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Learning objectives

- Attendees will learn about imposter syndrome
- Attendees will leave learning about techniques to help with critical thinking.
- Attendees will leave understanding how to improve the team's communication.
- Attendees will learn about M&M rounds

Lecture summary

What Forward thinking is NOT
"Just following Orders"
Yielding to tradition
It's always been done this way
Creative approaches are not tried
You become biased
You dislike the owners so you don't provide best care
You are right. All else is wrong.
You close the door to suggestions
You stop looking

Traits of a Forward Thinker
Inquisitive
Knowledge
Independence of Thought. Want to Learn
Self-Confidence
Open Mindedness & Flexibility to Consider Other Alternatives
Honesty
No Bias
Be Inquisitive
You cannot be the best technician for your patient if you do not:
Interpret: Understand & Explain the meaning of information or an event
Analyze: Investigate a course of action that is based on data
Evaluate: Assess the information you receive and determine if a reaction is needed
Constantly think WHY? Knowledge is power and is the first step of forward thinking skills. The more you know the better for the pets You can't think ahead if you don't know what you don't know
QUESTION
GET THE ANSWER
APPLY IT
Skills You Must Have to Critically Think
A good understanding of normal vs abnormals (bloodwork, TPR, etc) is imperative in helping you stay grounded in what you do know. Being sure you have the ability to react and voice concern is equally important. All the knowledge won't help if you never speak up. Invest in a good stethoscope so you can monitor patients well. Pay attention to detail and seek answers when you don't know something.
Embrace your inner nerd! Read books, journals and articles. Watch webinars or quick video clips and listen to podcasts. The nerdy the better! You will get the best forward thinking skills if you investigate everything your brain asks as "why".
Self-Confidence

One of the main reasons veterinary technicians fail to have forward thinking skills is because of imposter syndrome. Impostor syndrome (IS) refers to an individual believing that they are not as competent as others may perceive them to be. Ultimately IS is linked to the individual's perception of how society views them. Imposter syndrome effects every person on the planet no matter their social status, work background, skill level, education or expertise.. One study estimated that 7 in 10 adults experience IS at some point in their life.
Imposter Syndrome Results In
Self-doubt
Attributing your success to external factors
Never allowing yourself to celebrate your wins
Fear that you won't live up to expectations
Overachieving
Sabotaging your own success
Anxiety due to a fear of failing
Depression because of a perceived failure
Identification is Key
Do you agonize over any mistake? Big or small?
Do you feel you've never earned anything on your own?
Do you feel like you will eventually be seen as a phony or fraud?
Do you downplay your own expertise? Are you embarrassed when someone says "great job" to you?
Do you feel you are underserving of success?
Do you fail to pursue promotions or opportunities because you feel you aren't good enough?
Recognizing you are experiencing from IS is the first step to you taking steps to creating a better outlook about yourself.

Be Honest
Be sure you understand your own limitations and errors. Get comfortable with the phrases:
I Don't Know
Maybe You Are Right
That's Not My Area of Expertise
I Believe You May Be Wrong
Not you are wrong…start with the "I" statement
Recognize and Acknowledge Your Own Bias
That's right. Unfortunately, veterinary professionals also have bias. We have them against certain breeds, species and our human clients.
Veterinary biases because medical professionals to not offer the best care or treatment for a particular patent because they have a bias in their mind that a client will not be able to afford it. They may withhold sending home pain medication because they believe that the client may use it for themselves. They may believe that the client is making up symptoms because they believe that a certain race or gender is more prone to lying. While it's hard to imagine it happening it does. Every person on the planet holds biases. It's important that when we are practicing medicine we look to keep ourselves in check.
TO SUCCEED:
Pay Attention
Come in to work clear minded!
Communicate What Your Patient Needs
Own Your Patient
It's a team approach at ALL times
It's not the doctor's patient
You don't just follow orders

The septic patient; an interactive case discussion

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Learning objectives

- True understanding of pathophysiology of sepsis
- Application of ability to pick up trends and parameters to anticipate disease process
- Development of emergency and critical care thinking skills
- Mastery of intensive nursing skills required for septic patients

Lecture summary

Pathophysiology: Sepsis is defined as a dysregulated inflammatory response to infection and/or trauma. Normally, an infection or tissue injury triggers a local immune response. In some patients, however, a systemic spillover of inflammatory mediators triggers a more generalized reaction. The inflammatory response may initially start locally (e.g., abscess on a limb) but, if severe, can progress to cause systemic signs when mediators of inflammation enter the circulatory system and instigate global activation of the inflammatory system. The body's normal response to pathologic inflammation balances the activation of proinflammatory mediators with activation of anti-inflammatory mediators. During gram-negative sepsis there is an activation of macrophages, a transcription of cytokines and generation of anti-inflammatory cytokines. Dysregulated by cytokine release, tissue factor levels are upregulated to initiate the coagulation cascade, leading to the haemostatic balance favouring a procoagulant state. Other cellular processes in response to the immune triggers, result in vasodilation, apoptosis, cytokine storm, inflammation, and tissue damage. Endothelial injury occurs from microcirculatory derangements that increase vascular permeability.

Biomarkers and Clinical Manifestations: Currently, there is no ideal and clinical gold standard for the diagnosis of sepsis, as microbiology may not be sensitive enough and laboratory tests are unspecific for use as a reference standard. However, monitoring septic patients has traditionally been achieved by measuring physiologic parameters. Sepsis markers of note that can also be used to track trends easily within the general veterinary hospital, include activated partial thromboplastin time (aPTT), troponin and lactate.

Patients presenting with sepsis often have a nonspecific medical history. Clinical signs include lethargy, weakness, hyporexia or anorexia, abdominal discomfort or abnormal posturing, vomiting, diarrhoea, increased respiratory rate and/or effort, fever, erythema, swelling of extremities and/or joints (possible limping), abnormal wounds, changes in heart rate, change in respiration, injected mucous membrane colour, rapid capillary refill time, and altered level of consciousness. The clinical presentation of sepsis will vary depending on the severity and stage of the disease process. Physical parameters in early sepsis will reveal the following: fever; tachycardia; tachypnoea; brick-red or muddy mucous membranes; rapid capillary refill time (<1 second); bounding pulses; and mental depression. As the sepsis progressively worsens, cardiac output is decreased. Vascular leakage and vasodilation will result in decreased venous return and decreased cardiac output leading to decompensated sepsis or 'hypodynamic' septic shock.

SOFA Scoring: Sequential Organ Failure Assessment (SOFA) scoring is one of the most commonly organ dysfunction scoring systems in human medicine. By using markers commonly used to assess critically ill patients such as coagulation (thrombocytopaenia), hypoxaemia (measuring lung dysfunction), brain dysfunction (Glasgow coma score), liver dysfunction (increased total bilirubin concentration), heart and kidney dysfunction (hypotension and increased creatinine concentrations, respectively), it can predict potential outcomes. The SOFA score should be noted at admission and then repeated throughout the hospital stay to see progression or initiation of organ injury.

Treatment Bundles: Patients with SIRS or sepsis require immediate stabilisation and treatment. It is recommended that treatment be centred on fluid resuscitation, antimicrobial therapy, infectious source control, and overall supportive care (e.g., pain control, nutrition). A "bundle of care" refers to a group of therapies that, when initiated together, produce better results than if initiated alone and has been shown to decrease morbidity and mortality in human studies by prioritising and standardise sepsis protocols. Early goal-directed therapy was first proposed in human medicine in 2001, with the aim to ensure adequate tissue oxygenation and survival. Parameters that should be monitored intensively and managed aggressively to specified targets: Central venous pressure (CVP), Mean arterial blood pressure (MAP), Urine output, Mixed venous oxygen saturation (SvO2) and Haematocrit

Commonly, SIRS/sepsis patients require continuous electrocardiography monitoring and frequent blood pressure measurement. For those patients with respiratory system compromise, which will be flagged on any sequential SOFA scoring, oxygen therapy and monitoring of additional respiratory parameters (e.g., respiratory effort, SpO2, arterial blood gas) may be indicated. Urinary catheters require care every 6 to 8 hours to prevent a secondary infection. Feeding tubes require daily maintenance to ensure patency. In patients with DIC, blood component therapy with fresh frozen plasma may be indicated and require a transfusion with frequent monitoring. Postoperative patients may have wound drains that need to be quantified and maintained.

References:

- *Evans, L. et al (2021) Executive summary: Surviving sepsis campaign: International guidelines for the management of sepsis and septic shock 2021: Erratum, Critical Care Medicine, 50(4).*
- *Foreman, E. (2020) Nursing the sepsis patient - part 1, Veterinary Nursing Journal, 35(8), pp. 242-245.*
- *Foreman, E. (2020) Nursing the sepsis patient- part 2, Veterinary Nursing Journal, 35(9-12), pp. 372.*
- *Gray, K. (2017) The Veterinary Nurse's role in recognising sepsis, The Veterinary Nurse, 8(5), pp. 259. Montealegre, F. and Lyons, B.M. (2021) Fluid therapy in dogs and cats with sepsis, Frontiers in Veterinary Science, 8.*
- *Nesbitt, I. (2008) Guidelines, protocols, and the surviving sepsis guidelines: A critical appraisal, Sepsis, pp. 87-92.*
- *Surviving sepsis campaign international guidelines (2020) Pediatrics, 145(5).*

Ischemia and reperfusion injury

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Learning objectives

- Attendees will learn what ischemia is
- Attendees will learn what reperfusion is
- Attendees will learn about I/R injury and what causes it
- Attendees will learn about treatment options for I/R injury

Lecture summary

The Ischemic Cascade: The chain of events involved in I/R injury can be broken down into the ischemic cascade and reperfusion injury. An ischemic episode involves a series of events called *the ischemic cascade*. Within 5 minutes of the development of ischemia, the electrolyte balance within cells becomes disturbed. The ischemic cascade usually continues for 2 or 3 hours but can last for days, even after perfusion is restored to the affected area. The term *cascade* suggests that events follow a sequential pattern, which is not true of the ischemic cascade. Events can occur simultaneously and do not always occur in a linear pattern. To fully understand the ischemic cascade, it is important to consider adenosine triphosphate (ATP) and how it functions. ATP is a multifunctional nucleotide (a structural factor of DNA and RNA) that is considered to be the most important nucleotide responsible for transporting energy for metabolism within cells. One of the fastest ways that ATP is produced is by oxidative phosphorylation, implying that ATP production requires oxygen. Despite the importance of ATP, cells do not stockpile ATP. They only make what they need for a particular time. When ischemia occurs, oxygenation of cells ceases, resulting in anaerobic ATP production, which is less efficient. When oxygen becomes unavailable to cells, anaerobic glycolysis starts. This can be a lifesaving way for cells to obtain energy; however, this process is extremely wasteful. During the anaerobic process, pyruvic acid and hydrogen atoms combine with nicotinamide adenine dinucleotide (NAD) to form NADH and H^+. If the buildup of NADH and H^+ becomes too great, the anaerobic process stops, thus terminating energy production to maintain cells. However, NADH and H^+ combine to form lactic acid, which diffuses from cells rapidly so that the process can continue. Although this is not ideal, the body can safely continue this process for several minutes. If the process continues for too long, as in ischemia, lactic acid can build up, indicating worsening illness. As a consequence of lactic acidosis, pH decreases, injuring and inactivating mitochondria. Some researchers think that lactic acid may also interfere with the recovery of aerobic ATP production after ischemia. For all ischemic patients, a lactate level should be obtained. Values <2 mmol/L are normal. When ATP fails to form, cells become depolarized, allowing calcium and sodium (normal extracellular electrolytes) to enter cells. Potassium, which is normally found in cells, leaks rapidly into the extracellular space. Excessive calcium overexcites cells, creating free radicals and many calcium-dependent enzymes. The extent of ischemic damage is related to the amount of calcium that enters cells and the duration of time that the intracellular calcium level remains elevated. The longer calcium stays in cells, the greater the amount of harmful chemicals will be created. One of the most important events involving calcium is the conversion of xanthine dehydrogenase (XDH) to xanthine oxidase (XO). XO requires oxygen for activation. During ischemia, oxygen is not present, so XO accumulates without getting used. Later, during reperfusion, XO can damage cells. As the mitochondria break down, they release toxins, causing apoptosis. Apoptosis is the body's way of safely disposing of dead cell parts by autolysis (self-destruction) of cells. Another important event during ischemia is the activation of nuclear factor-kB (NFkB), leading to the production of inflammatory mediators. NFkB becomes activated during stress. NFkB activates inflammatory cytokines and their receptors as well as platelet-activating factor. This allows neutrophils to enter through the vascular endothelium. Activated neutrophils are generally more rigid and stiff because of hypoxia and acidosis, which accompany ischemia. Because of the alteration of the cell membrane and the high number of neutrophils, capillaries may become plugged or clogged by neutrophils. Even after reperfusion, the redistribution of blood to affected areas may not produce enough force to clear clogs.[7] The full pathway of NFkB is still not understood.

Reperfusion Injury: It would seem that simply reintroducing oxygen into the affected area would be beneficial. In patients with GDV, oxygen is restored when the stomach is decompressed or untwisted, allowing oxygen and blood to flow back into the stomach wall. However, the reintroduction of oxygen into affected areas initiates a complex chain of events. Despite the harsh effects of ischemia alone, they do not cause nearly as much damage as reperfusion does. The longer the onset of the ischemic event, the greater the insult from reperfusion injury. Due to small word count contact the author for the full lecture notes: VetTeamTraining@Gmail.com

Nursing the acute haemorrhagic diarrhoea syndrome (ahds) case

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Learning objectives

- Understand the aetiology of AHDS
- Diagnosis and treatment of the disease
- Specific nursing considerations for the AHDS patient

Lecture summary

Introduction: The term Acute Haemorrhagic Diarrhoea Syndrome (AHDS), formerly known as Haemorrhagic Gastroenteritis (HGE), essentially means inflammation of the stomach and intestines with bleeding present. This kind of inflammation can be seen in patients with inflammatory bowel disease, pancreatitis, parvovirus, or any number of other conditions but when the term AHDS is used, it refers to a more specific canine syndrome which is an entity on its own. This syndrome commonly affects young to middle-aged, small breed dogs, though any dog can be affected. It's characterised by the per acute onset of vomiting and diarrhoea with blood, which can lead to life-threatening dehydration and hypovolaemia.

Diagnosis: AHDS is a diagnosis of exclusion. A thorough history and clinical examination are the first steps for investigating a suspected case of AHDS. Similar clinical signs can be caused by infectious diseases, toxin ingestion or dietary indiscretion. Findings on the clinical examination such as cachexia, evidence of coagulopathies, abdominal pain or organomegaly may increase the suspicion of another clinical disease causing the haemorrhagic diarrhoea. Initial tests would include PCV and total solids, complete blood count and smear, serum biochemistry, electrolyte, and venous blood gas analysis as well as coagulation testing. Other differentials can be ruled out via further testing such as abdominal imaging, faecal analysis, and specialised laboratory tests.

Treatment: Fluid therapy is the cornerstone of treatment in AHDS patients. If signs of hypovolaemia are present i.e. tachycardia, hyperdynamic or poor peripheral pulses and pallor, initial crystalloid fluid therapy with boluses is recommended. These are guided by blood pressure measurements and an improvement in cardiovascular parameters. If hypovolaemia is not present, the level of dehydration should be assessed and a fluid plan to correct this should be initiated. The aim is to correct any fluid deficits over 12-48hrs as well as allowing for estimated maintenance requirements and ongoing losses. In most patients, a balanced electrolyte isotonic crystalloid, such as Hartmann's solution, is an appropriate first choice. However, some AHDS patients can be suffering from a severe hypoproteinaemia and re-establishing and maintaining euvolaemia can be challenging due to third spacing and ongoing losses. In these patients, products such as frozen plasma can be considered for their crystalloid sparing effects. Otherwise vasopressors, such as noradrenaline, can be used to maintain blood pressure. Abdominal pain is often encountered in AHDS patients but often underappreciated in patients whose mentation is affected. Multimodal analgesia should be administered, including opioid pain relief such as methadone. NSAID's should be avoided in all animals with gastrointestinal signs, however, other agents such as paracetamol may have a role to play in the management of abdominal discomfort. Medication to treat nausea, such as maropitant and ondansetron, play an important role in AHDS patients. Prokinetics, i.e. metoclopramide, can be administered if the patient develops ileus and where haematemesis has been noted, proton pump inhibitors i.e. omeprazole or pantoprazole can be considered. Historically, antibiotics i.e. metronidazole and amoxicillin/clavulanic acid have been used for a multitude of reasons, however, repeated clinical studies have failed to demonstrate any significant benefit to the administration of antibiotics for dogs with AHDS.

Nursing considerations: Early enteral nutrition is vital in these patients to improve gut motility and intestinal cell healing. If a patient remains anorexic despite the administration of antiemetics and prokinetics, then a feeding tube should be considered. Naso-oesophageal or naso-gastric tubes are a cost-effective and easy approach to providing enteral nutrition. Naso-gastric tubes have the added benefit of being able to remove gastric fluid to further relieve discomfort and nausea. Feeding can be initiated slowly until full energy requirements are met. AHDS patients often suffer from severe faecal scalding around the perianal area. This scalding can be very uncomfortable resulting in inflammation, redness and occasionally infection. Perianal care is vital in these patients and the use of barrier creams and/or faecal management systems should be considered to prevent this uncomfortable side effect. Patients with AHDS require close monitoring to ensure their fluid demands are being appropriately met. Dependant on the severity of the disease, these patients may be recumbent and require more intensive nursing care i.e. regular turning, physiotherapy, oral and ocular care.

Conclusion

AHDS is a common emergency presenting to veterinary practice which, if left untreated, can be life-threatening. However, with supportive care, close monitoring and appropriate nursing, dogs with AHDS have an excellent prognosis.

References

- *Boag A and Hughes D (2007). Fluid therapy. In King L and Boag A (eds), BSAVA Manual of Emergency and Critical Care (2nd edn), BSAVA, Gloucester: 30-45.*
- *Eirmann L and Michel K (2009). Enteral nutrition. In Silverstein D and Hopper K (eds), Small Animal Critical Care Manual (2nd edn), Elsevier Saunders, Missouri: 681-686.*
- *Ortiz V, Klein L, Channell S, Simpson B, Wright B, Edwards C, Gilbert R, Day R and Caddy SL (2018). Evaluating the effect of metronidazole plus amoxicillin-clavulanate vs amoxicillin-clavulanate alone in canine haemorrhagic diarrhoea: a randomised controlled trial in primary care practice, Journal of Small Animal Practice 59(7): 398-204.*
- *Trotman TK (2009). Gastroenteritis. In Silverstein D and Hopper K (eds), Small Animal Critical Care Manual (2nd edn), Elsevier Saunders, Missouri: 622-625.*
- *Unterer S, Strohmeyer K, Kruse BD, Sauter-Louise C and Hartmann K (2011). Treatment of aseptic dogs with hemorrhagic gastroenteritis with amoxicillin/clavulanic acid: a prospective blinded study, Journal of Veterinary Internal Medicine 25(5): 273-279.*
- *Walsh K (2019). Companion animal analgesia, Vet Times 49(24): 6-10.*
- *Weeth L (2016). Managing food refusal in critical care patients, Vet Times 46(2): 20-22.*

Non-technical skills for safer emergency care

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Learning objectives

- To comprehend the importance of non-technical skills.
- To understand the basic non-technical skills identified as important in all safety critical industries.
- To explore tools that support the use of non-technical skills and prevent error in clinical practice.

Lecture summary

In 2019, the World Health Organisation (WHO) reported that in high income countries it is estimated that one in ten patients are harmed whilst receiving hospital care (Slawomirski, Auraaen and Klazinga, 2017), that a range of adverse events (such as retained swabs or wrong site surgery) are known to cause this harm and that nearly 50% of adverse events could be prevented (De Vries *et al.*, 2008a). Over the past 30 years, research within the medical profession, has focused on developing an understanding of the types of errors which occur, where they arise and what causes them. The discipline of Human Factors seeks to understand the organisational, individual, environmental and job characteristics that influence behaviour in ways that can impact safety (CHFG, 2021). One of the key domains within Human Factors is non-technical skills. Mitchell and Flin, (2008) describe non-technical skills as the "cognitive and social skills that complement technical skills to achieve safe and efficient practice" and list seven basic non-technical skills which are typically required in the clinical setting; situational awareness, decision making, communication, teamwork, leadership, managing stress and coping with fatigue. When investigating the non-technical skills (NTS) required by surgeons in the operating room, Yule *et al.*, (2006) found that whilst technical skills are necessary, many causes of adverse events in human healthcare derive from non-technical aspects of performance, rather than lack of technical expertise and that to maintain high levels of performance over time within surgery, adequate attention needs to be paid to them. For each occupational domain the non-technical skills required must be identified and a framework customised to reflect the clinical tasks, workplace conditions and organisational/professional culture (Flin, 2013). Following research within each domain to establish non-technical skill's taxonomies, behavioural marker systems (BMS) for the evaluation of non-technical skills have been developed for Anaesthetists (ANTS) (Fletcher *et al.*, 2003), Anaesthetic Practitioners (ANTS-AP) (Rutherford et al, 2015), Surgeons (NOTSS) (Yule *et al.*, 2006) and Scrub Practitioners (SPLINTS) (Mitchell *et al.*, 2012) within human healthcare. Behavioural marker systems offer opportunities to accurately assess, train and measure the impact of non-technical skills (Dietz *et al.*, 2014) and comprise of a skill taxonomy, with defined skill categories, elements and behaviours, together with user advice and a rating scale for assessment (Flin and Martin, 2001). Unlike other safety critical industries such as aviation and healthcare very little research has been conducted into the behavioural components which may affect clinical veterinary practice. There is a paucity of research into non-technical skills in veterinary medicine and consequently no non-technical skills frameworks or behavioural rating systems exist within this field. Within the veterinary profession, patient safety culture is described as being embryonic (McMillan, 2014) and despite improvements in clinical governance, with the inclusion of clinical audits in the Royal College of Veterinary Surgeons Code of Professional Conduct (RCVS, 2021a) and their Practice Standards Scheme (RCVS, 2021b) there are still no compound mortality and morbidity figures (Oxtoby *et al.*, 2015). However, research has begun that enables parallels between human and veterinary healthcare to be identified. De Vries *et al's* work in 2008 was mirrored by Oxtoby *et al.*, (2015) whose research suggested that errors involving surgery were the type of error most frequently reported through veterinary healthcare insurance claims. Oxtoby *et al.*, (2015) concluded that there are "many similarities between the veterinary profession and other safety critical industries" with cognitive limitations being found to account for a large proportion of reported errors. Oxtoby *et al.*, (2015) also found that deficiencies in non-technical skills such as communication, leadership and teamwork contributed to errors, poor patient outcomes and quality of care. Through understanding the non-technical skills required in the veterinary emergency care setting we can implement strategies that make it easier for people to 'do the right thing', therefore reducing potential error and improving patient care. In this session we will explore the evidence-based tips and tools which can be practically applied within the ultra-adaptive, veterinary emergency care setting.

References

- *Fletcher, G. et al. (2003) 'Anaesthetists' Non-Technical Skills (ANTS): evaluation of a behavioural marker system † †Declaration of interest: The ANTS system was developed under research funding from the Scottish Council for Postgraduate Medical and Dental Education, now part of NHS', British Journal of Anaesthesia. doi: 10.1093/bja/aeg112.*
- *Flin, R., O'Connor, P. and Crichton, M. (2013) Safety at the sharp end: A guide to non-technical skills, Safety at the Sharp End: A Guide to Non-Technical Skills.*
- *Flin, R. (2013) Non-technical skills for anaesthetists, surgeons and scrub practitioners (ANTS, NOTSS and SPLINTS). Available at: www.abdn.ac.uk/iprc/ANTS Oxtoby, C. et al. (2015) 'We need to talk about error: Causes and types of error in veterinary practice', Veterinary Record. doi: 10.1136/vr.103331.*
- *McMillan, M. (2014) 'New frontiers for veterinary anaesthesia: The development of veterinary patient safety culture', Veterinary Anaesthesia and Analgesia. Blackwell Publishing Ltd, pp. 224-226. doi: 10.1111/vaa.12123.*
- *Mitchell, L. and Flin, R. (2008) 'Non-technical skills of the operating theatre scrub nurse: Literature review', Journal of Advanced Nursing. doi: 10.1111/j.1365-2648.2008.04695.x.*
- *RCVS (2021a) Code of Professional Conduct for Veterinary Surgeons - Professionals. Available at: https://www.rcvs.org.uk/setting-standards/advice-and-guidance/code-of-professional-conduct-for-veterinary-surgeons/*
- *RCVS (2021b) Practice Standards Scheme - Professionals. Available at: https://www.rcvs.org.uk/setting-standards/practice-standards-scheme/*
- *RCVS (no date) Day One Competences for Veterinary Nurses - Professionals. Available at: https://www.rcvs.org.uk/document-library/day-one-competences-for-veterinary-nurses/*
- *Rutherford, J.S., Flin, R., Irwin, A. and McFadyen, A.K., 2015. Evaluation of the prototype anaesthetic non-technical Skills for Anaesthetic Practitioners (ANTS-AP) system: a behavioural rating system to assess the non-technical skills used by staff assisting the anaesthetist, Anaesthesia, 70(8), pp.907-914.*
- *Slawomirski, L., Auraaen, A. and Klazinga, N. (2017), The Economics of Patient Safety; strengthening a value-based approach to reducing patient harm at national level.*
- *De Vries, E. N. et al. (2008a), The incidence and nature of in-hospital adverse events: A systematic review, Quality and Safety in Health Care. doi: 10.1136/qshc.2007.023622.*
- *De Vries, E. N. et al. (2008b), The incidence and nature of in-hospital adverse events: A systematic review, Quality and Safety in Health Care, 17(3), pp. 216–223. doi: 10.1136/qshc.2007.023622.*
- *Yule, S. et al. (2006), Non-technical skills for surgeons in the operating room: A review of the literature, Surgery. doi: 10.1016/j.surg.2005.06.017*

Low flow - provision of oxygen for patients in respiratory distress

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Learning objectives

- Describe hypoxaemia
- Consider methods of providing oxygen to potentially stressed, dyspnoeic patients
- Evaluate nursing considerations for the hypoxaemic patient
- Choose suitable sedation regimens depending on your patient's requirements
- Understand the technique of placement of indwelling nasal cannulae

Lecture summary

Hypoxaemia is defined as "deficient oxygenation of the blood" with a PaO_2 of <80mmHg or SpO_2 <95%. The primary causes of hypoxaemia are low inspired oxygen concentration, hypoventilation, and venous admixture (ventilation/perfusion (V/Q) mismatch, right-to-left shunts, diffusion impairment). Arterial blood gas (ABG) measurement is the gold standard for assessing oxygenation and ventilation and is a measure of the partial pressure of oxygen (PaO_2) and carbon dioxide ($PaCO_2$) in the arterial blood. The fraction of inspired oxygen (FiO_2) describes how much oxygen is in a mixture of gases, so for example, room air contains 21% oxygen, thus its FiO_2 is 21mmHg (or 0.21). As a general rule of thumb the PaO_2 should be approximately five times that of the FiO_2 at sea level. For a patient at sea level on room air then, we should expect their PaO_2 to be around 100mmHg as 21(%) x 5 ≈ 100(mmHg). A 'normal' PaO_2 is considered anything between 80-110mmHg. A patient with a PaO_2 <80mmHg is classed as hypoxaemic, while patients with a PaO_2 <60mmHg are deemed to be suffering from severe, potentially life-threatening, hypoxaemia.

Methods of providing oxygen include flow-by oxygen, oxygen chambers, nasal prongs, cannulae and masks. For me, the starting point is always flow-by oxygen as it doesn't require you to touch the patient. Flow-by provides around 30% FiO_2, but this depends on how the patient is breathing and whether they tolerate the flow. Oxygen chambers also allow for a hands-off approach and can provide a FiO2 of up to 80%. Prongs provide around 40% FiO_2, and nasal cannulae delivering between 40-80% FiO_2, as they terminate deeper in the nasal passages and tend to stay in place better. Masks would be my last choice in most situations. Whilst good at providing a generous FiO_2 of around 50-60%, they are not well tolerated by most patients. They can be useful in sedated or moribund patients, but you need to remember to leave a gap to prevent rebreathing of expired CO_2, so removal of the rubber diaphragm is advised.
Nursing considerations: reduction of stress is the number one priority for patients in respiratory distress as they may be at the limit of their physiological reserves. Restraint leads to stress, so this should be avoided where possible or minimised with a well-considered approach. At rest, the percentage of oxygen used on the 'work of breathing' is around 5%, whereas in dyspnoeic animals, this can increase to 70%! The 'work of breathing' increases massively due to:

- recruitment of additional muscles e.g. abdominal
- onset of active expiration - usually this is a passive process using recoil of the lungs and doesn't require any energy

PLUS there is the potential for less efficient gas exchange if the patient has pulmonary disease. If patients are in an oxygen chamber, continuous oxygen provision is difficult as you have to open the door to nurse the patient. Keep this in mind when creating your nursing plan to limit the number of times you need to access the patient. Also, don't forget that heat is generated as part of the 'work of breathing', meaning heat and humidity will increase in an enclosed area, which you will need to monitor and control. It is also important to consider ocular care for patients in chambers, as well as how and when to provide exercise or toileting opportunities. Nasal insufflation via prongs or cannulae is much more practical if appropriate, as you can nurse your patient without worrying about opening the doors! Sedation is often required in patients requiring oxygen therapy and is an important tool for relief of anxiety. This can lead to a reduction in oxygen demand for the patient as stress levels decrease. Butorphanol, acepromazine and dexmedetomidine can be considered and given as bolus injections I/M or IV as well as via a CRI. If the patient is eating, there is the option to add in gabapentin and/or trazodone to further help ease patient stress.

Placement of indwelling nasal cannulae is extremely useful for patients requiring long term oxygen therapy. Use of a soft, fenestrated tube is encouraged to ensure oxygen delivery doesn't result in trauma to the nasal mucosa, with local anaesthetic instilled into the nasal passages 10 minutes prior to placement. Measurement for depth of placement is from the alar fold to the medial canthus of the eye and once lubricated, the tube should be inserted ventromedially to avoid hitting the nasal turbinates. The tube can be secured using a fingertrap suture pattern.

References

Silverstein D.C., Small animal critical care medicine, St. Louis, USA,: Elsevier, Saunders; 2015, 15; 81-85.

Acute respiratory distress syndrome and acute lung injury

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Learning objectives

- Attendees will learn about defining how severe respiratory distress is.
- Attendees will learn how to diagnose ARDS and ALI
- Attendees will learn about treatment options of respiratory distress.
- Attendees will learn about nursing care of respiratory distress patients.

Lecture summary

Pathophysiology

Since they were first termed, many efforts have been made to fully understand acute respiratory distress syndrome (ARDS) and acute lung injury (ALI). The exact triggering mechanism of ARDS or ALI is not completely understood in human medicine, and even less research on the subject has been done in veterinary medicine. Dogs have been used as research models for human studies, which allows some insight into the syndromes. It is unknown if cats develop ARDS or ALI in a similar manner to dogs. Research regarding cats is far more limited. Many researchers suggest that ARDS and ALI do exist in the cat, but not by standards which currently define the syndromes in other species. This could possibly be because common risk factors (such as sepsis, pneumonia and non-fatal thoracic trauma) occur less in cats.

Only a small percentage of oxygen is actually dissolved into a physical form that it can be read in plasma. Some blood gas analyzers will offer the reading as PO_2. It is important to know if the sample was venous or arterial or the reading could be misinterpreted. Normal PaO_2, at sea level, is between 80 and 110 mmHg. When PaO_2 is less than 80 mmHg the patient is suffering from hypoxemia. Severe hypoxemia occurs with a PaO_2 less than 60 mmHg. In general, the PaO_2 should be approximately five times the FiO_2. Therefore a patient under anesthesia on 100% oxygen should have a PaO_2 of approximately 500 mmHg. Room air has a FiO_2 0.21. Therefore a patient under anesthesia on 100% oxygen should have a PaO_2 of approximately 500 mmHg. Room air has a FiO_2 0.21. Therefore a normal PaO_2/FiO_2 ratio is around 476 (100/0.21). If an arterial blood gas is performed and the patient has a PaO_2 of 59 mmHg on room air, the PaO_2/FiO_2 ratio is 280. Once an arterial blood gas analysis is performed, if safe and appropriate, chest imaging should be taken. One of the veterinary criteria for diagnosing ARDS is that there should be evidence of pulmonary capillary leak that was not associated with increased capillary pressures. While CT scanning may lead to more accurate images of the pulmonary parenchyma, it serves as no greater a diagnostic tool than a radiograph. With the onset of ALI or ARDS, bilateral alveolar infiltrates will be seen. There are other diseases that cause alveolar infiltrates. To diagnose a patient as having ARDS/ALI it is important to complete a thorough work up as well as obtain an accurate history from the owners.

Initial Treatment: Stress should be minimized to any animal in respiratory distress. Restraint, in most cases, should be minimal. Some cats will become more fractious and require immediate sedation so that you can safely work with them. Animals should be allowed to assume any position that provides them the most relief. Oxygen should be provided initially by the least-stressful route. Long term oxygen therapy can include the use of oxygen cages, nasal oxygen lines or a mechanical ventilator amongst others.

Monitoring Oxygenation: It is imperative to constantly assess oxygenation in ARDS/ALI patients. In a clinical setting we can monitor the four parameters: mucous membrane color, PvO_2, PaO_2 and SpO_2. The goal in administering oxygen should be to maintain an adequate PaO_2 while allowing for the lowest FiO_2 possible (room air 0.21). Mucous membrane color is one of the easiest parameters to monitor and should be part of every physical exam. Though not completely accurate (because poor lighting, anemia or icterus hides the appearance of cyanotic membranes) any presence of cyanosis indicates a life threatening oxygenation issue which needs to be addressed immediately.

References

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Gary stamp memorial lecture: Mechanical ventilation 101

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Learning objectives

- Know the 3 main indications for putting an animal on mechanical ventilation
- Become familiar with the major ventilator settings and how they are determined
- Understand the signs of ventilator patient distress

Lecture summary

Mechanical ventilation is becoming more common in veterinary intensive care and can be performed with anesthesia ventilators or the use of dedicated ICU ventilators. The most important aspect of successful ventilator patient management is the training and dedication of the nursing care team.

Indications: There are three main reasons to place patients on a ventilator. • Inability to oxygenate (hypoxemia) despite therapy (PaO_2<60mmHg or SpO_2 < 90%, despite administration of oxygen) • Inability to ventilate (hypoventilation) despite therapy ($PaCO_2$ > 60 mmHg despite therapy) • Excessive breathing effort. Once an animal is determined to require mechanical ventilation, the machine needs to be set up with a clean circuit, initial machine settings need to be entered, an appropriate patient bed with extra padding is needed and all the relevant monitoring equipment should be ready to go. If the animal needs emergent intubation, that should be performed quickly and the animal hand ventilated while the ventilator and related equipment is set up.

Ventilator settings: The major ventilator settings that need to be determined before starting an animal on the ventilator are the following: • Fraction of inspired oxygen (FiO_2) • Tidal volume or Inspiratory pressure • Respiratory rate • Positive end expiratory pressure (PEEP) • Inspiratory time • Inspiratory to Expiratory ratio • Inspiratory trigger.

Once the animal is established on the ventilator, arterial blood gas analysis is performed, if possible. In the absence of arterial blood gases, evaluation is based on venous blood gas and pulse oximetry. Ventilator settings are then titrated to achieve the blood gas goals.

Blood gas goals: A major aim of mechanical ventilation is normalization of the oxygenation and carbon dioxide levels. Frequent measurement of the partial pressure of arterial oxygen (PaO_2) and arterial carbon dioxide ($PaCO_2$) is ideal. Ventilator settings are adjusted to target the following blood gas values. PaO_2 of 80 - 120 mmHg (SpO_2 > 95%). In severe lung disease, tolerating lower PaO_2 values (55 - 80 mmHg; SpO_2 > 88%) maybe preferable to increasing ventilator settings. $PaCO_2$ of 35 - 55 mmHg (35 - 40mmHg in patients with brain disease). In severe lung disease, tolerating higher $PaCO_2$ values (50 - 70 mmHg) is often preferable to increasing ventilator settings.

PaO_2: Once the patient appears to be stable on the ventilator, assessment of arterial blood gases is performed. The first priority is to lower the FiO_2 to ≤ 60% while maintaining an acceptable PaO_2. Positive end expiratory pressure (PEEP) can increase the oxygenating efficiency of the lung. In animals with significant lung disease, higher levels of PEEP are often required to allow adequate reductions in FiO_2.

$PaCO_2$: An elevated $PaCO_2$ is due to reduced alveolar minute ventilation. Alveolar minute ventilation is the product of alveolar tidal volume and respiratory rate. If the $PaCO_2$ is greater than the targeted range, an increase in tidal volume and/or respiratory rate is indicated. As higher tidal volumes can be injurious to the lung, every attempt to avoid increasing tidal volume should be made. If the $PaCO_2$ is too low, the tidal volume or respiratory rate should be decreased. Other causes of hypercapnia in the ventilator patient are listed below and should be considered before changing ventilator settings. • Increased apparatus dead space - excess tubing / connectors between the patient and the ventilator circuit Y-piece • Endotracheal tube kink or obstruction • Incorrect assembly of the ventilator circuit • Pneumothorax • Large pulmonary embolism (uncommon).

Troubleshooting: When patients are fighting or 'bucking' the ventilator they will not be able to be ventilated effectively and it is common to note desaturation and hypercapnia. When patients begin to buck the ventilator, a systematic approach should be taken to determine the nature of the problem. Possible causes include: • Hypoxemia • Loss of oxygen supply. • Worsening of underlying disease or development of new pulmonary disease such as pneumothorax, VAP, aspiration pneumonia or acute respiratory distress syndrome • Hypercapnia • Circuit disconnect/leak, tube obstruction or kink, pneumothorax etc. • Pneumothorax; typified by a rapidly climbing PCO2 and a plummeting PaO_2. Need auscultation and potentially thoracocentesis to diagnose • Hyperthermia; anesthetized animals like to have relatively low temperatures, even 102°F/ 38.8°C may cause dogs to pant on the ventilator • Inadequate depth of anesthesia; rely on the routine clinical signs of anesthetic depth. This is one of the most common problems, but care should be taken not to blindly increase the anesthetic drug dose without fully assessing the patient.

References

• Hopper K. Mechanical Ventilation - Core Concepts. In: Silverstein DC, Hopper K, editors. Small Animal Critical Care Medicine, ed 3. St Louis, USA, Elsevier, 2023. pp. 101-112
• Shelledy DC, Peters JI. Mechanical Ventilation, ed 3. Burlington, USA, Jones and Bartlett Learning, Burlington, 2020. pp. 95-366
• Hess D, Kacmarek R. Essentials of Mechanical Ventilation, ed 4. New York, USA, McGraw-Hill, 2019. pp50-72

Recover CPR: Preparedness and Basic Life Support (BLS)

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Learning objectives

- Explain how preparing the veterinary health care team to identify and respond to cardiopulmonary arrest (CPA) can affect patient outcomes.
- Describe the critical team roles that must be filled when initiating cardiopulmonary resuscitation (CPR) in patients in CPA.
- Describe why rapid diagnosis and initiation of basic life support in a dog or cat CPA is crucial.
- Demonstrate correct chest compression technique for a dog or cat given a description of the patient's chest conformation and size.
- Demonstrate correct mouth-to-snout ventilation technique for a dog or cat in CPA and explain how your approach would differ if the animal were endotracheally intubated.

Lecture summary

Preparedness: Veterinary practices should be well prepared for early recognition of CPA. Studies in human medicine have shown that a combination of didactic CPR training and opportunities to practice skills is most effective. Training is recommended for all veterinary personnel who may be called upon to assist in a crisis. Refresher training and drills at least every 6 months have been shown to improve performance in human medicine. A fully stocked and regularly audited crash cart should be available. CPR algorithm charts and emergency drug dosing charts improve adherence to guidelines and individual performance during CPR.

Circulation - Chest Compressions: The initial goals of chest compressions are to provide (1) pulmonary blood flow for oxygen uptake and CO_2 elimination, and (2) tissue perfusion for oxygen delivery to restore cellular metabolic activity. Experimental evidence suggests that even well-executed external chest compressions produce at best 30% of normal cardiac output, making proper technique critical. Chest compressions should be started as soon as possible after diagnosis or suspicion of CPA. Delay in the start of high-quality chest compressions reduces the likelihood of return of spontaneous circulation (ROSC). Chest compressions should be done with the patient in lateral recumbency to a depth of 1/3 to 1/2 the width of the chest at a rate of 100-120 compressions per minute. Leaning on the chest between compressions must be avoided. Chest compressions should be delivered in uninterrupted cycles of 2 minutes and a new compressor should take over after each cycle to reduce the effect of rescuer fatigue.
The cardiac pump theory is based on the concept that the left and right ventricles are directly compressed, while the thoracic pump theory is based on the concept that external chest compressions raise overall intrathoracic pressure.

Blood flow generated by the thoracic pump mechanism likely predominates in large dogs with a round-chested conformation. Therefore, it is recommended that the chest be compressed over the highest point on the lateral thoracic wall with the patient in lateral recumbency. In keel-chested dogs, compressions directly over the heart are recommended. In flat-chested dogs (e.g., English Bulldogs), compressions over the sternum with the patient in dorsal recumbency may be more effective. The compressor should maintain locked elbows with one hand on top of the other, and the shoulders should be directly above the hands. This allows compressions to be done using the core muscles. Chest compressions should be done in cats and small dogs (< 7 kg) directly over the heart.

Airway and Breathing - Ventilation: The patient should be intubated as soon as possible. Both dogs and cats can be intubated in lateral recumbency, so chest compressions should continue during intubation. If an endotracheal tube is not readily available, mouth to snout ventilation is warranted. The patient's mouth should be held closed firmly with one hand. The neck is extended to align the snout with the spine, opening the airway as completely as possible. The rescuer makes a seal over the patient's nares with his/her mouth and blows firmly into the nares to inflate the chest. Thirty chest compressions should be delivered, immediately followed by two breaths. Alternating compressions and ventilations should be continued for 2-minute cycles, and the rescuers rotated every cycle to prevent fatigue. Chest compressions and ventilations should be performed simultaneously in intubated patients. Intubated patients should be ventilated at a rate of 10 breaths per minute with an inspiratory time of approximately 1 second.

References

- *Fletcher DJ, Boller M, Brainard BM, et al. RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 7: Clinical guidelines. J Vet Emerg Crit Care. 2012, 22; Suppl 1S102-31.*
- *Hopper K, Epstein SE, Fletcher DJ, et al. RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 3: Basic life support. J Vet Emerg Crit Care. 2012, 22; Suppl 1S26-43.*
- *McMichael M, Herring J, Fletcher DJ, et al. RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 2: Preparedness and prevention. J Vet Emerg Crit Care. 2012, 22; Suppl 1S13-25.*

Recover CPR: Advanced Life Support (ALS)

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Learning objectives

- Develop a prioritized list of monitoring equipment that is useful during cardiopulmonary resuscitation (CPR) and explain how to interpret the data provided by this equipment.
- Describe how to use the CPR ECG algorithm to diagnose the arrest rhythm during an inter-cycle pause in basic life support (BLS).
- Explain the rationale behind the ALS interventions you would use to treat a patient in asystole or pulseless electrical activity (PEA) and contrast them to the ALS interventions you would use to treat a patient with ventricular fibrillation (VF) or pulseless ventricular tachycardia (PVT).

Lecture summary

Monitoring: Many commonly employed monitoring devices are of limited use during CPR due to their susceptibility to motion artifact and the likelihood that decreased perfusion will compromise accurate readings. Low yield monitoring devices include pulse oximeter and indirect blood pressure monitors, including Doppler and oscillometric devices. The two most useful monitoring devices during CPR are the electrocardiogram (ECG) and end tidal CO_2 monitor ($ETCO_2$). An accurate rhythm diagnosis is essential to guide drug and defibrillation therapy. The goal of ECG monitoring during CPR is to diagnose which of the four most common arrest rhythms are present: (1) asystole, (2) PEA, (3) VF, or (4) PVT. Rhythms 1 and 2 are the "non-shockable" and rhythms 3 and 4 are "shockable". The ECG should be quickly evaluated while compressors are being rotated between 2-minute cycles of CPR, the rhythm diagnosis should be called out, and differing opinions should be solicited. Discussion should not prevent rapid resumption of chest compressions. The presence of measurable CO_2 by $ETCO_2$ monitoring is supportive of (but not definitive for) correct placement of the endotracheal (ET) tube. Because $ETCO_2$ is proportional to pulmonary blood flow, it can also be used as a measure of chest compression efficacy under conditions of constant quality of ventilation. Upon return of spontaneous circulation (ROSC), $ETCO_2$ dramatically increases due to the rapid increase in circulation, and therefore is a valuable early indicator of ROSC during CPR.

Drug Therapy: For non-shockable rhythms, vasopressors are recommended to increase peripheral vasoconstriction. Because cardiac output is low even during optimal external chest compressions, shunting blood away from the periphery is essential to maintain perfusion to the core. Epinephrine acts via α_1 receptors, but has β_1 and β_2 effects. The α_1 effects have been shown to be the most beneficial during CPR. Low doses (0.01 mg/kg IV/IO every other cycle of CPR) are recommended unless CPR has become prolonged and the prognosis is considered poor. Epinephrine may also be administered via ET tube (0.02 mg/kg) by feeding a long catheter through the ET tube. Vasopressin is an alternative that acts via peripheral V1 receptors. Atropine is a parasympatholytic drug that has shown some benefit in only a few studies. Atropine at a dose of 0.04 mg/kg IV/IO may be considered during CPR in dogs and cats, especially if there is evidence of increased vagal tone prior to the arrest. Although specific evidence of efficacy is not available, the use of reversal agents in dogs and cats in which reversible anesthetic/analgesic drugs were recently administered may be considered.

Electrical Defibrillation: The goal of defibrillation is to drive the ventricular myocardial cells into a refractory period, allowing the pacemakers to take over. Defibrillation should be done as soon as possible after diagnosis of a shockable rhythm. The use of biphasic defibrillators is recommended over monophasic defibrillators because a lower energy is required. For monophasic defibrillators, an initial dose of 4-6 J/kg should be used, while biphasic defibrillation should start at 2-4 J/kg. The second dose may be increased by 50%, but subsequent doses should not be further increased.

After defibrillation, chest compressions should be resumed immediately, and a full 2-minute cycle of CPR administered before reassessing the ECG and determining if the patient is still in VF and should be defibrillated again. Brief assessment of the ECG immediately after defibrillation to determine if a perfusing rhythm has resulted is reasonable but should minimally delay resumption of chest compressions.

References

- *Fletcher DJ, Boller M, Brainard BM, et al. RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 7: Clinical guidelines. J Vet Emerg Crit Care. 2012, 22; Suppl 1S102-31.*
- *Rozanski E a, Rush JE, Buckley GJ, et al. RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 4: Advanced life support. J Vet Emerg Crit Care. 2012, 22; Suppl 1S44-64.*
- *Brainard BM, Boller M, Fletcher DJ. RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 5: Monitoring. J Vet Emerg Crit Care. 2012, 22; Suppl 1S65-84.*

Recover CPR: Newborn resuscitation

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Learning objectives

- Develop an approach to resuscitation in the newborn puppy or kitten given it's post-natal heart rate.
- Explain why ventilation is prioritized over circulation in newborn resuscitation efforts.
- Describe the indications for the use of epinephrine during CPR in newborn puppies and kittens.

Lecture summary

The newborn puppy or kitten must rapidly transition its physiology to survive in the environment. The first few minutes may be the most life-threatening in dogs and cats. Mortality during this transitional period is common. RECOVER uses the term "newborn" for a dog or cat during the first few hours of life during which the animal is transitioning from intra- to extra-uterine physiology.

Identification of Newborns in Need of Resuscitation: Adult dogs and cats requiring resuscitation are unresponsive and apneic. Given the physiologic alterations of the newborn, patient selection is based upon those not in need of CPR. These criteria are: (1) normal parturition; (2) mother able to provide care; (3) vigorous: breathing (RR> 15 bpm), clear vocalization and a vigorous response when testing for reflex irritability. All others may need resuscitative measures. All animals born by C-section require resuscitative efforts.

The First 1-2 Minutes: Management of hypoxia is critically important and includes the following therapeutic steps: (1) establishing a patent airway, (2) supplementation of oxygen, (3) ventilation. Fetal membranes should be removed immediately, and the airway cleared by gentle aspiration using a suction bulb if an obstruction to spontaneous breathing is evident. Oxygen should be supplemented if the patient is cyanotic or bradycardic, but routine administration of 100% oxygen is currently not recommended in newborns due to the associated harm, including reduced survival rates compared to air. In veterinary medicine, it is reasonable to administer flow-by oxygen as needed if respiratory issues persist after airway clearance. In addition, rubbing of the animal with a warm towel may lead to stimulate and improve ventilation and circulation and should be initiated as early as possible, and maintenance of normothermia is important.

If apneic or gasping, ventilation should be actively supported by administering breaths with a tight-fitting face mask at a rate of 20-30 breaths per minute. A small gauge needle placement into GV26 may stimulate ventilation. Doxapram is likely not effective. The heart rate will guide resuscitation measures. Further intervention is recommended in newborn puppies and kittens with progressive, severe bradycardia (e.g., < 120 bpm). Atropine is likely not effective as bradycardia is the consequence of hypoxia rather than high vagal tone. Naloxone should be administered if the dam/queen received opioids prior to delivery of the newborn (0.1 mg/kg SQ, IM, preferentially IV/IO, consider intranasal/mucosal).

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

With more severe bradycardia, (<80 bpm) despite optimal ventilation, chest compressions should be conducted by positioning the thumb and indicator fingers of one hand on opposite sides of the chest just over the heart and compressing by approximately 30-50% of the chest width. There are two fundamentally different aspects from adult CPR: (1) chest compressions are initiated during bradycardia, (2) effective ventilation in newborns precludes concurrent chest compression. It is recommended that compressions and ventilations be delivered at a ratio of 3:1, administered at a rate such that 90 chest compressions and 30 breaths can be delivered in a minute (i.e., 120 events per minutes, 0.5 seconds for each).
Epinephrine is less important, as the core issue is asphyxiation. However, if CPR has continued for > 2 minutes without an increase in heart rate, intravenous or intraosseous epinephrine should be considered (0.01-0.03 mg/kg IV/IO). Hypoglycemia can occur during prolonged resuscitation and should be addressed.

References

- *Perlman JM, Wyllie J, Kattwinkel J, et al. Part 7: Neonatal Resuscitation: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Circulation 2015;132:S204-S241.*
- *Moon PF, Erb HN, Ludders JW, et al. Perioperative risk factors for puppies delivered by cesarean section in the United States and Canada. Journal of the American Animal Hospital Association 2000;36:359-368.*
- *Veronesi MC, Panzani S, Faustini M, et al. An Apgar scoring system for routine assessment of newborn puppy viability and short-term survival prognosis. Theriogenology 2009;72:401-407.*

Recover CPR: Post-cardiac arrest care

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Learning objectives

- Devise a therapeutic and monitoring plan to meet ventilation and oxygenation goals in a patient in the post-cardiac arrest period.
- Use global hemodynamic targets to develop a plan to optimize perfusion to tissues in a patient in the post-cardiac arrest period.
- Implement neuroprotective measures to improve neurologic function and outcome in the post-cardiac arrest period.

Lecture summary

After cardiopulmonary arrest (CPA), patient outcome is largely determined by the events that led to and the duration of CPA, but the processes that occur during and after reperfusion can also play a major role. Two thirds of human CPA victims that achieve ROSC die during the post-cardiac arrest (PCA) phase that starts after a return of spontaneous circulation (ROSC) occurs. In veterinary medicine, only 16% of dogs and cats initially successfully resuscitated survived to hospital discharge in one study. Clearly, PCA management strategies are important for improving survival from CPA.

Respiratory Optimization: Short-term mechanical ventilation to ensure optimal arterial oxygen (80 to 100 mm Hg) and CO_2 tension (35 to 40 mm Hg) and to prevent respiratory arrest in the comatose PCA patient is optimal if available, but is not required for patients that are ventilating sufficiently. When a mechanical ventilator is not available, placing the patient in sternal recumbency and careful titration of sedative and analgesic drugs, are alternative approaches to improve ventilation. It important to avoid both hypoxemia and hyperoxemia in the PCA period. Hypoxemia compromises already ischemic tissue beds and can be treated with supplemental oxygen administered by mask, nasal cannula, nasal catheter, or using an oxygen chamber. Significant experimental evidence also substantiates the detrimental effects of oxidative injury during the PCA period, particularly when reperfusion occurs under hyperoxic conditions. Oxygen supplementation during the PCA should be titrated, targeting SpO_2 between 94% and 98%.

Hemodynamic Optimization: Early hemodynamic optimization has proved effective in human cardiac arrest survivors. Central venous oxygen saturation of at least 70% or normalization of lactate concentrations should be used as end points for resuscitation. Hemodynamic optimization should be focused on maintaining a mean arterial blood pressure of 80-120 mmHg. Mild hypertension in the PCA period (systolic arterial blood pressure of up to 200 mmHg and mean arterial pressure of up to 120 mmHg) has been shown to be beneficial in human PCA patients, although more severe arterial hypertension should be avoided. The hemodynamic optimization algorithm in the RECOVER guidelines summarizes the recommendations.
For hypotensive patients (MAP < 80 mmHg, SAP<100 mmHg), fluid resuscitation should be used first. Patients with evidence of peripheral vasodilation or who do not respond to fluid boluses may benefit from vasopressor therapy. PCA myocardial dysfunction is common, and patients not responding to fluids and pressors may require inotropic support as well. For hypertensive patients (MAP > 120, SAP > 200), evaluation of pain control and titration of pressors (if initiated) should be the first steps before considering anti-hypertensive drugs. Normotension does not necessarily equate to adequate perfusion. Once normotension is achieved, measures of oxygen delivery (central venous oxygen saturation and lactate) as previously described should be evaluated. In patients not meeting the targets despite normotension, blood transfusions may be required.

Neuroprotection: Specific neuroprotective strategies shown to be effective in the PCA period include mild therapeutic hypothermia, osmotic therapies such as mannitol or hypertonic saline, and seizure prophylaxis / aggressive seizure control. Although not well investigated in veterinary clinical studies, these therapies are reasonable to consider in the PCA patient with persistent neurologic deficits.

References

- *Fletcher DJ, Boller M, Brainard BM, et al. RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 7: Clinical guidelines. J Vet Emerg Crit Care. 2012, 22; Suppl 1S102-31.*
- *Smarick S, Haskins SC, Boller M, Fletcher DJ. RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 6: Post-cardiac arrest care. J Vet Emerg Crit Care. 2012, 22 Suppl 1S85-101.*

Blood transfusion in my practice: is it realistic?

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Learning objectives

- Understand how to perform safe and effective blood transfusions in canine patient with an emphasis on product selection, appropriate storage, transfusion, and monitoring.
- Understand how to perform safe and effective blood transfusions in cats.
- Describe basic blood compatibility testing both in dogs and cats.

Lecture summary

Careful selection of healthy blood donors and blood collection: Depending on the geographical location, blood donors must be tested for different infectious agents transmitted via a blood transfusion.(1) The ideal canine blood donor is a healthy large breed dog (> 25 kg) to allow blood collection using a standard close-system (typically 450-500 ml with citrate-phosphate-dextrose-adenine (CPDA) +/- additive solution). Ideally, the donor should have a docile temperament to permit blood collection without sedation. Contrary to what is often reported in the veterinary literature, pregnancy does not sensitize bitches to erythrocyte antigens.(2) Feline donors should ideally weigh > 4.5 kg and be young indoor adult cats. The cat must have a good temperament to facilitate blood collection, but sedation is almost always required. About 10-15 ml/kg can be collected (i.e. 40-60 ml/cat); no commercial closed collection system suitable for feline blood sampling is commercially available. Most often, a 18-19G butterfly needle connected by a three-way valve to syringes/bags containing CPDA-anticoagulant is used. Since this constitute an open-collection system, the blood collected should be used within 24 hours to limit the risk of bacterial contamination. That said, if the blood aseptically, many veterinary centers store the blood for up to 20 days.

Blood components and storage: Blood can be separated into different compounds (packed RBC (pRBC), fresh frozen plasma, etc.). Whole blood and RBC concentrate are stored at 4°C in a dedicated refrigerator and should be gently rocked daily. When used alone, CPDA-1 allows storage of RBC for up to 20 days. Preservatives (e.g. Adsol or Nutrisol) added to canine pRBC increase storage time to 35-37 days. Plasma products should be kept at -20ºC (or colder) and has a shelf life of about one year. Feline patients are often transfused whole blood, but separation into components is quite feasible, even using simple sedimentation. The advantages of using blood components are: 1- to limit the risk of transfusion reaction, including vascular overload, by limiting the transfusion of non-essential blood elements, 2- a single unit of blood can be used for more than one patient, 3- coagulation factors are maintained active in FFP (decrease in factor V, VIII and vWF at 8-24 hours post-storage).

Appropriate administration of blood products: In addition to anemia where RBC containing products are required, numerous coagulopathies can benefit from specific blood products (ex: cryoprecipitate for von Willebrand factor deficiency and hemophilia A). The decision to administer a blood product should be based on the patient clinical signs. Indications for RBC transfusion in an anemic patient include weakness, exercise intolerance, tachycardia, tachypnea, weak pulse. Signs of coagulopathy may include petechiae, ecchymosis, hematomas, bleeding at venipuncture sites. Prolongation of clotting times may justify the use of blood products if an invasive procedure is planned (ex.: surgery, liver biopsy) or in actively bleeding patients. Blood products must be examined prior to administration (expiration date, any discoloration/hemolysis). The use of volume-appropriate filters is essential. Only approved pumps for transfusion purpose should be used. In fact, administration by simple gravity is ideal in dogs as even approved pumps may lead to significant hemolysis (4), while a syringe-pump administration appears safe in cats. A slower transfusion rate (0.25 ml/kg/hr) is initially recommended. The patient should be closely monitored regardless of the transfused blood product. Mental state, temperature, heart/respiratory rate, mucosal color and capillary refill time should be noted before starting the transfusion, and then 15 minutes after it starts. If vitals are stable, the transfusion rate can be increased to 5-10 ml/kg/hr with monitoring every 20-30 minutes throughout the transfusion. The patient should also be monitored for signs of vomiting, diarrhea, angioedema, urticaria and hemoglobinuria. Transfusions are better tolerated at slower rate, while respecting a 4-hour window to limit risk of bacterial growth. In severe acute hemorrhage, the administration can be done much faster. In opposite, cardiac patients may receive their transfusion over two successive 4 hour-periods, while the rest of the unit is in the refrigerator.

Principles of blood compatibility: blood typing and crossmatch: It is strongly advised to determine the DEA 1 status of canine patients/donors. Similarly, because of the presence of strong naturally occurring anti-A antibodies in type B cats, AB blood typing is crucial in cats. A crossmatch should be performed in patients that have already received a blood transfusion (> 4 days previously), and ideally in cats prior to any transfusion. Simple, fast and inexpensive DEA 1 and AB blood typing and crossmatching kits are now commercially available. Finally, the purpose of this presentation is to demystify blood transfusions.

References

- *Wardrop KJ, Birkenheuer A, Blais MC, Callan MB, Kohn B, Lappin MR, et al. Update on Canine and Feline Blood Donor Screening for Blood-Borne Pathogens. Journal of veterinary internal medicine. 2016;30(1):15-35.*
- *Blais MC et al. Lack of evidence of pregnancy-induced alloantibodies in dogs. Journal of veterinary internal medicine. 2009;23(3):462-5.*
- *Weatherton LK et al. Evaluation of fresh frozen plasma administration in dogs with pancreatitis: 77 cases (1995-2005). Journal of veterinary emergency and critical care 2009;19(6):617-22.*
- *McDevitt RI et al. Influence of transfusion technique on survival of autologous red blood cells in the dog. Journal of veterinary emergency and critical care (San Antonio, Tex : 2001). 2011;21(3):209-16.*
- *Kisielewicz C, Self IA. Canine and feline blood transfusions: controversies and recent advances in administration practices. Vet Anaesth Analg. 2014;41:233-242.*

How to fit a mansion into a tiny house - the pop-up emergency area

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Learning objectives

- Recognise essential equipement for receiving emergencies
- Understand that such equipement is in reach of every practitioner
- Design a pop-up emergency area for your practice
- Explain the value of preparedness to your practice manager

Lecture summary

From the dawn of emergency medicine during wars to modern emergency departments in human and veterinary hospitals, little essential equipment is needed to provide good quality care for emergency patients during stabilization. However, this equipment and consumables, little as they might need to be ready, functional, and dedicated to the eventuality of an emergency. While larger hospitals have fully functional dedicated spaces and teams ready to receive emergencies, such units are not readily available in smaller clinics, which makes receiving emergencies, especially during working hours challenging.

The space: Emergencies require a dedicated space, which can be occupied for a couple of hours at the time. While in after-hours admissions this space is more readily available - anaesthesia prep room or surgery room - during working hours emergencies will need to compete with ongoing routine procedures. A space that is very easily transformable into a "pop-up emergency room" is actually a regular consult room. However, some elements need to be brought into the room to ensure its transformation.

Oxygen source: Oxygen is required for almost every unstable emergency patient. While having a walled-in oxygen source is unrealistic for most practices, portable oxygen sources are readily available and at a reasonable price. The main two alternatives are oxygen bottles (or tanks) or oxygen condensers. Some of the modern oxygen condensers used for house hospitalization of people are the size of a kettle. There are even oxygen condensers from which oxygen bottles can be filled. Oxygen sources should be coupled with the necessary adaptors, tubing and humidifiers.

Suction device: Suction might not be commonly required for emergencies, but when it is needed it is really needed. Compact, portable suction devices that do not require a wall outlet, can be purchased for reasonable prices, and stored ready-to-use near the oxygen source.

Examination table and light source: Most consult rooms usually have and examination table and decent lighting; however, emergencies might require additional lighting. There is a myriad of available portable medical lights with varying price range. Furthermore, a good work light attached to various surfaces can also do the trick. Having a trolley and a gurney to transport the patient to the consult room is also beneficial.

Crash cart: Crash carts are a feature of every well-organized emergency department. They are set of trays, drawers or shelfs on wheels, carrying supplies and equipment for the management of emergencies. Rather than its size, the most important quality of the crash cart is that it is well-organized, clearly labelled and frequently audited. If the clinic has no space for an actual crash *cart*, essential equipment could fit into a toolbox, purpose-made backpack, or duffle bag. Similar to the oxygen source and suction, equipment from the crash cart is not removed and used for anything else than emergencies. This equipment includes, but it is not limited to airway management and ventilation, IV access, emergency drugs, IV fluids, a basic surgical set, glove, disinfectants, syringes, needles, including butterfly catheters and three-way stop cocks.

Warming devices: Hypothermia is not uncommon in unstable emergency patients and a warming device should be readily available for initial stabilization. From Hot Dogs™, Bair Huggers™, and warming pads to microwaved wheat bags and warm water filled gloves, clinicians should choose which technique suits their environment the most and have a device ready.

Monitoring and minimum database: Basic monitoring during initial stabilization includes non-invasive blood pressure, pulse oximetry and electrocardiogram. Veterinary specific multimodal monitoring devices can be purchased for reasonable prices, stored, and used only for emergencies. End-tidal CO_2 could be used for intubated patients. There are also non-invasive end-tidal CO_2 devices that work with nasal prongs. End-tidal CO_2 monitors are a step up in the price scale for monitoring and rational budgeting should be made before purchasing a unit. Most clinics have basic laboratory equipment available. If this is not the case, at least a PCV/TP centrifuge, glucometer, Azostix®, microscope with Diff Quik stains and potentially a lactate meter should be available in-house. At present, point-of-care ultrasound has become an essential element in stabilizing emergencies. Having a portable device that can be brought to the patient is ***a must*** for every modern clinic.

Team "emergency" and preparedness: Staffing varies between facilities and on a regular basis general practitioners have fully booked days. Emergencies on the other hand are not scheduled and even if they happen to be, stabilization requires more time than a regular consult. Veterinarians or veterinary nurses/technicians should stay near unstable patients *at all times*. Therefore, flexibility should be ensured for the staff members dealing with emergencies, allowing for transfer of consults to other staff members and potentially cancellations of routine consults. Ideally a veterinarian and a veterinary nurse/technician should be paired up for admitting emergencies. While everyone dreads the unannounced agonal emergency rushing through the door, most owners actually call before they bring in their sick pet. This allows some time to prepare the room for receiving, bring in additional equipment, quickly perform a final audit of the crash "box" and brief with the team. Dealing with emergencies is challenging. The negative stress from such challenges can, however, be tremendously reduced with having a flexible space, dedicated equipment, and a notion of preparedness. This will allow for satisfaction and a sence of empowerment to dominate such experiences. All this can be accomplished with little resources and realistic budgets.

Note: Estimates for prices will be provided during the presentation.

References

Upon request.

Let's chit-chat about cats - five pechakucha style presentations

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Learning objectives

- Understand the controversy in feline hypothermia
- Recognize the importance of assessing fluid responsiveness and avoiding volume overload in cats
- Revise the usefulness of lactate measurements in cats
- Appraise advantages and shortcomings of current BP measurement techniques

Lecture summary

Hypothermia: Hypothermia seems to accompany critical illnesses in cats and is an independent negative prognostic indicator in cats presented to the clinic with severe illness. It has been associated with hypotension and shock in cats presented to the emergency room for various conditions, providing some evidence for the empirically accepted feline shock triad of hypothermia, bradycardia, and hypotension. Based on experimental data, it is possible that hypothermia is centrally mediated during illness by a direct effect of catecholamines and potentially exacerbated by other factors. Responsiveness of the alpha-adrenergic receptors (and therefore ability for vasoconstriction) is therefore attenuated during hypothermia, even though some of the results are contradictory. While hypothermia can have detrimental effects, it is equaly possibly that it is a protective mechanism allowing metabolism to slow while healing is taking place. Active rewarming should be initiated in cats with naturally occurring hypothermia, with careful titrating of the speed of rewarming and the target temperature. Fluid challenges should be administered in hypovolemic cats with hypothermia, again with careful titrating to effect and conservative approach to repeat administration if no response is seen.
Intra-vascular volume overload: Cats are known empirically to be predisposed to intra-vascular volume overload (IVVO) and fluid overload (FO). Development of interstitial oedema, pleural effusions and other clinical manifestations of congestive heart failure are complications from IVVO. Interstitial oedema, while not obviously life-threatening can be viewed as a "silent killer" in the hospitalized cat. Interstitial oedema increases the distance that oxygen needs to travel from the capillary to the cells and therefore contributes to tissue hypoxia and subsequent organ damage, especially in already compromised patients. Indeed, many clinical studies report reduced survival rates in people with FO as opposed to people without FO. While robust convincing data for the origin of such predisposition in cats is lacking several factors that may contribute to intravascular volume overload have been identified. Cats have smaller blood volume compared to dogs and people. Experimental studies have convincingly demonstrated that feline blood volume ranges from ~ 50 - 60 ml/kg. Similar to dogs and people, blood volume in cats is proportionate to their lean body weight. Obese cats, therefore, have a relatively smaller per kilogram blood volume. As a consequence, fluid calculations should be preferably made on a lean body mass basis, especially in the severely obese cat. Cats have occult cardiomyopathies. This might be a somewhat outdated statement at this point. With increase of awareness and development of focused cardiac ultrasonography (FOCUS) techniques, early recognition of asymptomatic cardiomyopathies is becoming a capability of many veterinarians. Why is this important? Cardiomyopathies lead to IVVO due to activation of the renin-angiotensin-aldosterone system. A cat, nonetheless, can live a relatively long life with mild cardiomyopathy and succumb from other causes. However, if this cat happens to get in other trouble - trauma, sepsis, or even a routine procedure, and requires IV fluid administration, that might lead to decompensation and life-threatening congestive heart failure. Ideally a FOCUS should be part of the annual health check of every aging cat, just as an arterial blood pressure measurement. A FOCUS should be performed on every hospitalized cat, requiring IV fluid administration. Anaemia is associated with volume overload. Anaemia, whether acute or chronic, leads to volume overload. This has been well documented in several studies in cats using echocardiography. The proposed mechanism is a neuroendocrine response to "arterial underfilling". As a result, the renin-angiotensin-aldosterone system is activated leading to sodium and water retention. Anaemia is commonly diagnosed in the sick cat, requiring hospitalization and potentially fluid administration. Volume overload might be asymptomatic during presentation, but care should be taken not to exacerbate it. Post-transfusion volume overload complications have been documented in cats. Care should be taken with transfusion rates and volumes in the euvolemic, chronically anaemic cat and volume assessment should be performed regularly during and post-transfusion. Cats eliminate IV fluids relatively slowly. Volume kinetics is the study of distribution and elimination of IV fluids, similar to the term pharmacokinetics used to describe the distribution and elimination of drugs. It is a relatively novel field of research with a lot of potential in the current environment where IV fluids are recognized as drugs. A recent study in healthy conscious cats, has demonstrated that cats eliminate fluids slower compared to other species. A lot of research is yet to be done in this space, before it can be translated into clinical practice. These results, however, suggest that careful titration of subsequent bolus administration should be performed during fluid resuscitation in cats.

Fluid responsiveness: Accurate evaluation of fluid responsiveness is a "hot topic" in human, and as an extension, veterinary medicine. While terminology is sometimes contradictory and definitions lose, the term fluid responsiveness is used in the context of intravascular volume expansion (also known as fluid resuscitation, bolus administration etc). Fluid responsiveness occurs (or not) as a result of a "fluid challenge" (or bolus). A fluid challenge is meant to increase stroke volume and improve perfusion in the hypovolemic cat. Fluid responsiveness is manifested as improvement in perfusion parameters such as mentation, pulse rate and quality, mucous membrane colour, CRT, and temperature, alongside other parameters. Studies from people have demonstrated that a large proportion of patients admitted to the emergency department with circulatory shock will not respond to a fluid challenge. Are cats in non-cardiogenic circulatory shock responsive to fluid administration? There is much empirical knowledge and little research data to support that more often than not they might not be. A recent study in cats with circulatory shock defined as changes in 2 or more perfusion parameters, lactate, and arterial blood pressure, 42% of the 24 cats enrolled were non-responders. Another recent retrospective study on fluid resuscitation in hypotensive cats found that only 37% of the cats were responders. While the criteria for responsiveness differed between studies, they

both demonstrate relatively low magnitude of response. While a lot of questions regarding fluid responsiveness in cats remain to be answered, caution needs to be taken when administering fluid boluses in hypovolemic cats, presence or absence of a response monitored carefully and fluid administration ceased early during resuscitation if no response is seen.

Lactate: The value of lactate concentrations as a diagnostic tool or prognostic test has been understudied in sick cats as compared to people, dogs, and horses. Increases in lactate have been found in septic cats with multi-organ dysfunction, have been associated with increased animal trauma triage scores in cats with trauma and not surprisingly found during cardio-pulmonary resuscitation. Lactate measurements are part of the feline Acute Physiologic Patient and Laboratory Evaluation score (full and fast version). Currently, there are no studies that have purposefully evaluated serum lactate as a diagnostic biomarker for circulatory shock in cats. Some studies in cats, mostly in sick cats presented as emergencies with various types of diseases and unstratified for severity, have shown some association of higher lactate concentrations with non-survival, while others in similar patient populations have failed to demonstrate the utility of lactate as a prognostic indicator. A recent large scale retrospective study in a population of 444 cats with a lactate measurement within 4 hours of admission demonstrated an association between hyperlactatemia and mortality. In people, dogs and horses' normal initial lactate might be indicative of survival. This is yet to be proven in cats. In the meantime, clinicians should interpret both normal and high lactate in cats with caution and potentially not rely on lactate as a sole diagnostic and prognostic indicator.

Non-invasive blood pressure measurements: Currently there are three widely available techniques for non-invasive blood pressure measurements: oscillometric (O), high definition oscillometric (HDO) and Doppler ultrasonographic (DU). According to the literature, measurements obtained from these three techniques cannot be used interchangeably. Reference intervals therefore should be generated for each technique. These have not yet been reported in the literature. Doppler seems to be a preferred method for BP measurements in North America, while in Europe Doppler and O are used as frequently. A few studies have compared O to DU to HDO between each other or to a gold standard - direct BP measurements with contradicting results. Most of the studies using direct BP measurements as a standard are performed in anesthetized (and often hypotensive) cats. Oscillometric and HDO tend to overestimate low BP and underestimate high BP, with HDO showing overall better accuracy compared to O (with less data available). Systolic DU measurements DO NOT equal direct MAP measurements in cats. There is good correlation between them, but the agreement is poor. Overall agreement between measurement sites (tail vs limb) is poor, with coccygeal artery measurements being consistently higher for SBP. Therefore, the measurement site should remain the same for each patient. Measurements are more accurate when the cuff is placed more distally. Overall, there are very few studies evaluating the performance of BP measurement techniques in sick hypotensive cats. Current ISFM guidelines should be followed when performing BP measurements in cats. They are, though, tailored for screening for hypertension. They do not recommend the use of the O technique.

References

- *Amini-Sereshki-Kormi L. Effects of ambient temperature on thermoregulation during fever in cats. Pathobiology 1996;64:284-288.*
- *Aoki A, Mori K, Maeda M. Adequate cerebral perfusion pressure during rewarming to prevent ischemic deterioration after therapeutic hypothermia. Neurol Res 2002;24:271-280.*
- *Aoki A, Mori K, Maeda M, et al. Appropriate cerebral perfusion pressure during rewarming after therapeutic hypothermia. Acta Neurochir Suppl 2002;81:237-239.*
- *Battista AF. Deep hypothermia in the cat. Am J Physiol 1959;196:354-356.*
- *Borgeat K, Wright J, Garrod O, et al. Arterial thromboembolism in 250 cats in general practice: 2004-2012. J Vet Intern Med 2014;28:102-108.*
- *Brady CA, Otto CM, Van Winkle TJ, et al. Severe sepsis in cats: 29 cases (1986-1998). J Am Vet Med Assoc 2000;217:531-535.*
- *Brodeur A, Wright A, Cortes Y. Hypothermia and targeted temperature management in cats and dogs. J Vet Emerg Crit Care (San Antonio) 2017;27:151-163.*
- *Chopp M, Knight R, Tidwell CD, et al. The metabolic effects of mild hypothermia on global cerebral ischemia and recirculation in the cat: comparison to normothermia and hyperthermia. J Cereb Blood Flow Metab 1989;9:141-148.*
- *Culp WT, Weisse C, Kellogg ME, et al. Spontaneous hemoperitoneum in cats: 65 cases (1994-2006). J Am Vet Med Assoc 2010;236:978-982.*
- *Nielsen KC, Owman C. Control of ventricular fibrillation during induced hypothermia in cats after differential depletion of cardiac catecholamine stores with prenylamine (Segontin). Circ Res 1967;21:45-58.*
- *Clasen RA, Pandolfi S, Russell J, et al. Hypothermia and hypotension in experimental cerebral edema. Arch Neurol 1968;19:472-486. Nielsen KC, Owman C. Difference in cardiac adrenergic innervation between hibernators and non-hibernating mammals. Acta Physiol Scand Suppl 1968;316:1-30.*
- *Kar K, Sur RN, Pradhan SN. Depressor response to noradrenaline in hypothermic cats. Jpn J Pharmacol 1969;19:68-73.*
- *Weihe WH. The effect of temperature on the action of drugs. Annu Rev Pharmacol 1973;13:409-425.*
- *Kiley JP, Eldridge FL, Millhorn DE. Respiration during hypothermia: effect of rewarming intermediate areas of ventral medulla. J Appl Physiol (1985) 1985;59:1423-1427.*
- *Gautier H, Gaudy JH. Ventilatory recovery from hypothermia in anesthetized cats. Respir Physiol 1986;64:329-337. Hale FA, Anthony JM. Prevention of hypothermia in cats during routine oral hygiene procedures. Can Vet J 1997;38:297-299.*
- *Mori K, Maeda M, Miyazaki M, et al. Misery perfusion caused by cerebral hypothermia improved by vasopressor administration. Neurol Res 1999;21:585-592.*
- *Ruthrauff CM, Smith J, Glerum L. Primary bacterial septic peritonitis in cats: 13 cases. J Am Anim Hosp Assoc 2009;45:268-276.*
- *Boland LA, Angles JM. Feline permethrin toxicity: retrospective study of 42 cases. J Feline Med Surg 2010;12:61-71.*
- *Goic JB, Reineke EL, Drobatz KJ. Comparison of rectal and axillary temperatures in dogs and cats. J Am Vet Med Assoc 2014;244:1170-1175.*
- *Lenchner I, Aroch I, Segev G, et al. A retrospective evaluation of Vipera palaestinae envenomation in 18 cats: (2006-2011). J Vet Emerg Crit Care (San Antonio) 2014;24:437-443.*
- *Levy JK, Nutt KR, Tucker SJ. Reference interval for rectal temperature in healthy confined adult cats. J Feline Med Surg 2015;17:950-952.*
- *Reineke EL, Rees C, Drobatz KJ. Association of blood lactate concentration with physical perfusion variables, blood pressure, and outcome for cats treated at an emergency service. J Am Vet Med Assoc 2015;247:79-84.*
- *Kabatchnick E, Langston C, Olson B, et al. Hypothermia in Uremic Dogs and Cats. J Vet Intern Med 2016;30:1648-1654.*
- *Leister E, Morton J, Atwell R, et al. Clinical presentations, treatments and risk factors for mortality in cats with tick paralysis caused by Ixodes holocyclus: 2077 cases (2008-2016). J Feline Med Surg 2018;20:465-478.*
- *Ruiz MD, Vessières F, Ragetly GR, et al. Characterization of and factors associated with causes of pleural effusion in cats. J Am Vet Med Assoc 2018;253:181-187.*
- *Hassan MH, Abu-Seida AM, Torad FA, et al. Feline aortic thromboembolism: Presentation, diagnosis, and treatment outcomes of 15 cats. Open Vet J 2020;10:340-346.*
- *Cagle LA, Hopper K, Epstein SE. Complications associated with long-term positive-pressure ventilation in dogs and cats: 67 cases. J Vet Emerg Crit Care (San Antonio) 2022;32:376-385.*
- *Fitzgerald WR, Cave NJ, Yozova ID. Clinical parameters at time of admission as prognostic indicators in cats presented for trauma to an emergency centre in New Zealand: a retrospective analysis. J Feline Med Surg 2022;24:1294-1300.*
- *Hiebert EC, Barry SL, Sawyere DM, et al. Intestinal dehiscence and mortality in cats undergoing gastrointestinal surgery. J Feline Med Surg 2022;24:779-786.*
- *Nascimento DC, Costa Neto JM, Solcà MS, et al. Clinicoepidemiological profile and risk factors associated with mortality in traumatized cats admitted to a veterinary teaching hospital in Brazil. J Feline Med Surg 2022;24:381-388.*

- *Tindale C, Cinti F, Cantatore M, et al. Clinical characteristics and long-term outcome of lung lobe torsions in cats: a review of 10 cases (2000-2021). J Feline Med Surg 2022;24:1072-1080.*
- *Cooper Gt, Puga FJ, Zujko KJ, et al. Normal myocardial function and energetics in volume-overload hypertrophy in the cat. Circ Res 1973;32:140-148.*
- *Carey RA, Natarjan G, Bove AA, et al. Elevated collagen content in volume overload induced cardiac hypertrophy. J Mol Cell Cardiol 1980;12:929-936.*
- *Marino TA, Houser SR, Cooper Gt. Early morphological alterations of pressure-overloaded cat right ventricular myocardium. Anat Rec 1983;207:417-426.*
- *Breisch EA, White FC, Bloor CM. Myocardial characteristics of pressure overload hypertrophy. A structural and functional study. Lab Invest 1984;51:333-342.*
- *Cooper Gt, Kent RL, Uboh CE, et al. Hemodynamic versus adrenergic control of cat right ventricular hypertrophy. J Clin Invest 1985;75:1403-1414.*
- *Marino TA, Kent RL, Uboh CE, et al. Structural analysis of pressure versus volume overload hypertrophy of cat right ventricle. Am J Physiol 1985;249:H371-379.*
- *Oparil S. Pathogenesis of ventricular hypertrophy. J Am Coll Cardiol 1985;5:57b-65b. Lauva IK, Brody E, Tiger E, et al. Control of myocardial tissue components and cardiocyte organelles in pressure-overload hypertrophy of the cat right ventricle. Am J Anat 1986;177:71-80.*
- *Marino TA, Brody E, Lauva IK, et al. Reversibility of the structural effects of pressure overload hypertrophy of cat right ventricular myocardium. Anat Rec 1986;214:141-147.*
- *Wisenbaugh T, O'Connor WN. Right ventricular hypertrophy long after reversal of severe pressure overload in cats. Am J Physiol 1988;254:H1099-1104.*
- *Elzinga G, Toorop GP, Gross DR, et al. Geometry and pump function in cardiac ventricular hypertrophy. Am J Cardiol 1990;65:23g-29g. Marino TA, Cassidy M, Marino DR, et al. Norepinephrine-induced cardiac hypertrophy of the cat heart. Anat Rec 1991;229:505-510.*
- *Wang ZZ, He L, Stensaas LJ, et al. Localization and in vitro actions of atrial natriuretic peptide in the cat carotid body. J Appl Physiol (1985) 1991;70:942-946.*
- *Hori Y, Yamano S, Iwanaga K, et al. Evaluation of plasma C-terminal atrial natriuretic peptide in healthy cats and cats with heart disease. J Vet Intern Med 2008;22:135-139.*
- *Weingart C, Kohn B. Clinical use of a haemoglobin-based oxygen carrying solution (Oxyglobin) in 48 cats (2002-2006). J Feline Med Surg 2008;10:431-438.*
- *Wilson HE, Jasani S, Wagner TB, et al. Signs of left heart volume overload in severely anaemic cats. J Feline Med Surg 2010;12:904-909.*
- *Finch NC, Heiene R, Elliott J, et al. Determination of extracellular fluid volume in healthy and azotemic cats. J Vet Intern Med 2015;29:35-42.*
- *Black V, Adamantos S, Barfield D, et al. Feline non-regenerative immune-mediated anaemia: features and outcome in 15 cases. J Feline Med Surg 2016;18:597-602.*
- *Yiew XT, Bateman SW, Hahn RG, et al. Evaluation of the Distribution and Elimination of Balanced Isotonic Crystalloid, 5% Hypertonic Saline, and 6% Tetrastarch 130/0.4 Using Volume Kinetic Modeling and Analysis in Healthy Conscious Cats. Front Vet Sci 2020;7:587564.*
- *Adamantos S. Fluid Therapy in Pulmonary Disease: How Careful Do We Need to Be? Front Vet Sci 2021;8:624833.*
- *Boysen SR, Gommeren K. Assessment of Volume Status and Fluid Responsiveness in Small Animals. Front Vet Sci 2021;8:630643.*
- *Donaldson RE, Seo J, Fuentes VL, et al. Left heart dimensions in anemic cats and dogs before and after blood transfusion. J Vet Intern Med 2021;35:43-50.*
- *Zeiler GE, Dzikiti BT, Kamerman P, et al. Investigation of biomarkers for impending fluid overload in a feline acute haemorrhage-resuscitation model. Vet Anaesth Analg 2021;48:871-881.*
- *Tinson E, Talbot CT, Humm K. Incidence of acute haemolysis in cats receiving canine packed red blood cells (xenotransfusions). J Feline Med Surg 2022;24:e628-e635.*
- *Loughran KA, Rush JE, Rozanski EA, et al. The use of focused cardiac ultrasound to screen for occult heart disease in asymptomatic cats. J Vet Intern Med 2019;33:1892-1901.*
- *Boysen SR, Gommeren K. Assessment of Volume Status and Fluid Responsiveness in Small Animals. Front Vet Sci 2021;8:630643.*
- *Chalifoux NV, Hess RS, Silverstein DC. Effectiveness of intravenous fluid resuscitation in hypotensive cats: 82 cases (2012-2019). J Vet Emerg Crit Care (San Antonio) 2021;31:508-515.*
- *Donati PA, Tunesi M, Araos J. Caudal vena cava measurements and fluid responsiveness in hospitalized cats with compromised hemodynamics and tissue hypoperfusion. J Vet Emerg Crit Care (San Antonio) 2023;33:29-37.*
- *Anderson T, Beever L, Hall J, et al. Outcome following surgery to treat septic peritonitis in 95 cats in the United Kingdom. J Small Anim Pract 2021;62:744-749.*
- *Bachmann K, Kutter AP, Schefer RJ, et al. Determination of reference intervals and comparison of venous blood gas parameters using standard and non-standard collection methods in 24 cats. J Feline Med Surg 2017;19:831-840.*
- *Costello MF, Drobatz KJ, Aronson LR, et al. Underlying cause, pathophysiologic abnormalities, and response to treatment in cats with septic peritonitis: 51 cases (1990-2001). J Am Vet Med Assoc 2004;225:897-902.*
- *Di Mauro FM, Schoeffler GL. Point of Care Measurement of Lactate. Top Companion Anim Med 2016;31:35-43. Donati PA, Rabelo RC, Araos J, et al. Retrospective evaluation of jugular venous blood variables and mortality in critically ill hospitalized cats. J Vet Emerg Crit Care (San Antonio) 2022;32:777-783.*
- *Gilday C, Guieu L. Prognostic value of lactate in cats presented in respiratory distress to the emergency room. Front Vet Sci 2022;9:918029.*
- *Goic JB, Reineke EL, Drobatz KJ. Comparison of rectal and axillary temperatures in dogs and cats. J Am Vet Med Assoc 2014;244:1170-1175.*
- *Goutal CM, Keir I, Kenney S, et al. Evaluation of acute congestive heart failure in dogs and cats: 145 cases (2007-2008). J Vet Emerg Crit Care (San Antonio) 2010;20:330-337.*
- *Gower SB, Weisse CW, Brown DC. Major abdominal evisceration injuries in dogs and cats: 12 cases (1998-2008). J Am Vet Med Assoc 2009;234:1566-1572.*
- *Greenway CV, Lautt WW. Acute and chronic ethanol on hepatic oxygen ethanol and lactate metabolism in cats. Am J Physiol 1990;258:G411-418.*
- *Hayes G, Mathews K, Doig G, et al. The Feline Acute Patient Physiologic and Laboratory Evaluation (Feline APPLE) Score: a severity of illness stratification system for hospitalized cats. J Vet Intern Med 2011;25:26-38.*
- *Hoehne SN, Hopper K, Epstein SE. Association of point-of-care blood variables obtained from dogs and cats during cardiopulmonary resuscitation and following return of spontaneous circulation with patient outcomes. J Vet Emerg Crit Care (San Antonio) 2022. Hopper K, Borchers A, Epstein SE. Acid base, electrolyte, glucose, and lactate values during cardiopulmonary resuscitation in dogs and cats. J Vet Emerg Crit Care (San Antonio) 2014;24:208-214.*
- *Kohen CJ, Hopper K, Kass PH, et al. Retrospective evaluation of the prognostic utility of plasma lactate concentration, base deficit, pH, and anion gap in canine and feline emergency patients. J Vet Emerg Crit Care (San Antonio) 2018;28:54-61.*
- *Lee JA, Huang CM, Hall KE. Epidemiology of severe trauma in cats: An ACVECC VetCOT registry study. J Vet Emerg Crit Care (San Antonio) 2022;32:705-713.*
- *Lyons BM, Ateca LB, Otto CM. Clinicopathologic abnormalities associated with increased animal triage trauma score in cats with bite wound injuries: 43 cases (1998-2009). J Vet Emerg Crit Care (San Antonio) 2019;29:296-300.*
- *Maxwell K, Fraser B, Schaer M, et al. A retrospective evaluation of eastern coral snake envenomation and antivenom administration in cats: 30 cases (2012-2019). Toxicon 2021;191:38-43.*
- *Parsons KJ, Owen LJ, Lee K, et al. A retrospective study of surgically treated cases of septic peritonitis in the cat (2000-2007). J Small Anim Pract 2009;50:518-524.*
- *Rand JS, Kinnaird E, Baglioni A, et al. Acute stress hyperglycemia in cats is associated with struggling and increased concentrations of lactate and norepinephrine. J Vet Intern Med 2002;16:123-132.*
- *Redavid LA, Sharp CR, Mitchell MA, et al. Plasma lactate measurements in healthy cats. J Vet Emerg Crit Care (San Antonio) 2012;22:580-587.*
- *Redavid LA, Sharp CR, Mitchell MA, et al. Hyperlactatemia and serial lactate measurements in sick cats. J Vet Emerg Crit Care (San Antonio) 2016;26:495-501.*
- *Reineke EL, Rees C, Drobatz KJ. Association of blood lactate concentration with physical perfusion variables, blood pressure, and outcome for cats treated at an emergency service. J Am Vet Med Assoc 2015;247:79-84.*
- *Rosenstein PG, Tennent-Brown BS, Hughes D. Clinical use of plasma lactate concentration. Part 2: Prognostic and diagnostic utility and the clinical management of hyperlactatemia. J Vet Emerg Crit Care (San Antonio) 2018;28:106-121.*
- *Rosenstein PG, Tennent-Brown BS, Hughes D. Clinical use of plasma lactate concentration. Part 1: Physiology, pathophysiology, and measurement. J Vet Emerg Crit Care (San Antonio) 2018;28:85-105.*
- *Sachs EK, Julius TM, Claypool SA, et al. Comparison of cephalic and jugular plasma lactate concentrations in sick cats: a pilot study. J Vet Emerg Crit Care (San Antonio) 2017;27:193-197.*
- *Saint-Pierre LM, Hopper K, Epstein SE. Retrospective evaluation of the prognostic utility of plasma lactate concentration and serial lactate measurements in dogs and cats presented to the emergency room (January 2012 - December*

2016): 4863 cases. J Vet Emerg Crit Care (San Antonio) 2022;32:42-49.
- Sharma D, Holowaychuk MK. Retrospective evaluation of prognostic indicators in dogs with head trauma: 72 cases (January-March 2011). J Vet Emerg Crit Care (San Antonio) 2015;25:631-639.
- Shea EK, Dombrowski SC, Silverstein DC. Survival analysis of hypotensive cats admitted to an intensive care unit with or without hyperlactatemia: 39 cases (2005-2011). J Am Vet Med Assoc 2017;250:887-893.
- Troia R, Buzzurra F, Ciuffoli E, et al. Classification of Septic Shock Phenotypes Based on the Presence of Hypotension and Hyperlactatemia in Cats. Front Vet Sci 2021;8:692528.
- Tynan B, Kerl ME, Jackson ML, et al. Plasma lactate concentrations and comparison of two point-of-care lactate analyzers to a laboratory analyzer in a population of healthy cats. J Vet Emerg Crit Care (San Antonio) 2015;25:521-527.
- Wolf JM, Stefanovski D, Silverstein DC. Retrospective evaluation of the influence of azotemia on plasma lactate concentrations in hypotensive dogs and cats (2008-2018): 337 cases. J Vet Emerg Crit Care (San Antonio) 2020;30:449-454.
- Sparkes A, Garelli-Paar C, Blondel T, et al. 'The Mercury Challenge': feline systolic blood pressure in primary care practice - a European survey. J Feline Med Surg 2022;24:e310-e323.Navarro I, Summers S, Rishniw M, et al. Cross-sectional survey of non-invasive indirect blood pressure measurement practices in cats by veterinarians. J Feline Med Surg 2022;24:1195-1202.
- Murphy KM, Rishniw M, Silverstein DC. Use of vasopressors for treatment of vasodilatory hypotension in dogs and cats by Diplomates of the American College of Veterinary Emergency and Critical Care. J Vet Emerg Crit Care (San Antonio) 2022;32:714-722.
- De Rosa SM, Drobatz KJ, Reineke EL. Evaluation of coccygeal and radial artery Doppler blood pressure measurements in sick cats with and without abnormalities in tissue perfusion. J Vet Emerg Crit Care (San Antonio) 2021;31:749-757.
- Cerna P, Archontakis PE, Cheuk HO, et al. Comparison of Doppler ultrasonic and oscillometric devices (with or without proprietary optimisations) for non-invasive blood pressure measurement in conscious cats. J Feline Med Surg 2021;23:121-130.
- Calero Rodriguez A, van Zeeland YR, Schoemaker NJ, et al. Agreement between invasive and oscillometric arterial blood pressure measurement using a high-definition oscillometric device in normotensive New Zealand White rabbits using two different anaesthetic protocols. Vet Anaesth Analg 2021;48:679-687.
- Cremer J, da Cunha A, Aulakh K, et al. Validation of the oscillometric blood pressure monitor Vet20 SunTech in anesthetized healthy cats. Vet Anaesth Analg 2020;47:309-314.
- Cerejo SA, Teixeira-Neto FJ, Garofalo NA, et al. Effects of cuff size and position on the agreement between arterial blood pressure measured by Doppler ultrasound and through a dorsal pedal artery catheter in anesthetized cats. Vet Anaesth Analg 2020;47:191-199.
- Hori Y, Heishima Y, Yamashita Y, et al. Epidemiological study of indirect blood pressure measured using oscillometry in clinically healthy cats at initial evaluation. J Vet Med Sci 2019;81:513-516.
- Zeugswetter FK, Tichy A, Weber K. Radial vs coccygeal artery Doppler blood pressure measurement in conscious cats. J Feline Med Surg 2018;20:968-972.
- Whittemore JC, Nystrom MR, Mawby DI. Effects of various factors on Doppler ultrasonographic measurements of radial and coccygeal arterial blood pressure in privately owned, conscious cats. J Am Vet Med Assoc 2017;250:763-769.
- Payne JR, Brodbelt DC, Luis Fuentes V. Blood Pressure Measurements in 780 Apparently Healthy Cats. J Vet Intern Med 2017;31:15-21.
- Cerejo SA, Teixeira-Neto FJ, Garofalo NA, et al. Comparison of two species-specific oscillometric blood pressure monitors with direct blood pressure measurement in anesthetized cats. J Vet Emerg Crit Care (San Antonio) 2017;27:409-418. Reineke EL, Rees C, Drobatz KJ. Prediction of systolic blood pressure using peripheral pulse palpation in cats. J Vet Emerg Crit Care (San Antonio) 2016;26:52-57.
- Sansom J, Rogers K, Wood JL. Blood pressure assessment in healthy cats and cats with hypertensive retinopathy. Am J Vet Res 2004;65:245-252.
- Pedersen KM, Butler MA, Ersbøll AK, et al. Evaluation of an oscillometric blood pressure monitor for use in anesthetized cats. J Am Vet Med Assoc 2002;221:646-650.
- Sparkes AH, Caney SM, King MC, et al. Inter- and intraindividual variation in Doppler ultrasonic indirect blood pressure measurements in healthy cats. J Vet Intern Med 1999;13:314-318.
- Mishina M, Watanabe T, Fujii K, et al. Non-invasive blood pressure measurements in cats: clinical significance of hypertension associated with chronic renal failure. J Vet Med Sci 1998;60:805-808.
- Caulkett NA, Cantwell SL, Houston DM. A comparison of indirect blood pressure monitoring techniques in the anesthetized cat. Vet Surg 1998;27:370-377.
- Bodey AR, Sansom J. Epidemiological study of blood pressure in domestic cats. J Small Anim Pract 1998;39:567-573.
- Branson KR, Wagner-Mann CC, Mann FA. Evaluation of an oscillometric blood pressure monitor on anesthetized cats and the effect of cuff placement and fur on accuracy. Vet Surg 1997;26:347-353.
- Binns SH, Sisson DD, Buoscio DA, et al. Doppler ultrasonographic, oscillometric sphygmomanometric, and photoplethysmographic techniques for noninvasive blood pressure measurement in anesthetized cats. J Vet Intern Med 1995;9:405-414.
- Grandy JL, Dunlop CI, Hodgson DS, et al. Evaluation of the Doppler ultrasonic method of measuring systolic arterial blood pressure in cats. Am J Vet Res 1992;53:1166-1169.
- McLeish I. Doppler ultrasonic arterial pressure measurement in the cat. Vet Rec 1977;100:290-291.

Hepatic lipidosis in cats: emphasis on nutritional support

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Learning objectives

- Recognize feline hepatic lipidosis based on anamnesis, clinical signs, blood results, medical imaging as well as hepatic cytology/histology.
- Understand the importance of nutritional support during feline hepatic lipidosis.
- Describe the advantages and disadvantages of the different enteral nutrition tubes and be able to use them.

Lecture summary

Definition and etiology: Feline hepatic lipidosis (FLH), first reported in 1977, is the most common liver disease in cats. It is characterized by triglyceride accumulation in >80% of hepatocytes resulting in an increase of >50% in liver weight, secondary liver dysfunction and intrahepatic cholestasis, and develops in association with profound anorexia. Obese and overweight cats are at increased risk, and likely also account for the overlapping age (middle-aged adults) and geographic distribution (i.e. feeding habits of owners and obesity prevalence). Most cases of FHL occur secondary to an underlying condition that has led to a period of anorexia, which may be as short as 2-7 days. Co-morbidities include diabetes mellitus, pancreatitis, inflammatory bowel disease, cholangitis, renal disease, and neoplasia. The pathophysiology of FLH is not fully understood, but may consist of alternations in pathways of uptake, synthesis, degradation, and secretion of fatty acids exacerbated by negative energy balance.
Clinical presentation: On presentation, cats are commonly bright and alert despite profound anorexia, recent rapid weight loss (often > 25%) and jaundice. Additional clinical signs may reflect the underlying condition. Non-painful hepatomegaly is usually readily palpable. Neck ventroflexion is suggestive of severe hypokalemia. Coagulopathy secondary to decreased vitamin K absorption can result in easy bruising. Most consistent laboratory findings include poikilocytosis, Heinz bodies +/- mild anemia, hyperbilirubinemia, hypoalbuminemia, and increased ALP. ALT activity is less consistently increased. Whereas increases in GGT tend to parallel increases in ALP in other forms of liver disease, GGT is frequently normal in FHL.Abdominal ultrasound typically reveals an enlarged hyperechoic liver that is not diagnostic for FHL, but allows investigation into underlying conditions. Diagnosis is usually confirmed by fine needle aspiration cytology, Liver biopsy may be eventually warranted to investigate concurrent liver disease but is usually avoided early on due to hemorrhagic risk from a friable fatty liver and concomitant coagulopathy
Management: Early nutritional support is at the heart of therapeutic success, but immediate attention must be given to fluid therapy aiming at correcting hydration status as well as electrolytes and acid-base imbalances. Hypokalemia and hypophosphatemia may be severe and needs correction. Persistent hypokalemia despite IV supplementation may reflect concurrent hypomagnesemia, although IV supplementation is rarely required as magnesium is present in sufficient quantities in enteral diets. In cases of prolonged anorexia before presentation, altered glucose homeostasis as well as precipitous drops in potassium and phosphorus may be present secondary to refeeding syndrome; thus, daily monitoring (ideally BID) of glucose and electrolytes is essential for the first 72 hours. Dextrose supplementation is usually contraindicated and may exacerbate hyperglycemia since cats with FHL are typically glucose intolerant. Enteral nutrition should be initiated as early as possible and sustained until voluntary intake is sufficient to meet protein and caloric needs. A naso-esophageal/gastric tube is a great option in unstable patient as it is inexpensive, does not require anesthesia for placement and uses readily available supplies. Radiographic verification of its correct position is essential. Esophageal tube placement should be considered once the patient is stabilized. The tube is easy to place with a brief anesthesia, and will allow the use of blended canned food, feeding to eventually take place at home, as well as the administration of PO medications. Dietary proteins are most efficient at reducing hepatic lipid accumulation in cats with a negative energy balance.5 Enteral nutrition, either via bolus feed or CRI, should be introduced over several days with the aim of providing full RER by day 3-4. Smaller volumes are typically better tolerated. An example of a feeding record sheet with formulation for RER is available for download (2022 ISFM Consensus). Practical recommendations (diet selection, feeding schedule, complications of enteral feeding tubes) will be discussed during the presentation. To improve the cat's condition and food intake, nausea, vomiting, ileus and possibly pain must all be managed. Antiemetic therapy, typically using maropitant and metoclopramide CRI, is usually needed. In addition to vitamin K (coagulopathy), cobalamin parenteral supplementation is recommended since up to 40% of cats with FHL may have a subnormal level. Finally, cats suffering from FHL have low hepatic glutathione concentrations and could benefit from an antioxidant such as SAMe.
Prognosis: In the absence of serious underlying disease, early enteral feeding sustained until voluntary intake is resumed, time (weeks) and TLC from their owner will allow the gradual recovery of 80-85% of cats. Negative prognostic factors include older age, anemia, hypokalemia and hypoalbuminemia on admission.

References

- *Barsanti J, et al. Prolonged anorexia associated with hepatic lipidosis in 3 cats. Feline Pract 1977;7: 52–57.*
- *Valtolina C & Favier RP. Feline Hepatic Lipidosis. Vet Clin Small Anim 2017;47:683-702.*
- *Center SA. Feline hepatic lipidosis. Vet Clin North Am Small Anim Pract 2005;35(1):225–269.*
- *Center SA, et al. A retrospective study of 77 cats with severe hepatic lipidosis: 1975-1990. J Vet Intern Med 1993;7(6):349–359.*
- *Biourge VC, et al. Effects of protein, lipid, or carbohydrate supplementation on hepatic lipid accumulation during rapid weight loss in obese cats. Am J Vet Res 1994;55(10):1406–1415.*
- *Armstrong PJ, Blanchard G. Hepatic lipidosis in cats. Vet Clin North Am Small Anim Pract 2009;39(3):599-616.*
- *Lisciandro SC, et al. Coagulation abnormalities in 22 cats with naturally occurring liver disease. J Vet Intern Med 1998;12:71-75.*
- *Léveillé R, et al. Complications after ultrasound-guided biopsy of abdominal structures in dogs and cats: 246 cases (1984-1991). J Am Vet Med Assoc 1993;203:413-415.*
- *Taylor S, et al. 2022 ISFM Consensus Guidelines on Management of the Inappetent Hospitalised Cat. J Feline Med Surg. 2022 Jul;24(7):614-640. doi: 10.1177/1098612X221106353. PMID: 35775307.*
- *Cook S, et al. Retrospective evaluation of refeeding syndrome in cats: 11 cases (2013-2019). J Feline Med Surg. 2021 Oct;23(10):883-891. doi: 10.1177/1098612X20979706. Epub 2021 Jan 6. PMID: 33404281.*
- *Biourge V, et al. Effect of weight gain and subsequent weight loss on glucose tolerance and insulin response in healthy cats. J Vet Intern Med 1997;11:86-91.*
- *Simpson KW, et al. Subnormal concentrations of serum cobalamin (vitamin B12) in cats with gastrointestinal disease. J Vet Intern Med 2001;15:26-32.*
- *Blanchard G, et al. Plasma lipids, lipoprotein composition and profile during induction and treatment of hepatic lipidosis in cats and the metabolic effect of one daily meal in healthy cats. J Anim Physiol Anim Nutr (Berl) 2004;88(3-4):73-87.*

Management of canine and feline GI motility disorders in the intensive care unit

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Learning objectives

- Provide an overview of the pathogenesis of GI dysmotility in critical care and postoperative patients.
- Review the clinical signs of delayed gastric emptying and decreased GI motility.
- List the methods available to evaluate GI motility in dogs and cats in the clinical setting.
- Review the evidence on the clinical use of GI prokinetics and other support in people, dogs and cats with post-operative ileus and critical illness-related motility disorders.

Lecture summary

Disorders of gastrointestinal motility often represent a diagnostic and therapeutic challenge. The prevalence of non-obstructive GI motility disorders in dogs and cats is difficult to estimate because of the absence of appropriate epidemiologic studies.

Postoperative ileus (POI): It is a common complication of abdominal surgery in people which may prolong the length of hospital stay and increase morbidity in affected patients. The origin of POI is multifactorial, and manipulation of the intestines, administration of opioids, and post-operative stress all contribute to the problem. While the return of appetite following abdominal surgery is usually rapid in dogs, post-operative GI motility was decreased when measured with wireless motility capsules, breath test or pressure transducers. Clinical signs of POI in dogs include prolonged anorexia, nausea, vomiting, regurgitation, cranial abdominal discomfort, abdominal distension, and bloated abdomen. Intestinal sounds are usually absent on abdominal auscultation.

GI dysmotility in ICU patients: Critical illness-related motility disorders (CIRMD) are a prevalent concern in human ICU patients. Clinical signs include vomiting, abdominal distention, complaints of discomfort, and high gastric residual volumes. Abnormal gastric emptying compromises the effectiveness of enteral nutrition, an essential component of treatment. High gastric residual volume (GRV) may predispose patients to macro- or micro-aspiration with resulting airway disease and increase the risk for ventilator dependence.
There is no data documenting the existence of CIRMD in canine and feline ICU patients. However, most internists and criticalists commonly manage adynamic ileus and delayed gastric emptying in critically ill dogs and cats with diseases such as severe acute pancreatitis, gastroenteritis, and peritonitis among others. Clinical signs resemble those observed in POI, in association with signs of the primary underlying disease. Typically, abdominal radiographs show moderately distended bowel loops filled with gas and liquid content, and abdominal ultrasound reveals distended and hypomotile stomach and intestine. The presence of POI or CIRMD may increase morbidity and mortality and prolong the duration of hospitalization of dogs and cats housed in the ICU.

Pain management and GI motility: Mu-receptors are present on the myenteric plexus and submucosal neurons and inhibit the contraction of smooth muscle in the tunica muscularis. Their activation by exogenous opioids administered to control post-operative and critical illness-associated pain further compromises GI motility. Opioid-sparing lidocaine CRI is preferred as a 1st line analgesic in critical patients with abdominal pain (initially 0.025 mg/kg/min IV CRI after loading bolus of 0.5 mg/kg IV). It may also decrease the severity of POI and CIRMD through the drug's antinociceptive, anti-hyperalgesic, and anti-inflammatory properties.

Management of GI motility disorders in dogs and cats: Proper diagnosis and treatment of any underlying disease that might affect GI motility is an essential premise. If severe gastric fluid retention is observed on ultrasound and the animal is at risk of aspiration, placement of a nasogastric tube and suction of gastric content may be useful. An adapted diet is an essential part of the management and a commercial liquid diet with a low-fat content is commonly used in canine and feline ICU patients particularly if they are tube fed. The available published evidence on the effect of various prokinetic drugs in healthy dogs and cats is tenuous, while it is almost nonexistent for dogs and cats with GI dysmotility. Therefore, collective clinical experience plays an important role in most recommendations for drug usage and dose. They should only be administered after GI obstruction has been ruled out. Commonly used prokinetics for dogs include metoclopramide, a selective $5HT_4$ receptor agonist and $5HT_3$ receptor and dopamine type-2 receptor antagonist, which is administered as CRI of 1-2 mg/kg/day. In addition, macrolide antibiotics and motilin receptor agonists can be useful such as erythromycin 0.5-1.0 mg/kg IV q8h and azithromycin 2 mg/kg IV, PO q8h. Timely recognition and appropriate management of CIRMD often improve outcomes in diseases such as acute pancreatitis or severe gastroenteritis or enterocolitis.

References

- *Husnik R and Gaschen F. Gastric motility disorders in dogs and cats. Vet Clin North Am Small Anim Pract 2021;51:43-59.*
- *Deane AM, Chapman MJ, Reintam Blaser A, et al. Pathophysiology and treatment of gastrointestinal motility disorders in the acutely ill. Nutr Clin Pract; 2019;34:23-36.*

Digestive tract assessment with pocus: what's new in ICU?

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Learning objectives

- Become familiar with POCUS images consistent with small bowel obstruction.
- Understand the interest of digestive motility assessment.
- Describe how digestive motility can be assessed using POCUS.
- List POCUS views where tube feeding position can be confirmed.

Lecture summary

For decades now, Point of Care Ultrasound (POCUS) is used by emergency and critical care veterinarians to early detect abdominal free fluid potentially due to gastro-intestinal tract (GIT) perforation. Today, POCUS allows for a closer look at the GIT and obtain more information.

Digestive tract obstruction: Segmental dilation of intestinal loops and "back and forth" movement of intestinal content can suggest mechanical ileus. Abdominal POCUS performed in an emergency department has shown to have good sensitivity and specificity to diagnose small bowel obstruction (SBO). Five POCUS criteria are used in humans to diagnose and stage a SBO: intestinal dilation, GI intraluminal air, abnormal intestinal peristalsis, interloop free fluid and a thickened intestinal wall. The accuracy of interpretation could be increased with prior training and familiarity with sonographic appearance of SBOase. This assessment could be limited by GI gas and the physical condition of the patient. Compared to computed tomography, radiographs or complete ultrasound by a specialist, the use of POCUS to diagnose digestive obstruction could decrease time and cost and improve the management of patients. Ultrasonographic target-like appearance of intussusception is characteristic and helps differentiate this disease from feces or a foreign material. Moreover, authors detail external manual reduction of intestinal intussusceptions with ultrasound assistance.

Digestive tract motility: Delayed gastric emptying due to illness or medication is the most frequent gastric dysmotility in small animals and can have a negative impact such as impaired food delivery, increased intra-abdominal pressure and aspiration pneumonia. Ultrasonographic assessment of the antral area correlates with gastric residual volume in human patients. This technique is applicable in small animals with a low operator variability. Moreover, the motility index reflects gastric antral motility and can be obtained in dogs and cats. Motility can also be assessed in all GIT segments. Its assessment helps to guide prokinetics administration and detect post-operative complications.

Digestive feeding tube placement: Feeding tube placement is mandatory in malnourished patients and after several days of anorexia. Tube misplacement leads to significant morbidity and mortality. End-positioning of the tube in the stomach allows gastric decompression in addition to enteral feeding. Radiographic confirmation of proper positioning of the feeding tube in the GIT is the currentstandard. In critical care patients, monitoring devices and cardiopulmonary instability could limit transport of the patient. In people, ultrasound of the cervical esophagus and the stomach is a reliable technique to asses correct tube placement. Several techniques are described for this detection: direct visualization of the tube with transversal or longitudinal views of the cervical esophagus during placement, direct visualization of the tube in the stomach using the sub-xiphoid view of POCUS and indirect visualization of air or liquid filling the stomach using the same sub-xiphoid view. A 4-point ultrasonographic procedure has a high sensitivity to confirm correct position of a nasogastric tube in 10 minutes. Such confirmation is currently studied in critically ill small animals. In dog cadavers, a novel ultrasound-guided percutaneous dilatational esophagostomy tube placement technique has been described to avoid oral contamination of instruments. In canine and feline neonates, ultrasound can be used to check orogastric tube placement before feeding.

References

- *Becker BA, Lahham S, Gonzales MA, Nomura JT, et al. A Prospective, multicenter evaluation of point-of-care ultrasound for small-bowel obstruction in the emergency department. Acad Emerg Med 2019;26(8):921-930.*
- *Choi M, Seo M, Jung J, et al. Evaluation of canine gastric motility with ultrasonography. J Vet Med Sci 2002 Jan;64(1):17-21.*
- *Furthner E, Kowalewski MP, Torgerson P, et al. Verifying the placement and length of feeding tubes in canine and feline neonates. BMC Vet Res 2021;17(1):208.*
- *Gok F, Kilicaslan A, Yosunkaya A. Ultrasound-guided nasogastric feeding tube placement in critical care patients. Nutr Clin Pract 2015;30(2):257-260.*
- *Husnik R, Gaschen FP, Fletcher JM, et al. Ultrasonographic assessment of the effect of metoclopramide, erythromycin, and exenatide on solid-phase gastric emptying in healthy cats. J Vet Intern Med 2020;34(4):1440-1446.*
- *Jimeno Sandoval JC, Peak K, Moores AL. Letter to the Editor: External manual reduction of intestinal intussusceptions with ultrasound assistance. J Vet Emerg Crit Care (San Antonio). 2021;31(5):674.*
- *Li RT, Zhao Y, Zou XJ, et al. Overview of point-of-care ultrasound in diagnosing intestinal obstruction. World J Emerg Med 2022;13(2):135-140.*
- *Liao PT, Lawson CM, Zellner E. Description of a novel ultrasound-guided percutaneous dilatational esophagostomy tube placement in dog cadavers. J Vet Emerg Crit Care (San Antonio) 2022 33(2):263-266.*
- *Pourmand A, Dimbil U, Drake A, et al. The Accuracy of Point-of-Care Ultrasound in Detecting Small Bowel Obstruction in Emergency Department. Emerg Med Int 2018;2018:3684081.*
- *Sanderson JJ, Boysen SR, McMurray JM, et al. The effect of fasting on gastrointestinal motility in healthy dogs as assessed by sonography. J Vet Emerg Crit Care (San Antonio) 2017;27(6):645-650.*
- *Tsukamoto A, Ohno K, Tsukagoshi T, et al. Real-time ultrasonographic evaluation of canine gastric motility in the postprandial state. J Vet Med Sci 2011;73(9):1133-1138.*
- *Vigneau C, Baudel JL, Guidet B, et al. Sonography as an alternative to radiography for nasogastric feeding tube location. Intensive Care Med 2005;31(11):1570-1272.*
- *Zatelli M, Vezzali N. 4-Point ultrasonography to confirm the correct position of the nasogastric tube in 114 critically ill patients. J Ultrasound 2016;20(1):53-58.*

Gastroprotectants in critical patients

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Learning objectives

- Review the indications for use of gastroprotectants (acid suppressants and sucralfate) in dogs and cats with critical illnesses.
- Explain the risks associated with the excessive use of gastroprotectants in dogs and cats.
- Facilitate the decision to use proton pump inhibitors (PPI) or histamine type-2 receptor antagonists (H2RA) to increase gastric pH in dogs and cats.
- Make suggestions for the implementation of gastroprotective therapy in critically ill dogs and cats.

Lecture summary

Gastroprotectants are easily available and commonly used in small animal medicine. The optimal dosing regimen of gastric acid suppressants has been established for proton pump inhibitors (PPI) and histamine type-2-receptor antagonists (H2RA). A 2018 ACVIM consensus statement reviewed the available evidence and made recommendations for the optimal use of gastroprotectants in dogs and cats which are summarized at the end of this text. However, two recent studies showed that these drugs are often used in situations for which they have no documented benefits. A review of prescriptions for omeprazole and maropitant in the medicine and surgery wards of a UK referral and teaching veterinary hospital found that both drugs were overprescribed. In addition, omeprazole was often administered for reasons incompatible with its known effects. A retrospective evaluation of medical records of a tertiary referral and teaching hospital in the USA over 5 years between 2013 and 2018 identified an adequate indication for use of PPI in only 27% of the cases that received these drugs. In addition, the drugs were prescribed at an inadequate dose in half of cases. New data have become available since the ACVIM consensus statement on different clinically relevant aspects:

What are the best acid suppressants? In dogs, a famotidine CRI of 8 mg/kg/day after a 1 mg/kg IV bolus was effective in maintaining the gastric pH within the range required for mucosal healing unlike what occurred after multiple IV injections of the drug. Moreover, esomeprazole at 1 mg/kg q12h IV and famotidine administered as a CRI appeared superior to pantoprazole at 1 mg/kg q12h in maintaining a gastric pH above 3 or above 4 for a longer period. Does simultaneous administration of PPI and famotidine accelerate the increase in gastric pH? Initiating antacid treatment with both pantoprazole and famotidine did not provide a more rapid increase in gastric pH over treatment with pantoprazole only.

Does famotidine remain effective when administered for more than a few days? Continued administration of famotidine over 14 days resulted in a diminished effect on intragastric pH in dogs and in cats. Therefore, caution is advised when recommending long-term, daily oral administration of famotidine. What are the long-term complications of gastric acid suppression? Hypergastrinemia occurred in healthy dogs and cats treated with omeprazole. However, after 60 days of treatment, bone density and calcium metabolism parameters were not affected in cats.

Does rebound acid hypersecretion (RAH) occur after long-term treatment with PPI, and should PPI be progressively (i.e., not abruptly) withdrawn? RAH occurred in a few cats included in the 60-day PPI. The ACVIM consensus statement recommends: "Proton pump inhibitors should be gradually tapered after administration for ≥4 weeks to avoid rebound gastric acid hypersecretion The dose can be decreased by 50% on a weekly basis, with cessation of evening dosing during the first week."

Should PPI be used systematically in cats with chronic kidney disease (CKD)? Cats with CKD do not appear to have gastric hyperacidity or hypergastrinemia when compared to healthy cats and therefore do not need acid suppression. Finally, once-daily omeprazole (1 mg/kg PO) did not markedly increase appetite in cats with IRIS stage 2 or 3 CKD and should therefore not be used as a first-line treatment in the absence of GI ulceration. Should acid suppressants be used to prevent GI erosions or ulcerations in animals receiving NSAIDs? In a recent prospective, placebo-controlled, double-blinded study, oral administration of omeprazole (1 mg/kg q12h) or famotidine (1 mg/kg q12h) to dogs with cancer treated with piroxicam markedly increased both the number and severity of GI adverse events. Another study showed that omeprazole prophylaxis induced fecal dysbiosis (i.e., perturbed the gut microbiome) and increased intestinal inflammatory markers when co-administered with carprofen to otherwise healthy dogs with no other risk factors for GI bleeding.

Recommendations made in the 2018 ACVIM Consensus Statement:

Indications for gastric acid suppressants

• Gastroduodenal ulceration and erosion • Esophagitis secondary to gastroesophageal reflux (treatment and prevention).

Situations for which there is weak evidence of benefits from gastric acid suppressants

• Liver disease not associated with gastroduodenal ulceration and erosion • Stress-related GI mucosal damage.

Situations for which benefits of gastric acid suppressants are lacking

• Chronic kidney disease, IRIS stages I-III in dogs and cats • Acute or chronic gastritis, including helicobacter infections • Pancreatitis, unless there is evidence of gastroduodenal ulceration and erosion or a significant risk for esophagitis • Thrombocytopenia-induced GI bleeding • Prevention or management of glucocorticoid-associated gastroduodenal ulceration in dogs with spinal cord injury.

Situations for which gastric acid suppressants are contraindicated

• Prevention of gastroduodenal ulceration and erosion in dogs receiving NSAIDs.

References

• *Bazelle J, Threlfall A, Whitley N. Gastroprotectants in small animal veterinary practice – a review of the evidence. Part 1: cyto-protective drugs. J Small Anim Pract 2018. DOI: 10.1111/jsap.12867*
• *Duxbury s, Sorah E, Tolbert MK. Evaluation of proton pump inhibitor administration in hospitalized dogs in a tertiary referral hospital. J Vet Intern Med 2022;36:1622-1627.*
• *Marks SL, Kook PH, Papich MG, et al. ACVIM consensus statement for rational administration of gastrointestinal protectants to dogs and cats. J Vet Intern Med 2018;32(6):1823-1840.*
• *McCormack R, Olleay L., Glanemann B et al. Prospective observational study of the use of omeprazole and maropitant citrate in veterinary specialist care. Scientific Reports 2020;10:15727.*

The dark side of pancreatitis - internal medicine and critical care views on the management of complications associated with acute pancreatitis

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Learning objectives

- Recognize possible complications of acute pancreatitis.
- Describe the possible treatments and associated risks of extra-hepatic biliary tract obstruction in pancreatitis.
- Identify when the use of antithrombotic should be considered in acute pancreatitis.
- Describe management of thrombotic complications in pancreatitis.

Lecture summary

Background: Pancreatitis is a common condition in dogs and cats, but its diagnosis and management remain challenging. Acute pancreatitis (AP) is defined by the presence of neutrophilic inflammation within the body of the pancreas, and potentially extending to the peri-acinar fat, whereas the presence of fibrosis or acinar atrophy characterize chronic pancreatitis. Inflammation of pancreatic tissue leads to the passage of pancreatic enzymes into the peritoneal cavity and into the portal circulation potentially resulting in significant systemic complications such as ileus, hepatic necrosis, acute kidney injury (AKI), etc. Severity varies from mild to necrotic, often fatal, hemorrhagic pancreatitis. Although most often considered idiopathic, many etiologies or risk factors have been proposed, notably in dogs, such as hypertriglyceridemia, endocrine disease, adverse drug reaction (e.g. steroids, furosemide, azathioprine, sulfas), prior surgery and dietary factors (fatty diet, dietary indiscretion). Although most often sterile (33/46), feline pancreatitis may be infectious as demonstrated using fluorescence in situ hybridization, particularly moderate to severe (11/31) compared to mild forms (2/15).

Diagnosis: Histopathology remains the gold standard for diagnosis but is rarely performed due to its invasive nature and inherent limitations including missing localized lesions. Therefore, clinical signs combined with ultrasonographic findings and measurement of serum pancreatic lipase immunoreactivity (cPLI or fPLI) are routinely used. Mild to moderate pancreatitis is characterized by clinical signs such as anorexia, abdominal pain, vomiting and lethargy. In severe pancreatitis, acute pancreatic necrosis results in more severe clinical signs and multisystem complications such as systemic inflammatory response syndrome (SIRS), multiple organ dysfunction syndrome (MODS), or disseminated intravascular coagulation (DIC). Anorexia is commonly the only finding in cats. Improved technology and training have improved the diagnostic sensitivity of abdominal ultrasound (AUS), but it remains limited. A retrospective study in dogs concluded once more that AUS should not be used alone to diagnose pancreatitis and is a poor indicator of severity. AUS changes included pancreatic enlargement, echogenicity and altered mesenteric echogenicity with sensitivity and specificity of 89% and 43% (one criteria), and 43% and 92% (three criteria).6 Advanced imaging such as endoscopic ultrasound, CT or MRI have not yet been established as a routine diagnostic tool. Assays that detect lipase specific to the pancreas have become widely available and are considered the clinicopathological tests of choice for the diagnosis of both canine and feline pancreatitis. Cage-side semi-quantitative tests perform reasonably well. The sensitivity and specificity of qualitative tests in dogs ranges from 74-100% and 64-83%, respectively, with a better performance as the severity of pancreatitis increases. Given the limitations of the assays, it is crucial to interpret the results based on clinical signs and the presence of concomitant diseases. In both dogs and cats, increased 1,2-o-dilauryl-rac-glycero-3-glutaric acid-(6'-methylresorufin) ester (DGGR) lipase has been shown to correlate closely with increased pancreatic specific lipase (Spec PLI) which may be a useful economic tool for diagnosis and follow-up. To allow early prediction of short-term death, a Canine Acute Pancreatitis Severity (CAPS) score was proposed based on identified independent risk factors: presence of SIRS, coagulation disorders, increased creatinine, and ionized hypocalcemia.

Management: Early targeted fluid therapy, aiming at correction of dehydration, acid-base and electronic imbalances, is critical in severe AP. Crystalloids (Lactated Ringer preferred) are the first line fluids used, with colloids reserved for patients with proven low oncotic pressure. Unless DIC is present, there is no benefit in using fresh frozen plasma in terms of alpha-macroglobulin intake. Multi-modal analgesia, such as combination of opioids with lidocaine +/- ketamine, is often required. In human medicine, thoracic epidural may have prognostically beneficial effects due to suspected anti-inflammatory effects and increased splanchnic perfusion. Interestingly, early refeeding is part of the strategies to reduce pain. Early interventional feeding is now advocated in AP and is usually well tolerated. Antibiotic therapy remains controversial, but may be considered in patients with high risk of bacterial translocation or in severe feline pancreatitis. Corticosteroids are typically reserved for patients non-responsive to fluid resuscitation, although a recent study has suggested their use in the initial treatment of canine AP, resulting in earlier improvement of clinical signs.

Complications: Systemic complications such as AKI, acute respiratory distress syndrome, diabetes ketoacidosis or cardiac arrhythmias are treated on an individual basis. The coagulation status should be carefully monitored, and antithrombotic therapy initiated in most severe cases to avoid thrombotic complications. In case of local complications, such as acute fluid collections or extra-hepatic bile duct obstruction, ultrasound-guided intervention and/or biliary stent placement may be warranted. Surgical interventions result in high mortality rate and are usually reserved for infected pancreas (e.g. abscess) or gallbladder rupture.

References

- *Xenoulis PG. Diagnosis of pancreatitis in dogs and cats. J Small Animal Pract 2015;56:13-26.*
- *Watson P. Pancreatitis in dogs and cats: definitions and pathophysiology. J Small Animal Pract 2015;56:3-12.*
- *Lem, KY, Fosgate GT, Norby B, et al. Associations between dietary factors and pancreatitis in dogs. J Am Vet Med Assoc 2008;233:1425-1431.*
- *Simpson KW. Pancreatitis and triaditis in cats: causes and treatment. J Small Animal Pract 2015;56:40-49.*
- *Forman AM, et al. ACVIM consensus statement on pancreatits in cats. J Vet Intern Med 2021;35:703-723.*
- *Cridge H et al. Association between abdominal ultrasound findings, the specific canine pancreatic lipase assay, clinical severity indices, and clinical diagnosis in dogs with pancreatitis. J Vet Intern Med 2020;34:636-643.*
- *Cridge H, MacLeod AG, et all. Evaluation of SNAP cPL, Spec cPL, VetScan cPL Rapid Test, and Precision PSL Assays for the diagnostic of clinical pancreatitis in dogs. J Vet Intern Med 2018;32:658-664.*
- *Lee C, Kathrani A, et all. Retroscpective study of the diagnostic utility of Spec fPL in the assessment of 274 sick cats. J Vet Intern Med 2020;34:1406-1412.*
- *Kook PH, Kohler N, et all. Agreement of serum Spec cPL with the 1,2-o-dilauryl-rac-glycero glutaric acid-(6'-methylresorufin) ester (DGGR) lipase assay and with pancreatic ultrasonography in dogs with suspected pancreatitis. J Vet Intern Med 2014;28:863-870.*
- *Oppliger S, Hartnack S, et al. Agreement of the serum Spec fPLTM and 1,2-o-dilauryl-rac-glycero-3-glutaric acid-(6'-methylresorufin) ester lipase assay for the determination of serum lipase in cats with suspicion of pancreatitis. J Vet Intern Med 2013;27:1077-1082.*
- *Fabrès V, Dossin O, Reif C, et al. Development and validation of a novel clinical scoring system for short-term prediction of death in dogs with acute pancreatitis. J Vet Intern Med. 2019;33(2):499-507.*
- *Gliem et al. Management of severe acute pancreatitis: an update. Digestion 2021;102(4):503-507.*
- *Mansfield C and Beths T. Management of acute pancreatitis in dogs: a critical appraisal with focus on feeding and analgesia. J Small Animal Pract 2015;56:27-39.*
- *Weatherton LK, Streeter EM. Evaluation of fresh frozen plasma administration in dogs with pancreatitis: 77 cases (1995-2005). J Vet Emerg Crit Care 2009;19:617-622.*
- *Harris JP, Parnell NK, et all. Retrospective evaluation of the impact of early enteral nutrition on clinical outcomes in dogs with pancreatitis: 34 cases (2010-2013). J Vet Emerg Crit Care 2017;27(4):425-433.*
- *Chan DL, Jensen KB. Nutritional management of acute pancreatitis in dogs and cats. J Vet Emerg Crit Care 2014;24(3):240-250.*
- *Okanishi H, et al. Comparison of initial treatment with and without corticosteroids for suspected acute pancreatitis in dogs. J Small Anim Pract 2019;60:298-304.*
- *Gori E, et al. Acute pancreatitis and acute kidney injury in dogs. Vet J. 2019;245:77-81.*
- *deLaforcade A, Bacek L, Blais MC, et al. Update of the consensus on the rational use of antithrombotics and thrombolytics in veterinary critical care (CURATIVE) Domain 1 - Defining populations at risk. J Vet Emerg Crit Care 2022;32(3):1-26.*
- *Blais MC et al. Consensus on the rational use of antithrombotics in veterinary critical care (CURATIVE): Domain 3 - Defining antithrombotic protocols. J Vet Emerg Crit Care 2019;29(1):60-74.*

Cardiac trauma

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Learning objectives

- Describe mechanisms and types of cardiac trauma.
- Discuss the available diagnostic tools.
- Appraise selected treatment options.

Lecture summary

Several mechanisms and types of cardiac injury exist. Because the term cardiac or myocardial contusion is considered non-specific and lacks a clear definition, some authors replace it with blunt cardiac injury (BCI) as a catch-all term used to describe non-penetrating trauma to the heart. In humans, cardiac injury following blunt thoracic trauma has been reported in up to 76% of patients. The incidence of BCI in dogs and cats is unknown. The clinical presentation of a dog, cat or human with BCI is extremely variable in terms of injury severity, cardiac consequences and clinical consequences. Some patients with BCI seem unaffected; others show minimal clinical consequences, despite evidence of cardiac injury; others present in various states of clinical compromise, including death. Importantly, trauma affects the whole body. In the traumatized patient with tachycardia, arrhythmias, or other possible signs of cardiac injury, it is important to consider the whole patient - i.e., treat shock, hemorrhage, pain, etc. - rather than hyper-focusing on the heart. .

References

- *Singh S, Heard M, Pester JM, et al. Blunt cardiac injury. Stat Pearls [Internet] [Accessed February 28, 2023].*
- *Nair L, Winkle B, Senanayake E. Managing blunt cardiac injury. J Cardiothorac Surg 2023;18(1):71.*
- *Oldach MS. Blunt cardiac injury. In: Silverstein D.S., Hopper K, editors. Small Animal Critical Care edicine, M edition. St. Louis, USA, Elsevier Saunders. 2022:66-270.*

Diastology and left atrial filling pressure

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Learning objectives

- Summarize the key events during diastole
- Understand the contribution of left atrial pressure to the pulmonary circuit
- Explore echocardiographic tools to estimate left atrial filling pressure

Lecture summary

Diastology is the study of the heart as it relaxes (i.e., when it is not in systole). Ventricular diastole includes the filling period, where blood is transferred from the atria to the ventricles. As the ventricles are downstream to the atria, blood can only be transferred when the intra-atrial pressure is greater than the intraventricular pressure. If the diastolic pressure in a ventricle increases, the pressure in the afferent atrium must increase to maintain forward flow. The left atrium is downstream of the pulmonary circuit. If the pressure in the left atrium increases, the pressure in the pulmonary circuit must increase. This is post-capillary pulmonary hypertension. The pulmonary circulation can only tolerate increasing filling pressure to a point, beyond which pulmonary oedema develops. The commonly cited threshold is 18 mmHg (approximately 25 cmH2O), though patients with chronic heart failure may tolerate higher pressures. Knowledge of left atrial pressure may be useful in the critically ill patient, for example when assessing volume status or fluid tolerance, when investigating the origin of pulmonary oedema and when considering diuresis. In human medicine, the American Society of Echocardiography (ASE) and the European Association of Cardiovascular Imaging (EACVI) provide recommendations for echocardiographic assessment of diastolic function. These guidelines were not written for intensive care patients, though have been used to evaluate critically ill humans. Data on the assessment of diastolic function are available for dogs and cats, though there is no consensus on their use in critically ill patients. The contents of this lecture do not replace the integrated clinical approach to a patient.

Causes of elevated left atrial pressure

1. Diastolic dysfunction
2. Systolic dysfunction
3. Latrogenic fluid overload
4. Eccentric left ventricular overload (e.g., degenerative mitral valve disease or dilated cardiomyopathy)
5. Mitral regurgitation
6. Mitral stenosis

The ASE/EACVI recommended parameters

1. Left atrial size – The left atrium dilates with chronically increased pressure.1 A dilated left atrium does not necessary indicate currently elevated left atrial pressure, as it depends on atrial filling volume and the atrial compliance curve. With acute increase in LAP, the left atrium may not be dilated.
   i. Left atrial diameter (LAD) is the 2D measurement of the minor axis of the left atrium via the right parasternal long axis 4 chamber view. Measurements are made just before the opening of the mitral valve, parallel to the annulus. In cats, the normal LAD is < 16 mm; values > 19 mm are often found in heart failure. For dogs, LAD is normalized to bodyweight (nLAD = LAD/BW0.31) and should be < 1.7.4
   ii. Left atrial-to-aortic diameter (LA:Ao) is commonly used. The left atrium is indexed to the aorta when measured in the right parasternal short axis view at the level of the heart base. Normal values for dogs and cats are < 1.6.
   iii. Left atrial volume (LAV) can be measured via the right parasternal long axis 4 chamber view just before the opening of the mitral valve. Values ≤ 1.5 ml/Kg are expected in normal dogs. In dogs with mitral valve disease, values < 2.25 ml/Kg suggest against congestive heart failure.
2. Transmitral flow – The inflow into the left ventricle from the left atrium is graphically mapped using Doppler echocardiography as E and A waves. E is the early filling that occurs as the mitral valve opens and is governed by atrial-ventricular pressure differences and left atrial volume. A is the result of atrial contraction. Classic profiles exist in patients with diastolic dysfunction.
   i. Normal patterning is characterized by an E wave peak greater than the A wave (E>A). Blood is sucked from the left atrium by the relaxing left ventricle. The maximal speed of the E wave is approximately 1 m/s.
   ii. Delayed relaxation (grade 1 diastolic dysfunction) is characterized by E<A. Delayed relaxation reduces ventricular sucking, so there is decreased early ventricular filling. This pattern indicates that left atrial pressure is not elevated (as the volume, thus pressure, of the left atrium has not been augmented to overcome the delayed relaxation in early diastole).

iii. Pseudonormal (grade 2 diastolic dysfunction) is when the normal E>A pattern returns. The volume-pressure of the left atrium has increased. The increased pressure pushes blood from the left atrium into the ventricle. This pattern appears normal, making it difficult to interpret; however, LAP is increased and patients are at risk of heart failure/overload/pulmonary oedema. This pattern can occur with healthy patients (normal), increased sympathetic tone and volume overload. Importantly, any changes to the left heart (for example, hypertrophy in cats, dilation in dogs or increased left atrial size in both) supports a pseudonormal pattern and volume intolerance. In dogs with mitral valve disease, maximum E velocity < 1.2 m/s suggests against congestive heart failure (i.e., elevated LAP) and > 1.5 m/s is supportive (but not diagnostic) of elevated LAP.

iv. Restrictive (grade 3 diastolic dysfunction) occurs when diastolic function worsens further; the left ventricle relaxes poorly and its distensibility is reduced (decreased compliance). The E wave > 2 is supportive of high LAP.

3. Tissue Doppler Imaging (TDI) of the mitral annulus is an advanced form of echocardiography. It is very useful in assessing diastolic function. The interested reader is referred to the references.
4. Tricuspid regurgitation - It is normal for the tricuspid valve to leak (i.e., tricuspid regurgitation [TR] during systole). The velocity of this regurgitation can be measured and roughly reflects the pulmonary artery systolic pressure. Class 2 pulmonary hypertension occurs secondary to left heart disease (i.e., elevated LAP). An increased velocity TR is not diagnostic for elevated LAP; however, a low velocity TR suggests against elevated LAP.

Additional methods

1. B-lines can be identified via lung ultrasound.8 In humans, an A-line lung pattern supports LAP < 18 mmHg.9 B-lines are concerning for elevated LAP; however, other aetiologies exist.8
2. Doppler peak systolic left ventricular-to-left atrial pressure gradient and systolic blood pressure technique – if the patient has a measurable mitral valve regurgitation, the left ventricular-to-left atrial systolic pressure gradient can be measured. By subtracting this gradient for the systolic blood pressure, the difference is the LAP: Systolic BP – (LVPs-LAPs) = LAP

Where systolic BP is measured in mmHg, LVPs is left ventricular pressure in systole (mmHg) and LAPs is left atrial pressure in systole (mmHg); (LVPs-LAPs) is derived from the peak velocity of the mitral regurgitation using continuous wave Doppler echocardiography and the modified Bernoulli equation. This technique requires further validation and is limited by image acquisition, Doppler alignment and the high prevalence of eccentric mitral regurgitation jets in dogs with mitral valve disease. The systolic blood pressure measured should be the central systolic pressure, so pressures measured at distal sites (for example, the dorsal pedal or metatarsal artery) may provide different estimates of LAP.

References

- *Bowcock EM and Mclean A. Bedside assessment of left atrial pressure in critical care: a multifaceted gem. Crit Care2022;26(1):247.*
- *Reinero C, Visser LC, Kellihan, et al. ACVIM consensus statement guidelines for the diagnosis, classification, treatment, and monitoring of pulmonary hypertension in dogs. J Vet Intern Med 2020;34(2):549-573.*
- *Nagueh SF, Smiseth OA, Appleton CP, et al. Recommendations for the Evaluation of Left Ventricular Diastolic Function by Echocardiography: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2016;29(4):277-314.*
- *Ware WA and Bonagura JD, editors. Cardiovascular disease in companion animals, 2nd edition. Boca Raton, FL, USA, CRC Press, Taylor & Francis Group, 2022.*
- *Visser LC, Ciccozzi MM, Sintov DJ, et al. et al. Echocardiographic quantitation of left heart size and function in 122 healthy dogs: A prospective study proposing reference intervals and assessing repeatability. J Vet Intern Med 2019;33(5):1909-1920.*
- *Höllmer M, Willesen JL, Tolver A, et al. Left atrial volume and function in dogs with naturally occurring myxomatous mitral valve disease. J Vet Cardiol 2017;19(1):24-34.*
- *Morgan KRS, Monteith G, Raheb S, et al. Echocardiographic parameters for the assessment of congestive heart failure in dogs with myxomatous mitral valve disease and moderate to severe mitral regurgitation. Vet J 2020;263:105518.*
- *Boysen S, Gommeren K and Chalhoub S, editors. The essentials of veterinary point of care ultrasound: pleural space and lung. Palm Beach Gardens, FL, USA, Edra Publishing 2022.*
- *Lichtenstein DA, Mezière GA, Lagoueyte J-F, et al. A-lines and B-lines: lung ultrasound as a bedside tool for predicting pulmonary artery occlusion pressure in the critically ill. Chest 2009;136(4):1014-1020.*
- *Pressman GS, Rupesh R and Olex S. A simple technique for bedside estimation of left atrial pressure. Echocardiography 2019;36(11):1972-1978.*
- *Zheng H, Jin Y, Fu Y, et al. Experimental study: using the continuous wave Doppler spectrum technique to detect left atrial pressure. Ann Transl Med 2022;10(2):105.*

Haemodynamic optimisation: asking the right questions

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Learning objectives

- Apply a rational question-based approach to assessing shock
- Understand how traditional and novel diagnostic modalities contribute to an understanding of shock physiology
- Prescribe rational treatments based on shock assessment

Lecture summary

Shock in the ICU is a complex entity. Severe life-threatening shock prompts rapid resuscitation, often based on limited information. However, once life-threatening shock has been resolved, a process of haemodynamic optimisation is necessary. In this phase of treatment, the potential benefits of any intervention should be carefully weighed against the risks. A vast array of diagnostic tools is available to aid in these decisions, but meaningful interpretation of the data is often challenging. I approach this situation through a sequential set of questions that aid me in interpreting what the various diagnostic results are really telling me.

Does tissue oxygenation appear to be adequate for cellular respiration? This question centres on whether there is truly a haemodynamic problem that is *worth treating*. It can be answered by looking at 'upstream' haemodynamic parameters such as perfusion parameters or blood pressure. However, whilst severe abnormalities in these warrant treatment, mild abnormalities may not. Use of 'downstream' markers of inadequate cellular oxygenation such as lactate concentration, venous oxygenation parameters, and some advanced blood gas calculations may aid in this determination.

What are the causes of inadequate cellular oxygenation? The major treatable causes are reduced cardiac output, excessive vasodilation, anaemia, and hypoxaemia. The latter two are easily excluded, though determining what severity should be treated can sometimes be challenging. Differentiating between low cardiac output and inadequate vascular tone is challenging, and often both are present together. Careful evaluation of perfusion parameters and arterial blood pressure tracings (preferably from an arterial catheter) are helpful. Measurement of cardiac output is informative but challenging. However, non-invasive methods are constantly being refined. Assumptions can also be made from the underlying disease state (e.g., sepsis will usually have some vasodilatory component). Some blood gas parameters can also help.

In cases with reduced cardiac output, is it preload responsive? Traditionally, this question was approached using 'static' indices of volume status such as the central venous pressure. Subsequent research has revealed that these are poor predictors of 'preload responsiveness', which is where an increase in preload leads to an increase in stroke volume/cardiac output. 'Dynamic' indices such as pulse pressure variation through the respiratory cycle are superior predictors but are not practical to measure in all patients. Thus, sometimes a fluid challenge must be employed. This involves administration of a small bolus of fluid to increase preload, and close observation of whether it causes evidence of increased cardiac output. The volume depends on how directly the response can be measured. 2 mL/kg may be sufficient in a patient with very direct monitoring of stroke volume, whereas 10 mL/kg may be necessary if physical examination parameters are relied upon for evidence of a response. Notably, not all preload responsive patients *require* that increase in stroke volume. That is why this is only determined after the first two questions have been answered.

Is intravenous fluid therapy the best way to increase preload? If the clinician reaches the point of deciding they have a patient with evidence of inadequate cellular oxygenation, primarily due to low cardiac output that is preload responsive, the next decision is whether to administer fluid to increase preload. This carries risks, as fluid overload is both common and detrimental in critically ill patients. Point of care ultrasound can aid in determining risk of fluid overload, mainly through assessment of vena cava compressibility and for evidence of pulmonary oedema. In a patient with a normal to high absolute blood volume, preload may also be increased with catecholamine administration. This can cause venoconstriction and an increase in the stressed venous volume.

References

- *Berkenstadt, H., Friedman, Z., Preisman, S., Keidan, I., Livingstone, D. and Perel, A., 2005. Pulse pressure and stroke volume variations during severe haemorrhage in ventilated dogs. British Journal of Anaesthesia, 94(6), pp.721-726.*
- *Boyd C, Smart L. Hypovolemic Shock. In: Drobatz KJ, Hopper K, Rozanski E, Silverstein DC, editors. Textbook of small animal emergency medicine. Hoboken: John Wiley and Sons; 2019, pp. 986-992.*
- *Boysen, S.R. and Gommeren, K., 2021. Assessment of volume status and fluid responsiveness in small animals. Frontiers in Veterinary Science, 8, p.630643.*
- *Hoste, E.A., Maitland, K., Brudney, C.S., Mehta, R., Vincent, J.L., Yates, D., Kellum, J.A., Mythen, M.G. and Shaw, A.D., 2014. Four phases of intravenous fluid therapy: a conceptual model. British journal of anaesthesia, 113(5), pp.740-747.*
- *Lamia, B., Monnet, X. and Teboul, J.L., 2006. Meaning of arterio-venous PCO2 difference in circulatory shock. Minerva anestesiologica, 72(6), pp.597-604.*
- *Malbrain, M.L., Marik, P.E., Witters, I., Cordemans, C., Kirkpatrick, A.W., Roberts, D.J. and Van Regenmortel, N., 2014. Fluid overload, de-resuscitation, and outcomes in critically ill or injured patients: a systematic review with suggestions for clinical practice. Anaesthesiology intensive therapy, 46(5), pp.361-380.*
- *Michard, F., 2005. Changes in arterial pressure during mechanical ventilation. The Journal of the American Society of Anesthesiologists, 103(2), pp.419-428.*
- *Rosenstein, P.G., Tennent-Brown, B.S. and Hughes, D., 2018. Clinical use of plasma lactate concentration. Part 1: Physiology, pathophysiology, and measurement. Journal of Veterinary Emergency and Critical Care, 28(2), pp.85-105.*
- *Thomovsky, E., Brooks, A. and Johnson, P., 2016. Fluid overload in small animal patients. Topics in Companion Animal Medicine, 31(3), pp.94-99.*
- *Vincent, J.L. and Weil, M.H., 2006. Fluid challenge revisited. Critical care medicine, 34(5), pp.1333-1337.*
- *Vistisen, S.T. and Juhl-Olsen, P., 2017. Where are we heading with fluid responsiveness research?. Current opinion in critical care, 23(4), pp.318-325.*
- *Walley, K.R., 2011. Use of central venous oxygen saturation to guide therapy. American journal of respiratory and critical care medicine, 184(5), pp.514-520.*
- *Walton, R.A. and Hansen, B.D., 2018. Venous oxygen saturation in critical illness. Journal of Veterinary Emergency and Critical Care, 28(5), pp.387-397.*
- *Zengin, S., Al, B., Genc, S., Yildirim, C., Ercan, S., Dogan, M. and Altunbas, G., 2013. Role of inferior vena cava and right ventricular diameter in assessment of volume status: a comparative study: ultrasound and hypovolemia. The American journal of emergency medicine, 31(5), pp.763-767.*

Fluid therapy in 2023: where are we and where do we go from here?

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Learning objectives

- Discuss current evidence on the risks and advantages of different fluid therapy products
- Understand the remaining gaps in knowledge and future research directions

Lecture summary

Intravenous fluid therapy is one of the most common treatments provided to critically ill small animals. Despite this, there is still a lot we do not know about how to provide optimal fluid therapy that maximises the benefits and minimizes the risks. This uncertainty even leads to controversy and heated debate about some topics. Three of the current topical issues in small animal fluid therapy are colloid vs crystalloid fluids, high chloride fluids, and the appropriate volume of fluid to administer.

Synthetic colloid fluids are now infrequently used in human medicine. However, it is a misconception to state that colloid fluids *overall* are rarely used in human beings, as the natural colloid albumin is now administered to most patients deemed to require colloidal support. As there is limited availability of species-specific albumin solutions in veterinary medicine, most veterinarians must choose between either crystalloid or synthetic colloid fluids. The risks and benefits of administering a synthetic colloid rather than a crystalloid in small animals are unclear. The main synthetic colloids available to veterinarians in most countries are hydroxyethyl starch (HES) or gelatin. There is now a large body of evidence showing that HES can impair coagulation, though the clinical relevance of this is less certain. It is probably more relevant in patients with haemorrhagic shock than other forms of shock. The smaller body of evidence for gelatin suggests it also can impair coagulation in dogs. The main reason for the decrease in synthetic colloid use in human medicine is due to an association with acute kidney injury (AKI), especially with HES. Whether synthetic colloid fluids are associated with clinically relevant increases in AKI risk in veterinary medicine is unclear. A small number of experimental and retrospective studies and a single clinical trial with HES have shown mixed findings. A single study showed gelatin causes increases in urine AKI biomarkers in dogs, with unknown clinical relevance. The benefits are also unclear, as it is controversial as to whether the colloid osmotic pressure increase and volume-sparing effects of colloid fluids are relevant in naturally occurring critical illness. This controversy relates to the role of the endothelial glycocalyx in critical illness and fluid therapy, which is incompletely understood. I use synthetic colloid fluids sparingly and choose HES over gelatin in the small number of cases where I do administer them.

Fluids high in chloride have also been associated with an increased risk of AKI in human medicine, likely due to inappropriate activation of tubuloglomerular feedback. High-chloride crystalloids such as 0.9% NaCl are not used as frequently in small animals as they are in human medicine, but they are preferred by some veterinarians. It is unknown whether they contribute to AKI in small animals. However, it has been demonstrated in some studies that hyperchloraemia is a negative prognostic indicator in dogs and cats, so there is a possible link. Further research is required into the effects of other sources of chloride such as hypertonic crystalloids and chloride-containing electrolyte supplements (such as potassium chloride).
The volume of fluid to administer is also highly controversial. It has long been recognised that hypovolaemia and dehydration are detrimental. However, there is now growing appreciation of the severity of the adverse impact of hypervolaemia and fluid overload. These include delayed wound healing, reduced gastrointestinal function, and pulmonary oedema. The kidney is again a focus, as fluid overload-induced renal oedema can substantially contribute to AKI due to the kidney's rigid capsule. Oedema leads to an increase in intraparenchymal pressure and subsequent decreases in glomerular filtration rate and renal blood flow. Critically ill patients are complex, and it is difficult to know when fluid administration will cause overall benefit and when it will cause overall harm. The human Acute Dialysis Quality Initiative (ADQI) group developed a conceptual model for fluid administration called ROSE, an acronym for Rescue, Optimisation, Stabilisation, and de-Escalation. This model may also be applicable to veterinary medicine, though adaptation and further research are required.

References

- *Boyd, C.J., Brainard, B.M. and Smart, L., 2021. Intravenous fluid administration and the coagulation system. Frontiers in Veterinary Science, 8, p.662504.*
- *Boyd, C.J., Claus, M.A., Raisis, A.L., Cianciolo, R., Bosio, E., Hosgood, G., Nabity, M., Mori, T., Barden, A., Sharp, C.R. and Smart, L., 2019. Evaluation of biomarkers of kidney injury following 4% succinylated gelatin and 6% hydroxyethyl starch 130/0.4 administration in a canine hemorrhagic shock model. Journal of Veterinary Emergency and Critical Care, 29(2), pp.132-142.*
- *Boyd, C.J., Sharp, C.R., Claus, M.A., Raisis, A.L., Hosgood, G. and Smart, L., 2021. Prospective randomized controlled blinded clinical trial evaluating biomarkers of acute kidney injury following 6% hydroxyethyl starch 130/0.4 or Hartmann's solution in dogs. Journal of Veterinary Emergency and Critical Care, 31(3), pp.306-314.*
- *Goggs, R., De Rosa, S. and Fletcher, D.J., 2017. Electrolyte disturbances are associated with Non-Survival in Dogs—a multivariable analysis. Frontiers in Veterinary Science, 4, p.135.*
- *Goggs, R., De Rosa, S. and Fletcher, D.J., 2018. Multivariable analysis of the association between electrolyte disturbances and mortality in cats. Journal of feline medicine and surgery, 20(12), pp.1072-1081.*
- *Hayes, G., Benedicenti, L. and Mathews, K., 2016. Retrospective cohort study on the incidence of acute kidney injury and death following hydroxyethyl starch (HES 10% 250/0.5/5: 1) administration in dogs (2007–2010). Journal of Veterinary Emergency and Critical Care, 26(1), pp.35-40.*
- *Hoste, E.A., Maitland, K., Brudney, C.S., Mehta, R., Vincent, J.L., Yates, D., Kellum, J.A., Mythen, M.G. and Shaw, A.D., 2014. Four phases of intravenous fluid therapy: a conceptual model. British journal of anaesthesia, 113(5), pp.740-747.*
- *Myburgh, J.A., Finfer, S., Bellomo, R., Billot, L., Cass, A., Gattas, D., Glass, P., Lipman, J., Liu, B., McArthur, C. and McGuinness, S., 2012. Hydroxyethyl starch or saline for fluid resuscitation in intensive care. New England Journal of Medicine, 367(20), pp.1901-1911.*
- *Rein, J.L. and Coca, S.G., 2019. "I don't get no respect": the role of chloride in acute kidney injury. American Journal of Physiology-Renal Physiology, 316(3), pp.F587-F605.*

- *Self, W.H., Semler, M.W., Wanderer, J.P., Wang, L., Byrne, D.W., Collins, S.P., Slovis, C.M., Lindsell, C.J., Ehrenfeld, J.M., Siew, E.D. and Shaw, A.D., 2018. Balanced crystalloids versus saline in noncritically ill adults. New England Journal of Medicine, 378(9), pp.819-828.*
- *Semler, M.W., Self, W.H., Wanderer, J.P., Ehrenfeld, J.M., Wang, L., Byrne, D.W., Stollings, J.L., Kumar, A.B., Hughes, C.G., Hernandez, A. and Guillamondegui, O.D., 2018. Balanced crystalloids versus saline in critically ill adults. New England Journal of Medicine, 378(9), pp.829-839.*
- *Sigrist, N.E., Kälin, N. and Dreyfus, A., 2017. Changes in serum creatinine concentration and acute kidney injury (AKI) grade in dogs treated with hydroxyethyl starch 130/0.4 from 2013 to 2015. Journal of veterinary internal medicine, 31(2), pp.434-441.*
- *Sigrist, N.E., Kälin, N. and Dreyfus, A., 2017. Effects of hydroxyethyl starch 130/0.4 on serum creatinine concentration and development of acute kidney injury in nonazotemic cats. Journal of veterinary internal medicine, 31(6), pp.1749-1756.*
- *Thomovsky, E., Brooks, A. and Johnson, P., 2016. Fluid overload in small animal patients. Topics in Companion Animal Medicine, 31(3), pp.94-99.*
- *Yozova, I.D., Howard, J., Sigrist, N.E. and Adamik, K.N., 2017. Current trends in volume replacement therapy and the use of synthetic colloids in small animals—an internet-based survey (2016). Frontiers in veterinary science, 4, p.140.*
- *Yozova, I.D., Howard, J. and Adamik, K.N., 2016. Retrospective evaluation of the effects of administration of tetrastarch (hydroxyethyl starch 130/0.4) on plasma creatinine concentration in dogs (2010–2013): 201 dogs. Journal of veterinary emergency and critical care, 26(4), pp.568-577.*
- *Yozova, I.D., Howard, J. and Adamik, K.N., 2017. Effect of tetrastarch (hydroxyethyl starch 130/0.4) on plasma creatinine concentration in cats: a retrospective analysis (2010–2015). Journal of feline medicine and surgery, 19(10), pp.1073-1079.*

Feline transfusion medicine: from conscious collection to xenotransfusions

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Learning objectives

- Compatibility testing: Blood typing and crossmatching.
- Blood donation: Donor selection and blood collection procedures
- Feline donors and sedation
- Xenotransfusion and alternative transfusion options

Lecture summary

This lecture will cover the fundamentals of feline transfusion medicine, explore developments in blood collection methods, donor welfare considerations and blood product availability. Blood transfusions have increasingly become an integral part of veterinary medicine. They can be a life saving measure, providing an improved quality of life for patients with a large range of clinical conditions. Although generally safe, there are several risks associated with their provision when they are used improperly and without clear understanding. Sourcing blood products can sometimes be challenging, which is especially true of feline blood products due to several reasons, including: limited access to commercially available feline blood products, donor compliance and compatibility restrictions. Nurses generally take on the responsibility of blood component administration and increasingly are also responsible for performing the blood collection; this responsibility requires nurses to have a good understanding of the fundamentals of transfusion medicine, blood collection, donor selection and welfare.

Compatibility testing: Blood typing and crossmatching: Cats can possess naturally occurring alloantibodies to red cell antigens that can cause even a transfusion naive cat to have a potentially severe haemolytic transfusion reaction when delivered type-mismatched blood. Donor and recipient cats must be blood typed before transfusion so that type compatible blood can always be administered. Owing to the frequency of naturally occurring alloantibodies, crossmatching as an additional compatibility testing measure is optimal where possible before every red cell transfusion in cats. This lecture will cover the recognised feline blood groups and the principles of blood typing whilst reviewing the current attitude towards crossmatching.

Blood donation: Donor selection and blood collection procedures: Blood collection methods are categorised as 'open' or 'closed'. A closed collection is one where the contents of the donation bag are only exposed to the air immediately prior to venepuncture as the needle is uncapped. An open collection is one in which the donated blood has had more potential for bacterial contamination due to multiple exposures to the environment. There have recently become more commercially available closed blood collection systems for feline blood donation that we will explore, but often in general practice the donation is needed immediately, and an open collection system is adopted. A donor's temperament should be suitable such that they do not become stressed or unhappy during their visit, carefully selecting donors based on their temperament and a clear understanding of the required donor criteria ensures their welfare is upheld above all else. We will review suggested donor criteria and additional behavioural considerations that can help identify the 'perfect' donor.

Feline donors and sedation: How do we maximise the chances of a successful donation whilst ensuring the donor's welfare is of paramount importance? Historically chemical restraint has always been considered a necessity for feline blood donors, but it is possible to perform blood donation in conscious cats with careful donor selection, creation of a feline friendly environment and an approach that aims to minimise any disruption, pain or anxiety in the feline donor. Sedation is however still necessary and suitable for certain situations and every donor should be evaluated on an individual basis. Where the donation has the potential to create a negative experience, an appropriate sedation for that donor should be chosen.

Xenotransfusion and alternative transfusion options: When compatible feline blood is not available or in a life-threatening time sensitive situation there are alternative transfusion options. A xenotransfusion of canine blood to cats is widely recognized and utilized in veterinary medicine. Its application in an emergency is supported by the relative ease of accessing canine blood products and the absence of severe adverse effects. It is imperative to recognize however that delayed haemolytic reactions are common and so the benefit is short lived, and they must only ever be performed once in a cat's lifetime, as subsequent xenotransfusion has proven to be fatal. Autologous transfusion can also be a safe and effective technique for patients presenting with intracavitary haemorrhage or surgical blood loss. Both provide an adequate provision of blood but have patient dependent considerations that might affect their use.

References

- *Humm, K. & Chan, D., Prospective evaluation of the utility of cross-matching prior to first transfusion in cats: 101 cases, Journal of small animal practice, 2020, 61;285-291*
- *Le Gal, A., Thomas, E. K. & Humm, K. R., Xenotransfusion of canine blood to cats: a review of 49 cases and their outcome, Journal of Small Animal Practice, 2020, 61; 156-157*
- *Russo, C. & Humm, K., Feline donor selection, In: Manual of Veterinary Transfusion Medicine and Blood Banking. Eds K. Yagi and M. K. Holowaychuk, Ames, USA,: John Wiley & Sons, Ltd; 2016, 212-222*
- *Taylor, R. & Humm, K., Feline blood collection, In: Manual of Veterinary Transfusion Medicine and Blood Banking. Eds K. Yagi and M. K. Holowaychuk, Ames, USA,: John Wiley & Sons, Ltd; 2016, 223-236*
- *Taylor, S., Spada, E., Callan, M., et al. 2021 ISFM Consensus Guidelines on the Collection and Administration of Blood and Blood Products in Cats, Journal of Feline Medicine and Surgery, 2021, 23; 419-424*

Death is coming; disseminated intravascular coagulation

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Learning objectives

- Understanding of the pathophysiology of DIC
- Overview of thrombin generation
- Review of the coagulation pathway and fibrinolysis
- Application of diagnosis and treatment

Lecture summary

Disseminated intravascular coagulation (DIC) is a haemostatic disorder which can be described as an excessive fibrin deposition leading to the occlusion of blood vessels and organ damage that is associated with consumption of coagulation factors and platelets. DIC is the excessive activation of haemostasis, with subsequent generation of excess thrombin and formation of microvascular thrombi. Haemorrhage will occur as coagulation factors and platelets are consumed, which then will act as a viscous cycle of haemostasis activation, beginning with a trigger to activate coagulation and thrombin in a 'compensated' state, until consumption and inhibition of these stages occurs, moving the patient from a hypercoagulable state (at risk of thrombosis) to a 'decompensated' hypocoagulable state (at risk of uncontrolled haemorrhage). As this loss of control over haemostasis occurs, the endothelium becomes dysfunctional or injured and inflammation ensues, with this comes the release of damage associated molecular patterns (DAMPs), including extracellular or cell-free DNA, histones, and nucleosomes (DNA-histone complexes), neutrophil proteases and other molecules and inflammatory nuclear-based cytokines. These DAMPs can activate coagulation, injure other tissues, and upregulate tissue factor. Thrombin generation occurs as a product of haemostasis, will exert inflammatory mediation. Haemostasis is regulated by 3 antithrombotic systems, including antithrombin, which holds anticoagulant and anti-inflammatory effects which work with heparin to become more active. During this stage, microvascular thrombi are forming, and platelets and coagulation factors are being consumed in excessive clot formation, and proceeds unchecked; at which point the DIC becomes "uncontrolled."

Coagulation Cascade: Coagulation consists of three pathways- the extrinsic, intrinsic, and common pathways that all work together to form a stable blood clot. Both the extrinsic and intrinsic pathway lead to a final pathway that activates factor X, with the extrinsic pathway initiated by factor III (tissue factor) and Factor VII, in contrast to the intrinsic pathway which is activated by factors XII, XI, IX and VIII. The latter pathway requires factor VIII, found in the blood, and activated by thrombin (therefore an important player when we consider DIC), and calcium to help form the factor X. The extrinsic pathway is triggered by injury to the endothelial tissue, exposing factor III (tissue factor), which also requires calcium, along with vitamin K to bind with factor VII, to bind with to activate factor X. These lead to the common pathway which uses factors X, V, II, I and XIII, and results as both pathways activate factor X. Again, this pathway requires calcium to form prothrombinase, further into prothrombin and thrombin. The thrombin will then cleave fibrinogen (factor I) and factor XIII (a stabilising factor) into factors Ia and XIIIa, respectively. This then binds with calcium to help stabilise the clot. As demonstrated, the thrombin has many functions including activating platelets and as a key player in the coagulation cascade, as is greatly affected in production in the disease process of DIC.

Diagnostics and Treatment: The clinical symptoms of DIC can often be nonspecific and can vary with the degree of coagulation and the organ systems affected. Often a primary clinical sign will be profuse spontaneous bleeding (either primary or secondary), or with signs of micro thromboses. However, clinical signs can be highly variable depending on the underlying primary disease process, therefore laboratory diagnostics including prothrombin time (PT), activated partial thromboplastin time (aPTT), D-dimers, platelet counts and blood smear evaluation along with the usual diagnostics, will highlight any abnormalities. Once DIC is suspected/confirmed, the primary underlying disease should be addressed to eliminate the cause of DIC, as well as implementing treatment for any variable signs of secondary complications such as hyperthermia and hypoxaemia. The aims of treatment for DIC include stopping intravascular coagulation and haemorrhage, maintaining good parenchymal organ perfusion, and preventing secondary complications. Replacement therapy, using fresh frozen plasma or frozen plasma and packed red blood cells, to replace like for like fluid resuscitation and lost clotting factors, is a mainstay of treatment. Heparin administration is usually used in a dual approach with blood component therapy to halt intravascular coagulation.

References

- *Cornell University (2019) DIC and thrombosis, Cornell University College of Veterinary Medicine. Available at: https://www.vet.cornell.edu/animal-health-diagnostic-center/laboratories/comparative-coagulation/clinical-topics/dic-and-thrombosis*
- *Guillaumin, J. (2019) Disseminated intravascular coagulation, Clinician's Brief. Available at: https://www.cliniciansbrief.com/article/disseminated-intravascular-coagulation*
- *Estrin, M.A. et al (2006) Disseminated intravascular coagulation in cats, Journal of veterinary internal medicine, 20 [6], pp. 1334-1339.*
- *Levi, M. and Schultz, M.J. (2016) Disseminated intravascular coagulation in the critically ill, Oxford Medicine Online [Preprint].*
- *Mischke, R. (2010) Disseminated intravascular coagulation in dogs: Are scoring systems of value?, The Veterinary Journal, 185(3), pp. 243–244.*

Running a patient-friendly ICU

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Learning objectives

- Understand sources of anxiety in the ICU
- Understand the effect of hospitalisation on sleep patterns
- Implement management strategies to reduce anxiety and promote sleep

Lecture summary

The ICU is a confronting and stressful environment for small animals. Two major effects of this abnormal environment are anxiety and impairment of normal sleep patterns. Prevention or amelioration of these problems has multiple potential benefits, including: Improved patient welfare, Improved recovery from illness, Greater ability to examine the patient and interpret physiologic changes. Whilst they are discussed separately below, anxiety and sleep interact. Ensuring adequate sleep can lower anxiety and treating anxiety can allow for better sleep.

Anxiety is an adverse emotional response to stimuli perceived as associated with potential danger. In nature, anxiety can be a beneficial trait when it allows an animal to avoid potential threats. However, in the ICU environment, there are many stimuli that may be interpreted as threatening and therefore cause anxiety. I use the phrase 'hospital-associated anxiety' to describe this situation. I prefer this term to 'hospital-induced anxiety', as some animals may have an underlying chronic anxiety condition that is exacerbated by hospitalisation. Research on the prevalence of hospital-associated anxiety in the veterinary ICU is lacking, but anecdotally it seems to be very common. Research in the human ICU has also found anxiety is common in that environment. Anxiety can activate many adverse neurohormonal responses, such as the sympathetic nervous system and cortisol release, that can interfere with recovery. Additionally, they can cause alteration of vital signs that complicates monitoring of the patient's illness. Recognition of anxiety should include thorough assessment of both behavioural and physiological indicators. Validated scoring systems can assist, though these are in the early stages of development for hospitalised small animals. Management must involve a broad range of both non-pharmacologic and pharmacologic strategies. Identification of environmental factors that are contributing allows for their modification, for example choosing a cage where a dog cannot see other dogs or providing a cat with a hiding box. Low stress handling techniques should also be employed. Pheromones and constant physical pressure jackets are beneficial to some individuals but not others. Major anxiolytic medications include trazodone, a_2 agonists such as medetomidine, benzodiazepines, and gabapentin. Opioids and phenothiazines such as acepromazine are not truly anxiolytic and should not be employed as the sole pharmacologic therapy for anxiety. However, the additional tranquilisation/sedation they provide is sometimes a beneficial component of a multimodal treatment plan. Despite the large amount of time we spend doing it, sleep is still poorly understood. Sleep is initiated by either the circadian rhythm via hormonal signals such as melatonin, or through a homeostatic drive for sleep. Both the sympathetic nervous system and cortisol inhibit sleep. Sleep deprivation, in terms of both quantity and quality, is often a problem in human ICU patients. Pilot research at our hospital suggests that dogs in the ICU are also often awake during normal sleeping hours. This important component of patient welfare often goes overlooked. Bright lighting can disrupt the normal circadian rhythm, and consideration should be given to night 'lights-off' time where possible. The ICU is also a loud place, with research showing veterinary ICUs have sound levels well above World Health Organisation recommendations for the human ICU. Monitoring the sound level with a highly visible sound meter can encourage staff to make efforts to limit excess noise. Particular individual patients may also benefit from interventions such as ear plugs and eye masks, though appropriate patient selection is critical. Choice of pharmacologic measures must be carefully considered. Whilst some drugs such as benzodiazepines may sedate the animal, they may impair good quality sleep. a_2 agonists such as medetomidine work through similar pathways to natural sleep, so they may be better choices for this purpose. Melatonin is often administered as a sleep aid to human patients, though investigation of its efficacy in small animals is lacking. Further research is urgently required into this overlooked aspect of patient welfare.

References

- *Damon M, Rozanski E, Spagnoletti C, Sharp C. Use of the Thundershirt to control canine anxiety in the ICU (abstract). Journal of Veterinary Emergency and Critical Care. 2014;24(S1):S5-S6.*
- *Gilbert-Gregory SE, Stull JW, Rice MR, Herron ME. Effects of trazodone on behavioral signs of stress in hospitalized dogs. Journal of the American Veterinary Medical Association. 2016;249(11):1281-1291.*
- *Hammerle M, Horst C, Levine E, et al. 2015 AAHA canine and feline behavior management guidelines. Journal of The American Animal Hospital Association. 2015;51(4):205-221.*
- *Hu RF, Jiang XY, Hegadoren KM, Zhang YH. Effects of earplugs and eye masks combined with relaxing music on sleep, melatonin and cortisol levels in ICU patients: a randomized controlled trial. Critical Care. 2015;19:115.*
- *Huang H-W, Zheng B-L, Jiang L, et al. Effect of oral melatonin and wearing earplugs and eye masks on nocturnal sleep in healthy subjects in a simulated intensive care unit environment: which might be a more promising strategy for ICU sleep deprivation? Critical Care. 2015;19(1):124.*
- *Lefman SH, Prittie JE. Psychogenic stress in hospitalized veterinary patients: Causation, implications, and therapies. Journal of Veterinary Emergency and Critical Care. 2019;29(2):107-120.*
- *Mahmoud M, Mason KP. Dexmedetomidine: review, update, and future considerations of paediatric perioperative and periprocedural applications and limitations. British Journal of Anaesthesia. 2015;115(2):171-182.*
- *McKinley S, Stein-Parbury J, Chehelnabi A, Lovas J. Assessment of anxiety in intensive care patients by using the Faces Anxiety Scale. American Journal of Critical Care. 2004;13(2):146-152.*
- *Pisani MA, Friese RS, Gehlbach BK, Schwab RJ, Weinhouse GL, Jones SF. Sleep in the intensive care unit. American Journal of Respiratory and Critical Care Medicine. 2015;191(7):731-738.*
- *Sinclair MD. A review of the physiological effects of alpha2-agonists related to the clinical use of medetomidine in small animal practice. The Canadian Veterinary Journal. 2003;44(11):885-897.*
- *Tembo AC, Parker V. Factors that impact on sleep in intensive care patients. Intensive and Critical Care Nursing. 2009;25(6):314-322.*
- *Tembo AC, Parker V, Higgins I. The experience of sleep deprivation in intensive care patients: findings from a larger hermeneutic phenomenological study. Intensive and Critical Care Nursing. 2013;29(6):310-316.*
- *van Haaften KA, Forsythe LRE, Stelow EA, Bain MJ. Effects of a single preappointment dose of gabapentin on signs of stress in cats during transportation and veterinary examination. Journal of the American Veterinary Medical Association. 2017;251(10):1175-1181.*
- *Vinke C, Godijn L, Van der Leij W. Will a hiding box provide stress reduction for shelter cats? Applied Animal Behaviour Science. 2014;160:86-93.*
- *Xie H, Kang J, Mills GH. Clinical review: The impact of noise on patients' sleep and the effectiveness of noise reduction strategies in intensive care units. Critical Care. 2009;13(2):208.*

Civility saves lives: why behaviour matters and how to thrive as a team

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Learning objectives

- To explore the core concepts of incivility
- To understand the impact of incivility
- To discover the facts and science associated with incivility
- To understand what we can do when faced with incivility.

Lecture summary

Incivility is defined as rude, condescending behaviour, and ostracizing acts that violate workplace norms of respect (Cortina et al, 2017). Incivility can be either blatant (for example blaming, public humiliation or aggressive body language) or insidious (for example with-holding information for personal gain, inappropriate humour or inconsistent expectations). When considering how to approach incivility it is vital that we understand two core concepts: Firstly, that incivility is defined by the interpretation of the recipient, regardless of the intent and secondly that if we perceive someone as being uncivil towards us we must consider whether we may have misunderstood them.

To understand the impact that incivility can have on our patients we must first understand what happens to us when we perceive someone as being uncivil to us. Incivility is considered a threat and when humans perceive a threat (whether actual or supposed), the fight, flight or freeze (FFF) response is triggered, and our attention is focused on escaping or reducing that threat. Research suggests that when this response occurs we suffer a reduction in cognitive bandwidth and our ability to use our working memory to engage in problem-solving behaviour is decreased by 61%. Thus, incivility impairs task performance and engagement. Research also suggests that when we witness incivility it causes a 20% decrease in performance and a 50% reduction in willingness to help others, and clients who witnessed incivility were found to be 75% less enthusiastic for the organisation (Porath & Pearson, 2013).

When surveying doctors and nurses in human healthcare, Rosenstein and O'Daniel (2008) found that 75% identified bad behaviours within their teams which lead to medical errors and that 25% of those surveyed were convinced that these behaviours contributed to the deaths of their own patients. It is therefore imperative that we understand that incivility reduced our cognitive bandwidth which affects our ability to deliver our skills and knowledge, which may cause us to make errors and may ultimately may cause our patients to have poor outcomes. Research on incivility in veterinary practice has recently identified that higher levels of client rudeness are currently experienced than coworker rudeness, that veterinary nurses experience more rudeness from senior staff and co-workers and that this is linked to job satisfaction, job intention turnover and burnout (Irwin, Silver-MacMahon & Wilke, 2022).

Whilst research has shown that it is not possible to mitigate for incivility, through understanding the common coping mechanisms and conversational structures that can be used to successfully reduce incivility we can ensure that all members of the veterinary have the information and tools to ensure that they can manage incivility.

In this session we will explore how calling it out with compassion and non-violent communication can be used alongside self-awareness, empathy and compassion to manage incivility as individuals and teams, with both clients and co-workers. We will understand the importance of creating psychological safety and we will consider how workplaces can adopt social contracts and reduce external stressors (including workload, hunger and fatigue) to improve workplace culture and prevent the evolution of incivility.

References

- *Cortina, L. M., Kabat-Farr, D., Magley, V. J., & Nelson, K. (2017). Researching rudeness: The past, present, and future of the science of incivility. Journal of Occupational Health Psychology, 22(3), 299–313. https://doi.org/10.1037/ocp0000089*
- *Porath, C. & Pearson, C. (2013) The price of incivility. Harv Bus Rev. 2013 Jan-Feb;91(1-2):114-21, 146*
- *Rosenstein, A. H., & O'Daniel, M. (2008). A survey of the impact of disruptive behaviors and communication defects on patient safety. Joint Commission journal on quality and patient safety, 34(8), 464–471. https://doi.org/10.1016/s1553-7250(08)34058-6*
- *Irwin, A., Silver-MacMahon, H., & Wilcke, S. (2022). Consequences and coping: Investigating client, co-worker and senior colleague incivility within veterinary practice. Veterinary Record, 191(7), [e2030]. https://doi.org/10.1002/vetr.2030*

I only shout at the TV when they shock asystole! Getting to grips with how and when to use your defibrillator

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Learning objectives

- Compare and classify ECGs seen during cardiopulmonary arrest (CPA)
- Identify what fibrillation is
- Understand what defibrillation is and when it is indicated
- Demonstrate how to perform defibrillation
- Explain safe use of the defibrillator

Lecture summary

Interpretation of ECGs are vital during CPR. Decision making on next steps depends on accurate rhythm diagnosis and the determination of a shockable or non-shockable rhythm. There are four ECG rhythms seen during CPA. Two are non-shockable rhythms that should be treated with drugs and two are shockable rhythms requiring defibrillation, with gold standard technique being the use of a defibrillator, if you have one available. In order to tell which is which we need to work through a series of questions that can help us determine the rhythm. Are there consistent repeating complexes? If so, are pulses associated with these complexes? If not, is the rate more than 200bpm? If there are no consistent repeating complexes, is the ECG a flat line? Answering these questions will help us determine if there is a perfusing rhythm, and if not, which of the four CPA rhythms is present.

Reviewing the electrical pathways of the heart will help us identify and understand what fibrillation is. During normal sinus rhythm, the myocytes (heart cells) depolarise in turn. This is controlled by the sinoatrial node which stimulates the atria to contract, followed by the atrioventricular node which stimulates the ventricles to contract. Fibrillation is a cardiac arrhythmia characterised by rapid, random excitation of the myocytes with no coordinated contraction of the heart. It can be further described as atrial or ventricular, depending on where the fibrillation is occurring, with each having very different causes and outcomes. Atrial fibrillation results in reduced filling of the heart and although cardiac output is reduced, the patient can still cope. Ventricular fibrillation is much more dangerous and imminently life-threatening, leading to cardiac arrest if not treated immediately. The ventricles quiver fast and erratically meaning they are not able to contract, dropping cardiac output to zero. Ventricular fibrillation usually causes a loss of consciousness and absence of pulses. If recognised, CPR should be initiated immediately while the defibrillator is set up.

Defibrillation does what it says on the tin, that is, it stops fibrillation. There are 3 methods of defibrillation available: mechanical – using the precordial thump, chemical – using drugs such as lidocaine and amiodarone in certain situations, or electrical – using an electrical defibrillator. This is the most successful method and works by passing an electrical current through the myocardium in an attempt to depolarise the myocytes all at the same time. By doing this, it stops the cells firing at random, where they can't come together to produce a ventricular contraction. Defibrillation is only useful for treatment of shockable rhythms during CPR.

How to perform defibrillation

Clip fur either side of thorax (if time)
Set energy required on the defibrillator depending on patient weight (2-4J/KG)
Apply electrode gel to both defibrillator paddles
Press charge
Place the patient in dorsal recumbency, and stabilise them with paddles on either side of the chest wall at the level of the costochondral junction
Hold paddles as firmly as possible against the chest wall, directly over the area of the heart
Shout CLEAR and ensure everyone is clear from the table including yourself!
Administer a shock by pressing the red buttons on the paddles and compressing the thorax between the paddles while discharging the shock

Safe use of the defibrillator includes

Ensure no alcohol on the patient - clean off with water if necessary
Remove any equipment from the table
Remove any metal objects from the patient (collar) or table
Turn off any oxygen supply

References

• Fletcher D.J et al., RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 7: Clinical guidelines, Journal of Veterinary Emergency and Critical Care 22(S1) 2012, pp S102–S131 doi: 10.1111/j.1476-4431.2012.00757.x

Urinary catheters made easy

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Learning objectives

- Attendees will learn about how to place female urinary catheters
- Attendees will learn about how to place male urinary catheters
- Attendees will learn about urinary catheter maintenance
- Attendees will learn about preventing complications in urinary lines.

Lecture summary

Female Dog Urinary Catheter Blind Placement: Some individuals elect to use lidocaine jelly in the vulva at a maximum dose of 2mg/kg. This will help with analgesia as this procedure is very uncomfortable. Most female dogs needs to be sedated or under anesthesia as they will not tolerate it awake.
Most commonly the dog is placed in sternal or lateral recumbency, but this author knows veterinary personal that have placed catheters in D/V position as well. If utilizing sternal recumbency many technicians will place a towel until the abdomen to lift up the pelvis for better access.

Shave and prep the area. While it's impossible to maintain complete sterility the procedure should be as sterile as possible. Due to the difficult nature of the procedure temporary urinary catheters to obtain urine or drain bladders are not usually performed. Most often a longer term foley catheter (with balloon) is placed. Sterile gloves must be worn when handling a foley catheter. Lubricate index finger with sterile lubricant (may need to use pinky finger in very small dogs) and insert into vulva.

HINT: with the patient in lateral recumbency, the author inserts the finger of the hand that matches the recumbency of the patient, for example, in left lateral, the author uses the left hand. This avoids having to contort oneself into a very awkward position. Using your non-dominate hand use your index finger caudally and move dorsally to move up and over the pubic bone (pinkie in a small dog). Typically the papilla is felt just beyond the pubic bone as a small fleshy bump, or a divot. Position the finger on the papilla and direct the urinary catheter into the vagina with your dominate hand.
If you meet resistance you are likely in the vagina. Advance the catheter until the hub reaches the vulva, then remove finger and inflate the balloon of the foley, then gently pull on the catheter until mild resistance is met. DO NOT overinflate or pull too hard. Regardless of skill it may not be possible to place a urinary catheter in some female dogs. It's a hard skill to perform.

Female Dog Urinary Catheter Visual Placement: If you are fortunate your hospital will purchase the nasal speculum by Welch Allyn that fits on top of the otoscope handle. Contact the company to be sure you order the right head. It comes with a light and fits perfectly in to the vulva of dogs that are about 20 lbs [9kg] or larger. If it's a smaller dog you will have to use an otoscope cone (the same for ears).
You will perform the same steps in the non-visual technique, but you will look through the otoscope with the cone or, ideally, use the nasal speculum. With the nasal speculum you can continuously visual the urethra. With the cone you will have to pear through the cone (like you do for ears). If using the otoscope cone it will be left behind and remains on the outer portion of the catheter until catheter removal. While not ideal there is no way to remove it. Patients do not seem bothered by it, but it's not ideal.

Male Cat Urinary Catheter: Many times veterinarians like to place urinary catheters in cats suffering a urinary obstruction so they feel for the obstruction and be on hand in case of a bladder or urethra rupture. The position depends on the preference of the person placing it. In the case of an unblocking most start with a tom-cat open ended catheter. Some will prefer an metal olive tip catheter. Once the obstruction has been passed or pushed back in to the bladder a red rubber is placed. Wear sterile gloves and prep the area sterilely. Most times the person placing is also extruding the penis by pushing down. Locate the urethral opening and insert the catheter. Pulling the penis caudally will straighten the urethra and facilitate placement. There are a variety of ways to suture or secure the red rubber. Ideally a post-radiograph is taken to look for stones and proper placement.

Due to small word count allowed, please contact author for full lecture notes: VetTeamTraining@Gmail.com

The nurses role in the placement and management of enteral feeding tubes

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Learning objectives

- Understand the importance of enteral nutrition in our critically ill patients
- What feeding tube options do we have and their indications, advantages, and disadvantages
- Nurses role in formulating and initiating a feeding plan
- Nursing considerations for patients with feeding tubes

Lecture summary

Introduction: Nutrition is often overlooked in our hospitalised veterinary patients, which depending on what the patient is being treated for, can worsen the situation. Providing sufficient calories is critical for reducing protein catabolism and its consequences i.e. muscular atrophy, immunodeficiency, and delayed healing. There are two routes for providing nutrition, enteral and parenteral. If the patient has a functioning digestive system, then enteral nutrition is the preferred route as maintenance of normal gut flora relies on normal gastrointestinal motility and nourishment of the enterocytes. Enteral feeding is also less expensive, stimulates the immune system, minimises the metabolic consequences of refeeding and decreases hospitalisation stays.
Tube feeding: The placement of a feeding tube should be considered in all patients that have not eaten 50% of their resting energy requirements (RER) for ≥3 days, as well as those who are unable to or should not eat by mouth such as jaw fractures or oesophageal surgery. There are many feeding tube options available to us each with their own indications, advantages, disadvantages and risks as detailed below.

Nasogastric (NG tube):
Short term use, < 7days
Inexpensive and easy to place (can be placed by an RVN)
No anaesthesia required
Allows for aspiration of gastric content
Small lumen so easily blocked
Liquid diets only
Not tolerated by all patients, buster collar required
Radiograph required to confirm placement
Risk of misplacement causing iatrogenic pneumothorax
Contraindicated in coagulopathies, facial trauma, megaoesophagus, dyspnoea, increased ICP, vomiting and unconsciousness/loss of gag reflex
Oesophagostomy (O tube):
Longer term use, ≥5 days requirement
Inexpensive, easy to place
Can use more calorific diets
Requires anaesthesia
Requires functioning oesophagus
Indicated in oral and pharyngeal disease
Stoma site can become infected
Cellulitis can occur if removed too early
Possibility of placement of tube through oesophagus into mediastinum
Patient can be discharged with tube in place
Gastrostomy (G or PEG tube):
≥14 days requirement, can remain in situ for weeks to months
Can feed blended canned diets
Indicated in oral, pharyngeal, or oesophageal disorders or altered level of consciousness
Easy removal after 14 days once fistula developed
Requires surgery or endoscopy to place

Incision through abdominal and gastric wall
Risk of peritonitis or abdominal abscessation
Patient can be discharged with tube in place
Jejunostomy (J tube):
≥7 days requirement
Bypasses mouth, oesophagus, stomach, and duodenum
Indicated in pyloric obstruction, pancreatitis, gastric motility disorders, biliary disease
Liquid, monomeric diet only, fed via a constant rate infusion
Bolus feeding associated with nausea and pain
Requires laparotomy or endoscopy and PEG tube (J thru G tubes) to place
Primarily used for hospital feeding of critical patients
Cannot be removed for a minimum of 10 days
Risk of peritonitis or abdominal abscessation

Formulating and delivering a feeding plan: Even in patients with severe malnutrition, the immediate goals of therapy should focus on resuscitation, stabilisation, and identification of the primary disease process after which the formulation of a nutritional plan should be created to prevent or correct overt nutritional deficiencies and imbalances. Veterinary nurses are crucial in providing this nutritional support. We spend the most contact time with patients and are therefore best placed to identify patients that require nutritional support, assist with the placement of feeding tubes, implement a feeding plan, and monitor the patient for complications. The patient's RER approximates the number of calories required for maintaining homeostasis at rest. Illness factors were advocated in the past, but these have not been shown to counter protein catabolism or improve patient outcome.

A common way to calculate the RER is to use the following formula:
2 - 45kg body weight
RER (kcal/day) = (30 x Bodyweight in kgs) + 70
<2 or >45kg body weight
RER (kcal/day) = 70 x (bodyweight in kgs)$^{0.75}$

Dependent on underlying disease and tube selection, an appropriate diet should be selected. Nutritional support should be introduced gradually and aim to reach target levels in 48-72hrs. These tube feeds can be provided via either bolus feeds or trickle feeding via a constant rate infusion. All patients receiving enteral nutrition should be routinely assessed to include perfusion and hydration parameters and body weight. Changes in mentation or level of consciousness should prompt investigations for glucose levels, electrolyte imbalances, refeeding syndrome or hepatoencephalopathy. Monitoring the gastric residual volume can also be helpful in assessing tolerance to enteral feeding.

Conclusion

Enteral feeding has been proven to positively influence patient outcomes and increase survival rates. Despite the possibility of tube-related complications, the benefits of feeding tubes and provision of nutrition to critically ill patients generally outweigh the potential disadvantages.

References

• *Brunetto MA, Gomes MOS, Andre MR, et al. Effects of nutritional support on hospital outcome in dogs and cats. J Vet Emerg Crit Care (San Antonio). 2010;20(2):224–231.*
• *Chan DL (2015). Estimating energy requirements of small animal patients. In Chan DL, (editors) Nutritional Management of Hospitalized Small Animals. John Wiley & Sons Ltd, Chichester:7–13.*
• *Chan DL (2021). Nutritional support for the critically ill patient. In Battaglia A and Steele A (editors) Small Animal Emergency and Critical for Veterinary Technicians (4th ed), Elsevier, Missouri: 80-91.*
• *Eirmann L (2015). Esophagostomy feeding tubes in dogs and cats. In Chan DL, (editor) Nutritional Management of Hospitalized Small Animals. John Wiley & Sons Ltd, Chichester:29–40.*
• *Eirmann L and Michel K (2009). Enteral nutrition. In Silverstein D and Hopper K (editors), Small Animal Critical Care Manual (2nd ed), Elsevier Saunders, Missouri: 681-686.*
• *Hurley KJ and Michel KE (2011). Nutritional support for the critical patient. In King, L.K. and Boag, A. (editors) BSAVA Manual of Canine and Feline Emergency and Critical Care, (2nd ed), BSAVA: 327-338.*
• *Tonozzi C (2017). Nutritional status. In Kirby R and Linklater A (editors), Monitoring and Intervention for the Critically Ill Small Animal: The Rule of 20 (1st ed), John Wiley & Sons Ltd, Oxford: 285-301.*

Taking the septic patient to theatre (and out of it)

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Learning objectives

- Learn about pre and post-operative strategies in septic animals
- Become familiar with anaesthetic protocols for the septic patient
- Recognise inotropes and vasopressors
- Recognise essential equipment and how to use it to inform your clinical choices

Lecture summary

Dogs with septic peritonitis surgically corrected had a mortality of 57-64% in a study (Bentley et al, 2007). The surviving sepsis campaign was developed to standardise the treatment of sepsis in people (bundle of treatments and timings to improve prognosis). In veterinary medicine we do not have a consensus on a standardised treatment for sepsis, we should aim at:
Identify source of infection
Assess and correct fluid imbalances and blood pressure (early use of vasopressors)
Discuss prognosis/treatment with the owner
Deciding about treatment
The minimum database should include:
Complete blood count
Serum biochemical profile
Blood lactate
Radiographs and A-FAST + T-FAST scan

Preoperatively
Monitor temperature
Passive and active heating measures
Place 2 intravenous cannulas/central line
Place arterial line
Clip for surgery
Fluid resuscitation
Address anemia
Will giving more fluids improve the animal's blood pressure?
We can use static parameters:
Blood pressure, heart rate
Central venous pressure
lactate measurement
Or dynamic parameters:
Echocardiography
Arterial waveform analysis (PPV) (in ventilated animals) (Fantoni et al., 2017)
Plethysmograph variability index, in ventilated animals
Fluid challenge
Body weight and urinary output

Fluid resuscitation
Balanced crystalloids first choice of fluids
Albumin is given in people when large amounts of fluids are required. Beware anaphylaxis risks.
Fresh frozen plasma. Can be used to treat coagulopathies.
Whole blood/packed red blood cells to treat anaemia, thrombocytopaenia

Premedication
Opioids (butorphanol, methadone, fentanyl)
Benzodiazepines (might offer sedative effects if mentation is depressed)
Major sedatives (acepromazine should be avoided, medetomidine can be administered in early stages of an uncompromised animal).

Induction: Preoxygenation is recommended. Induction is rapid and with a head-up position in cases of obstruction of the gastro- intestinal system. Induction agents that can be used include:
Propofol/alfaxalone
Ketamine+benzodiazepines
Fentanyl+benzodiazepines
Co-induction is recommended
Psatha and colleagues (2011) compared alfaxalone alone to fentanyl+diazepam in critical cases, with no differences in blood pressure between the 2 groups.

Maintenance: Due to their vasodilatory properties we must aim to the lowest possible dose of inhalant anaesthetic agents. Intraoperative analgesia continuous rate infusions can help in reducing requirements of inhalant agents:
Fentanyl 1-6mcg/kg/hr IV
Lidocaine 25-200 mcg/kg/ IV NOT in CATS (Bellini & Seymour, 2016)
Ketamine 2.5-10 mcg/kg/min IV
Dexmedetomidine: for the future? Laboratory animals' studies have shown promising results (Aidoni at al, 2020).
Respiratory acidosis complicating a metabolic acidosis must be avoided, hence mechanical ventilation should be ready to be implemented.

Inotropes and vasopressors: Noradrenaline is the vasopressor of choice in people and most of the veterinary surgeons use it as first line too. When compared to dopamine, it possesses beta agonist activity at lower doses too. Dopamine causes frequent arrhythmias in people and it's not recommended. We don't have quite the same incidence of arrhythmias in small animals, it can be considered. At lower doses, dopamine can actually cause renal vasodilation. Dobutamine is the first line treatment to counteract low contractility, but it can cause arrhythmias, especially when used in animals with low pressure. Phenylephrine has predominantly alpha 1 agonist activity and can cause splanchnic vasoconstriction (and low perfusion) but, together with epinephrine and vasopressin, can be used as a rescue treatment once the animal becomes refractory to noradrenaline.

Recovery: Hypothermia and hypoglycemia will delay recovery. Slowly weaning the inotropes/vasopressors helps determining if mean blood pressure remains above 65 mmHg. Aim for 0.5-1ml/kg/hr urine production. Food intake: Plan while under anaesthesia but delay if level of consciousness is low.

References

- *Aidoni, Z., Pourzitaki, C., Stamoula, E et al., 2020. Circulatory effects of dexmedetomidine in early sepsis: a randomised controlled experimental study. Naunyn-Schmiedeberg's Archives of Pharmacology, 393(1), pp.89-97.*
- *Bellini, L. and Seymour, C.J., 2016. Effect of intraoperative constant rate infusion of lidocaine on short-term survival of dogs with septic peritonitis: 75 cases (2007–2011). Journal of the American Veterinary Medical Association, 248(4), pp.422-429.*
- *Bentley, A.M., Otto, C.M. and Shofer, F.S., 2007. Comparison of dogs with septic peritonitis: 1988–1993 versus 1999–2003. Journal of Veterinary Emergency and Critical Care, 17(4), pp.391-398.*
- *Evans L, Rhodes A, et al. Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock 2021. 2021 Crit Care Med. Nov 1;49(11):e1063-e1143. Available at: https://www.sccm.org/Clinical-Resources/Guidelines/Guidelines/Surviving-Sepsis-Guidelines-2021.*
- *Fantoni DT, Ida KK, Gimenes AM, Mantovani MM, et al. 2017 Pulse pressure variation as a guide for volume expansion in dogs undergoing orthopedic surgery. Vet Anaesth Analg. 2017 Jul;44(4):710-718.*
- *Hamzaoui O, Scheeren T, et al. 2017 Norepinephrine in septic shock: when and how much? Curr Opin Crit Care. Aug;23(4):342-347.*
- *Psatha, E., Alibhai, H.I., Jimenez-Lozano, A., et al. 2011. Clinical efficacy and cardiorespiratory effects of alfaxalone, or diazepam/fentanyl for induction of anaesthesia in dogs that are a poor anaesthetic risk. Vet Anaesth Analg, 38(1), pp.24-36.*

Septic peritonitis: a criticalist's view

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Learning objectives

- Describe the common causes and clinical presentations of patients with septic peritonitis
- Understand the diagnostic approach to such cases, and recall some of the underpinning physiologic derangements common in these patients
- Outline a rational management plan, with specific consideration given to fluid therapy, cardiovascular, nutritional support and antimicrobial selection
- Recall commonly associated clinical sequelae for this patient population

Lecture summary

Septic peritonitis is one of the leading causes of sepsis in multiple studies from the last 15 years in both cats and dogs. Patients with septic peritonitis span a wide spectrum of clinical presentations with some displaying surprising stability and others descending into sepsis, multiple organ dysfunction and death despite timely diagnosis and aggressive management. The former is easy to manage, the latter far harder; many patients fall somewhere in the middle of this range and a sensible and timely approach to their care is needed to reduce progression into a downward spiral.

Diagnosis: Septic peritonitis should be considered in any patient presenting with peritoneal effusion, especially those with acute presentations or associated cardiovascular and/or metabolic derangements until excluded as a differential. Cytological assessment of peritoneal fluid is the fastest way to obtain a diagnosis, however animals currently receiving antimicrobial administration or those with a source that has low bacterial load (such as gastric perforation) may have scant bacteria present, and so culture should always be considered if clinical concern exists. In cases with a less difficult diagnosis, evidence exists to show that markers such as the difference in glucose or lactate concentration between peritoneal fluid and blood may be useful (Martiny & Goggs, 2019). Septic peritonitis should also be a leading differential in any patient with recent gastrointestinal surgery, although for this population assessment tools such as glucose gradients from blood to peritoneal fluid should not be used.

Management: Patients should have major body systems assessed and any life-threatening aberrations dealt with, i.e. resuscitation for cardiovascular collapse. Baseline blood work helps assess the current clinical state and provides a baseline for ongoing comparison given these patients are at high risk of developing organ dysfunction throughout their hospitalisation period. Once diagnosed broad spectrum intravenous antimicrobials are recommended, my personal preference is IV amoxiclav 20mg/kg IV q8hr currently, with the addition of enrofloxacin if the patient has received amoxiclav in the preceding 3 months (based on unpublished data of resistance profiles from my hospital population). It is my practice to institute norepinephrine infusion early for its beneficial effects on not only arterial blood pressure but also reducing excessive intravenous fluid administration and potentially reducing tissue and organ oedema. In addition, in humans undergoing major surgery the use of norepinephrine to maintain baseline blood pressure during surgery rather than bolus ephedrine as deemed needed during surgery is associated with reduced organ dysfunction in the days following the procedure (Futier et al., 2017). Analgesia is mandatory given that pain causes undue welfare concerns in addition to physiologic alterations (such as immunosuppression and sympathetic nervous system upregulation); agents chosen vary depending on case specifics but NSAIDs should be avoided. Abnormalities in acid-base status/electrolytes should be corrected if causing clinical consequences, and glucose should be carefully monitored. Source control surgery is recommended as soon as is practical. This serves to reduce ongoing peritoneal contamination, removal of the 'cytokine soup' within the peritoneum which may otherwise activate numerous systems (including inflammatory, anti-inflammatory and coagulation cascades) and in some cases to obtain definitive diagnosis of the underlying disorder if imaging has not been able to elucidate the cause. Imaging prior to surgery is recommended to ascertain if the available surgical team can deal with the expected eventuality (for example, complex hepatic abscessation may be beyond the level of comfort for some outside of specialist hospitals). Imaging modality will depend on the likely causal process after obtaining a good history and performing a thorough physical examination. Both before and after surgery these patients are at risk of numerous conditions including aspiration pneumonia, acute kidney injury, coagulation disturbances, cardiovascular aberrations (such as sepsis induced myocardial depression and various arrhythmias), endothelial dysfunction and failure of any organ system; hyperbilirubinaemia in the absence of functional liver failure is common and should not be confused with such. Close attention to trends and prompt re-assessment (including blood work) and intervention are recommended to limit the risk of avoidable deterioration. At the time of surgery proactive consideration of feeding tube placement should occur, oesophagostomy tubes take little time to place and starvation is not an appropriate treatment for any known disease process. Septic patients are in a hyper-catabolic state, with proteolysis common; early enteral nutrition is recommended in all patients who can tolerate it, otherwise parenteral nutrition should be used.

References

- *Martiny, P., Goggs, R., Biomarker guided diagnosis of septic peritonitis in dogs, Front Vet Sci, 2019, 19(6);208 Futier, E., Lefrant, J.Y., Guinot, P.G., et al., Effect of individualised vs standard blood pressure management on postoperative organ dysfunction among high-risk patients undergoing major surgery: A randomised clinical trial, JAMA, 2017, 318;1346-57.*
- *Foulon, P., De Backer, D., The hemodynamic effects of norepinephrine: far more than an increase in blood pressure!, Annals of Translational Medicine, 2018, 6(Suppl 1):S25*

Pneumonia and Pneumonitis

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Learning objectives

- To recognise the spectrum of disease that can occur after an aspiration event.
- To understand the pathophysiology involved in disease caused by aspiration.
- To understand that it is outdated to administer antimicrobials for 4-6 weeks to treat cases of aspiration pneumonia.
- To appreciate the benefit of monitoring disease trajectory, and the concept of a delayed antimicrobial prescription.

Lecture summary

We appear to have made progress over recent years with regards reducing both the frequency with which antimicrobials are prescribed, and the length of time for which they are prescribed. But there is always more room for improvement. Although stated in many reference texts, it is out-dated to administer antimicrobials for 4 to 6 weeks to patients with aspiration pneumonia or until 1 week after radiographic resolution, and significantly shorter prescription lengths will usually suffice (Lappin et al. 2017, Viitanen et al. 2017, Wayne et al. 2017, Rodriguez et al. 2022, Chwala et al, 2022). The ISCAID guidelines suggest antimicrobial use be re-evaluated no later than 10 to 14 days after starting treatment, being extended, adjusted or ceased based on the clinical response (Lappin et al. 2017). In people with aspiration pneumonia, courses of 5 to 7 days in length are recommended where no extrapulmonary foci of infection exist, and where a good response to initial treatment has been demonstrated (Mandell & Niederman 2019). According to the American College of Veterinary Internal Medicine (ACVIM) consensus statement on therapeutic antimicrobial use in animals and antimicrobial resistance "a common misconception is the need to complete a minimum duration of an antimicrobial drug to prevent the emergence of resistance." The committee also states that antimicrobials should never be continued once there is clinical and microbiological evidence that an infection has been eliminated simply because of a perceived need for a minimum duration of administration (Weese et al. 2015). These concepts set the scene for more vigorously interrogating the cases in which aspiration has occurred, but for which antimicrobials are not even required at all. Aspiration pneumonitis is a condition often described conceptually, but rarely diagnosed and managed as such. It describes the situation in which pulmonary injury occurs due to inhalation of chemical irritants. Unfortunately, it is unlikely this simple though, as the initial aspiration may include gastric acid, oropharyngeal flora, blood, saliva, food particles etc. Aspiration pneumonia (usually) refers to the bacterial infection that can develop after aspiration (Marik 2001, Goggs & Boag 2015). That infection may be as a result of direct inoculation or subsequent colonisation. The term aspiration pneumopathy recognises this spectrum of disease. Teasing apart those animals in which predominantly a chemical irritation has occurred, from those with transient bacterial burden, and further still, from those with a relevant infection is the hard part; but recognition of this continuum, as well as knowledge of the underlying pathophysiology, is key. Early clinical signs of aspiration are referable to potentially self-limiting processes such as neurogenic inflammation (peaking at 1 to 2 hours post-aspiration) (Martling & Lundberg 1988) and bronchoconstriction. Experimentally, sterile aspiration events induce biphasic inflammation (at approximately 1 and 4 hours) (Kennedy et al. 1989) but which is maximal at 6 to 8 hours and occurs independently of the presence or proliferation of any bacterial isolates (Knight et al. 1993). The two variables that appear most significant in inducing an inflammatory response are the acidity and the particulate matter content of the aspirated material. Recognising these, there is a window of opportunity for simply monitoring the animal's response over time, and attempting to plot the trajectory for both diagnostic and prognostic purposes. We will discuss the use of biomarker profiles and imaging findings in such cases, and how these can be incorporated into decision making, recognising that there are a great deal of uncertainties yet to be explored! It should be recognised that there is likely a subpopulation of immunocompromised animals in which this approach is undesirable, or in which seeding of a genuine bacterial pneumonia is more likely. For example, in people, where a gastrointestinal obstructive process exists, or when proton pump inhibitors are being used, there is perceived to be a higher risk of a clinically relevant bacterial component to an aspiration event.

References

- *Chwala, M, Hartmann, F, Waller, K, Dusick, A, Viviano, K. (2022) Change over time and agreement between clinical markers of disease resolution in dogs with aspiration-induced lung injury. Vet Rec. 2022;e2280*
- *Fernandes Rodrigues, N, Giraud, L, Bolen, G, et al. (2022) Antimicrobial discontinuation in dogs with acute aspiration pneumonia based on clinical improvement and normalization of C-reactive protein concentration. J Vet Intern Med. 2022; 36(3): 1082-1088*
- *Goggs, R. A. N. & Boag, A. K. (2015) Aspiration pneumonitis and pneumonia. In: Small Animal Critical Care Medicine. 2nd edn. Eds D. C. Silverstein and K. Hopper. Elsevier, St. Louis, Mo Kennedy, T.P., Johnson, K.J., Kunkel, R. G., et al. (1989) Acute acid aspiration lung injury in the rat: biphasic pathogenesis. Anesthesia and Analgesia 69, 87-92*
- *Knight, P.R., Rutter, T., Tait, A.R., et al. (1993) Pathogenesis of gastric particulate lung injury: a comparison and interaction with acidic pneumonitis. Anesthesia and Analgesia 77, 754- 760*
- *Lappin, M.R., Blondeau, J., Boothe, D., et al. (2017) Antimicrobial use guidelines for treatment of respiratory tract disease in dogs and cats: antimicrobial guidelines working group of the International Society for Companion Animal Infectious Diseases. Journal of Veterinary Internal Medicine 31, 279-294*
- *Mandell, L.A. & Niederman, M.S. (2019) Aspiration Pneumonia. The New England Journal of Medicine 380, 651-663*
- *Marik, P.E. (2001) Aspiration pneumonitis and aspiration pneumonia. The New England Journal of Medicine 344, 665-671 Martling, C. R. & Lundberg, J. M. (1988) Capsaicin sensitive afferents contribute to acute airway edema following tracheal instillation of hydrochloric acid or gastric juice in the rat. Anesthesiology68, 350-356*
- *Viitanen, S.J., Lappalainen, A. K., Christensen, M.B., et al. (2017) The utility of acute-phase proteins in the assessment of treatment response in dogs with bacterial pneumonia. Journal of Veterinary Internal Medicine 31, 124-133*
- *Wayne, A., Davis, M., Sinnott, V. B., et al. (2017) Outcomes in dogs with uncomplicated, presumptive bacterial pneumonia treated with short or long course antibiotics. The Canadian Veterinary Journal 58, 610- 613*
- *Weese, J.S., Giguere, S., Guardabassi, L., et al. (2015) ACVIM consensus statement on therapeutic antimicrobial use in animals and antimicrobial resistance. Journal of Veterinary Internal Medicine 29, 487- 498*

Gary stamp memorial lecture: pyothorax: Medical or surgical management

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Learning objectives

- Know the key aspects of medical management of pyothorax in dogs and cats
- Consider the potential indications for surgical management of pyothorax in dogs and cats
- Review the published evidence for management and outcome of pyothorax in dogs and cats

Lecture summary

Pyothorax is the presence of septic exudate in the pleural space and can have a variety of causes including thoracic bite wounds, esophageal perforation, hematogenous spread of infection, rupture of an intrathoracic abscess and migrating foreign material. Dogs and cats with pyothorax frequently present for increased respiratory rate and effort and other general signs of systemic illness such as fever, lethargy and inappetence. Diagnosis generally includes imaging such as thoracic radiographs and/or thoracic ultrasound demonstrating pleural effusion. The diagnosis is confirmed by evidence of organisms on cytology and/or culture of the effusion. It is important to note that bacteria may not be evident if animals are on antibiotic therapy at the time of evaluation and non-bacterial causes of pyothorax such as yeast have been reported.

Outcome: Pyothorax can be managed medically or surgically, the indications for surgical management are influenced by the geographical region, the species and the results of diagnostic imaging. Medical management of pyothorax includes pleural fluid drainage, antibiotic therapy, and supportive care. Cats with pyothorax are most commonly managed medically with thoracostomy tube placement and this approach is reported to have good outcomes with 73-95% survival to discharge.[1-3] The success of medical management of pyothorax in dogs varies between studies with survival to hospital discharge ranging from 71 to 100%.[4-6]

Initial management: Initial evaluation of animals with pyothorax should focus on respiratory and cardiovascular stability. All animals with pyothorax should have thoracocentesis performed and the pleural space maximally drained. Pleural fluid samples should be collected in an aseptic manner for cytology as well as culture and susceptibility testing. Empiric, broad spectrum, antimicrobial therapy should be initiated quickly and can be reviewed once the culture and susceptibility results are available. The bacteria identified in pyothorax are most commonly anaerobes but many others including Staphylococcus spp., Pasteurella spp., Actinomyces spp. and Nocardia organisms have been isolated.[7] Advanced imaging such as CT scanning can be beneficial in evaluation of the extent of the disease as well as helping determine if surgery is indicated.

Thoracostomy tube placement: Thoracostomy tube placement is recommended for the majority of pyothorax patients although thoracocentesis alone has been successful in cats and dogs. If a very small volume of pleural effusion is present, thoracocentesis maybe sufficient but with larger volume effusion, chest tube placement is strongly recommended. Thoracostomy tubes are left in place until the fluid production rate decreases and the nature of the fluid has improved. There is no exact fluid volume indication for chest tube removal, 3 to 5 ml/kg/day has been suggested as a general guideline. Infusion of medications via the thoracostomy tube such as antimicrobial drugs, heparin or fibrinolytic drugs has not been found to be of benefit in human medicine and is not recommended in the treatment of pyothorax.

Antimicrobial therapy: The International Society for Companion Animal Infectious Diseases recommends parenteral administration of a fluoroquinolone with either a penicillin or clindamycin as the first line drug options for pyothorax.[7] Intravenous antimicrobial drug administration is recommended in the initial period given the potential for life threatening sepsis. Duration of antimicrobial therapy in the treatment of pyothorax is debatable. Traditionally long treatment periods (weeks to months) have been recommended. The International Society for Companion Animal Infectious Diseases recommends treatment for a minimum of 3 weeks and ideally 4 to 6 weeks.[7] Although it is acknowledged that more evidence is needed to determine the optimal treatment duration. One suggestion is to re-evaluate animals every 2 weeks after discharge with thoracic radiographs and to continue antimicrobial therapy for 2 weeks after radiographic resolution.[8]

Surgical management: Surgical management of pyothorax is most commonly performed through a median sternotomy approach to allow full exploration of the thoracic cavity. The goals of surgical intervention include identification and removal of any inciting cause, removal of grossly abnormal or necrotic tissue, breakdown of fibrous adhesions, lavage of the pleural cavity to remove infected fluid and to decrease bacterial load, and placement of bilateral thoracostomy tubes.[9] The indications for surgical intervention vary and have not been clearly defined. It is important to recognized that geographical differences in etiology of pyothorax will influence this decision process. In areas where migrating foreign bodies are common, early surgical intervention is recommended in dogs.

References

- *Barrs VR, Allan GS, Martin P, et al. Feline pyothorax: a retrospective study of 27 cases in Australia. J Feline Med Surg 2005;7:211-22.*
- *Demetriou JL, Foale RD, Ladlow J, et al. Canine and feline pyothorax: a retrospective study of 50 cases in the UK and Ireland. J Small Anim Pract 2002;43:388-94.*
- *Waddell LS, Brady CA, Drobatz KJ. Risk factors, prognostic indicators, and outcome of pyothorax in cats: 80 cases (1986-1999). J Am Vet Med Assoc 2002;221(6):819-24.*
- *Boothe HW, Howe LM, Boothe DM, et al. Evaluation of outcomes in dogs treated for pyothorax: 46 cases (1983-2001). J Am Vet Med Assoc 2010;236(6):657-63.*
- *Rooney MB, Monnet E. Medical and surgical treatment of pyothorax in dogs: 26 cases (1991-2001). J Am Vet Med Assoc 2002;221(1):86-92.*
- *Johnson MS, Martin MWS. Successful medical treatment of 15 dogs with pyothorax. J Small Anim Pract 2007;48:912-6.*
- *Lappin MR, Blondeau J, Boothe D., et. al. Antimicrobial use guidelines for treatment of respiratory tract disease in dogs and cats: Antimicrobial guidelines working group of the international society for companion animal infectious disease. J Vet Intern Med 2017;31:279-294*

Upper respiratory emergencies

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Learning objectives

- Learn normal anatomy and physiology of the upper airways
- Identify common causes of upper respiratory disease in dogs and cats including brachycephalic airway syndrome, laryngeal paralysis, tracheal collapse, nasopharyngeal polyps and oropharyngeal neoplasia
- Learn how to diagnosis and manage these diseases in the ER including sedation, oxygen therapy and definitive treatments.

Lecture summary

Pathophysiology: The upper respiratory system includes the nasal passages, nasopharynx, oropharynx, larynx and cervical tracheal. Upper airway disease can occur secondary to anatomic abnormalities, edema, degenerative diseases, foreign bodies, neoplasia or infection. Upper airway obstruction develops secondary to narrowing of the upper airways. This narrowing may be static or dynamic. Static disease is always present and typically results in clinical signs of both inspiratory and expiratory dyspnea. Dynamic lesions may produce dyspnea during inspiration or expiration pending on the site of obstruction. Intrathoracic obstruction typically results in increased effort during expiration while extra thoracic obstruction results in increased effort during inspiration.
Clinical Signs: Clinical signs associated with upper airway disease may vary based on the site and severity of disease. One of the hallmark signs of upper airway disease is the presence of stertor, a low pitched "snoring" sound that signifies abnormalities orad to the larynx or stridor, a high-pitched noise typically heard on inspiration that is associated with disease of or aborad to the larynx. Laryngeal and pharyngeal disease can also result in dysphonia (change in bark/meow).
Emergency treatment: Animals with upper respiratory obstruction can present in varying degrees of respiratory distress. Immediate stabilization should focus on administering supplemental oxygen and sedating the patient. Commonly used sedatives include acepromazine (0.005 - 0.02mg/kg IV or 0.01- 0.05 mg/kg IM) and Butorphanol (0.1 - 0.5 mg/kg IV or IM), which can be used independently or in combination for sedation. For patients in severe distress where sedation and supplemental oxygen are not improving their distress, intubation and/or tracheostomy following a propofol titration (0.05 - 2 mg/kg IV titration) may be necessary for stabilization. Most animals with upper airway obstruction will have a marked improvement and normalization of their breathing patterns and mucous membrane colors with intubation. Anti-inflammatory glucocorticoid therapy (dexamethasone sodium phosphate 0.05 - 0.15 mg/kg IV or IM), if not contraindicated, may be administered to help decrease upper airway inflammation. These patients also commonly present hyperthermic. Active cooling with room temperature intravenous fluids, fans and room temperature baths should be instituted in patients with a temperature > 104°F. Once their temperature has reached 103°F they should be dried and active cooling stopped as their temperature will likely continue to drop and avoidance of hypothermia is important. Ice packs should not be placed on the patient as this causes local vasoconstriction. Placing alcohol on the pads of the feet is also not recommended as the surface area is too small to be effective. Once the patient is stable the underlying cause of their upper airway disease can be addressed.
Brachycephalic airway syndrome: Brachycephalic airway syndrome is a constellation of clinical signs including stenotic nares, elongated soft palate, everted laryngeal saccules, everted tonsils, laryngeal edema and collapse, tracheal hypoplasia, and nasopharyngeal turbin ates. Affected animals include brachycephalic breeds such as English Bulldogs, Pugs, French Bulldogs and Boston Terriers. Emergency stabilization followed by definitive surgical correction is often required.
Laryngeal paralysis: Laryngeal paralysis results from dysfunction of the recurrent laryngeal nerve which innervates the cricoarytenoideus muscle responsible for laryngeal abduction during inspiration. Most affected animals are middle aged to older, large breed dogs, with Labrador retrievers overrepresented. Younger animals may also be affected with a congenital type of laryngeal paralysis. It is now thought that laryngeal paralysis may be part of a larger neuromuscular disorder termed geriatric onset laryngeal paralysis polyneuropathy (GOLPP) as many dogs have esophageal motility disorders or hind limb paresis concurrently. Emergency stabilization followed by definitive surgical correction is often required.
Tracheal collapse: Tracheal collapse is a chronic, progressive disorder of tracheal cartilage that primarily affects toy and small breed dogs, with the Yorkshire Terrier overrepresented. Clinical signs include a "honking" cough, stridor, gagging after eating or drinking, and exercise intolerance or collapse. While some of these patients are able to be medically managed, others will require definitive correction (extraluminal tracheal rings or intraluminal stent placement). Feline upper airway disorders: Cats are less likely to present for signs of upper airway obstruction and when they do, they are likely to suffer from space occupying lesions such as nasopharyngeal polyps or neoplasia. Emergency management is similar to dogs. Definitive treatment for nasopharyngeal polyps is either traction-avulsion or ventral bullae osteotomy and overall prognosis is good. Definitive treatment of oropharyngeal neoplasia may prove challenging and is dependent on the type and location at the time of presentation.

References

- *Allen, HS, Broussard J, Noone K. Nasopharyngeal diseases in cats: a retrospective study of 53 cases (1991-1998). J Am Anim Hosp Assoc (1999) 35 (6): 457-461. Available at https://doi.org/10.5326/15473317-35-6-457*
- *Bookbinder, LC, Flanders J, Bookbinder PF, et al. Idiopathic Canine Laryngeal Paralysis as One Sign of a Diffuse Polyneuropathy: An Observational Study of 90 Cases (2007-2013). Veterinary Surgery (2016) 45(2): 254-260. Available at https://doi.org/10.1111/vsu.12444*
- *Clarke, D. Upper Airway Disease. In: Silverstein, DC, Hopper K (eds.) Small Animal Critical Care Medicine. 2nd ed. St. Louis, MO. Elsevier Saunders; 2015 p. 92-104.*
- *Dupre, G, Heidenriech D. Brachycephalic Syndrome. Vet Clinics North America: SA Practice. (2016) 46 (4): 691-707. Available at: https://doi.org/10.1016/j.cvsm.2016.02.002*
- *Ginn, JA, Kumar MSA, McKiernan, BC, Powers, BE. Nasopharyngeal Turbinates in Brachycephalic Dogs and Cats. J Am Anim Hosp Assoc (2008) 44 (5): 243-249. Available at: https://doi.org/10.5326/0440243*
- *Maggiore, AD. An Update on Tracheal and Airway Collapse in Dogs. Vet Clinics North America: SA Practice. (2020) 50 (2): 419-430*

It takes a team: evidence-based communication tools for the veterinary team

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Learning objectives

- Explain the principles of good leadership of a veterinary healthcare team.
- Describe the 4 fundamental characteristics of good communication in a veterinary healthcare setting.
- Demonstrate techniques and tools for achieving situation awareness through situation monitoring skills.
- Describe the principles of mutual support and the behaviors that can maximize a team's ability to ask for and offer help to allocate resources where they are needed.

Lecture summary

Teamwork is defined by a set of interrelated knowledge, skills, and attitudes (KSAs) that facilitate coordinated, adaptive performance, improving a group's ability to meet its objectives. It is estimated that 80% of serious medical errors in human health care occur due to miscommunication among the team of caregivers, which led to the development of TeamSTEPPS® by the Department of Defense (DoD) and the Agency for Healthcare Research and Quality (AHRQ). An adaptation of this framework for veterinary medicine, VetTeams, applies the 4 basic concepts of TeamSTEPPS® training, leadership, communication, situation monitoring, and mutual support to veterinary clinical practice.

Leadership: Effective leaders manage resources and facilitate team actions to ensure that all members of the team are seeking information, plan and continually refine team duties, coordinate team actions, resolve conflict among team members, and provide coaching and feedback. Leaders must balance the role of handing down solutions to problems with that of facilitating problem solving the team. By developing a shared vision with the team, facilitating coordination and collaboration, and motivating team members, effective leaders maximize the performance of each team member and improve patient care. Three communication tools can be used by team leaders to establish a supportive, collaborative environment in which each member of the team can maximally contribute to patient care: briefs, huddles, and debriefs.

Communication: In general, all communication between team members should adhere to the following 4 principles of effective communication - (1) Complete: communicate all relevant information, (2) Clear: convey information that is plainly understood (3) Brief: communicate all information concisely, (4) Timely: make information available when needed, verify its accuracy, and acknowledge the information. Briefs and huddles are excellent tools to foster understanding among all team members. In addition, two specific communication tools, call-outs and check-backs, can help reduce miscommunication.

Situation Monitoring: Situation monitoring is an individual skill that involves active assessment of the clinical setting and continual scanning of the environment and allows each member of a health care team to maintain an accurate awareness and understanding of the current "situation." It is essential that all members of the health care team maintain these monitoring activities, which contribute to team cognition. By practicing situation monitoring, the team can develop a shared mental model of any clinical situation and work as a highly effective unit in which each member is contributing maximally to patient care.

Mutual Support: Mutual support can be thought of as the employment of "back-up behaviors" among members of a team It involves reallocating resources to a member of a team to help that team member achieve the desired goal when it is apparent that the team member is failing. At its core, mutual support is simply the concept of helping others on the team perform their tasks to optimize patient care and address team member limitations and other demands. Ultimately, mutual support in a healthcare environment is an acknowledgement that the clinical setting is frequently characterized by a high workload in combination with the requirement to efficiently complete acute, time-sensitive tasks.

References

- *https://www.ahrq.gov/teamstepps*
- *Mohsen MM, Gab Allah AR, Amer NA, Rashed AB, Shokr EA. Team Strategies and Tools to Enhance Performance and Patient Safety at primary healthcare units: Effect on patients' outcomes. Nurs Forum. 2021 Oct; 56(4):849-859.*
- *Stead K, Kumar S, Schultz TJ, Tiver S, Pirone CJ, Adams RJ, Wareham CA. Teams communicating through STEPPS. Med J Aust. 2009 Jun 1;190(S11):S128-32.*

Looking at medical excellence with a dual process lens

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Learning objectives

- Discuss the dual process approach to clinical based reasoning
- Compare and contrast the utilization of System 1 and System 2 thinking in clinical based reasoning
- Discuss unconscious bias and its impact on clinical reasoning
- Be able to utilize the concepts of unconscious bias and dual process theory to discuss adverse events related to clinical based reasoning

Lecture summary

Clinical teachers have an obligation to promote and foster high-quality patient care, which requires one to simultaneously be able to diagnose the patient as well as the learner's ability and skill to do so. This requires a clinical teacher to understand and apply how clinician's think in their approach training. One approach, called the dual process theory, has been adapted from psychology literature to describe how clinicians think. This dual process involves a Systems 1 thinking that is fast, intuitive, and mostly based on pattern recognition. This is how you end up at your house when driving home from work without really thinking about it. This is why a veterinarian thinks of cancer when one mentions a sick older golden retriever. You develop these patterns over time with exposure and clinical experience. Systems 2 thinking is slower, cognitive, and analytical in nature. It is the problem-based approach to clinical reasoning and requires clinicians to systematically consider multiple etiologies to avoid making diagnostic errors and assumptions. It works best with complicated and atypical scenarios, where more than one process may be involved. In dual process theory, the brain searches for patterns first and flips through variations on patterns, resorting to analysis, or System 2, when the available patterns do not fit. These mental shortcuts, or heuristics, are part of how the human brain has evolved to avoid cognitive overload.

In dual process theory, which system is used primarily depends upon a clinician's experience as reflected by the depth and breadth of their illness scripts. An illness script defines a classic or typical clinical presentation or pattern of a disease process by incorporating several factors: epidemiology (signalment, regionality, etc..), time course, salient clinical signs, typical key diagnostics, and treatment plan, as well as anticipated response and prognosis. There are often three or four disease processes with similar characteristics but as one gains experience, key findings in a script help to differentiate between them. For example, cats with asthma and heart failure can present with similar respiratory effort and distress, but on examination their body temperature, point of care ultrasound findings, and presence of absence of a historical cough can often differentiate them. Clinicians with less experience may focus more on the presence or absence of a murmur, bloodwork changes, and items that clinicians with more experience recognize to have lower diagnostic yield. However, there is an inherent risk if one exclusively uses a System 1 approach, as previous experiences may invoke tunnel vision or introduce unconscious biases. For this reason, as a clinical teacher it is important to discuss establishing rejecting features of the diagnosis in any illness script. This allows the clinician to pause, reflect on their script and prove they are correct by sorting through factors that confirm similar scripts to be incorrect. In other words, develop the habit to transiently switch to Systems 2 thinking after knowingly utilizing Systems 1 in clinical decision making. Unconscious or implicit bias may also come into play in both types of thinking, although type 1 is most prone. Bias is the psychological tendency to make a decision based on incomplete information and subjective factors instead of empirical evidence. Some of the most common in clinical medicine include anchoring bias, in which the clinician fixates on a particular aspect of the of the presentation and ignores other facts as a result. Premature closure is a bias in which the clinician stops inquiring further once they believe they have found the solution or diagnosis. Confirmation bias occurs when a clinician preferentially weights findings that confirm or reinforce a pre-existing opinion. These are a few examples of hundreds of unconscious biases that exist. Clinicians should be made aware of them, along with tools to prevent or reconcile biases when they occur.

It is hoped that exploration and understanding of these approaches to thinking and clinical reasoning may help to prevent errors in clinical decision making. A crucial component to this exploration may be the ability of a teacher and/or mentor to normalize and discuss clinical reasoning as part of rounds. Similar to morbidity and mortality rounds, one can promote the conscious application of Systems based thinking, reflection, and recognition of unconscious bias when reviewing a clinical scenario with a suboptimal outcome. The author will share examples of this in the seminar.

References

- *Doherty T, et al. Believing in overcoming cognitive bias. AMA Journal of Ethics, Sept 2020, 22 (9): E773-778*
- *Yuen T, et al. Cognitive bias: Its influence on clinical diagnosis. J of Family Practice, June 2018, 68(6): 366-372.*
- *Croskerry P, et al. Better clinical decision making and reducing diagnostic error. J R Coll Physicians Edinb 2011; 41:155-162*

Life-threatening endocrine emergencies - internal medicine and critical care views on the management of complications associated with hypoadrenocorticism

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Learning objectives

- Recognize possible complications of hypoadrenocortism
- Describe the treatment options of hypoadrenocortism in the acute phase including appropriate fluid therapy
- Understand the treatment options of hypoadrenocortism in the long-term management

Lecture summary

Primary hypoaldosteronism (Addison's disease) is an endocrine condition characterized in most cases by a lack of glucocorticoids and mineralocorticoids which is most often caused by immune-mediated destruction of the adrenal cortex. Although often diagnosed in middle-aged purebred dogs, hypoaldosteronism (HA) has also been reported in young, older, and cross-bred dogs, as well as in cats.[1,2] HA manifests itself by vague clinical signs such as inappetence, weight loss, weakness, lethargy, or waxing and waning gastrointestinal signs. Some dogs do not present the typical combination of hyponatremia and hyperkalemia. These cases are called "atypical hypoadrenocorticism". However, the name "eunatremic, eukalemic hypoadrenocorticism" is now suggested. In fact, although hypocortisolism alone exists, dogs can have a deficiency in both hormones without having electrolyte imbalances. This would be due to the presence of compensatory mechanisms protecting against changes in natremia and kalemia.[3] Only the measurement of aldosterone allows differentiation of the two.

Diagnosis: Hypoadrenocorticism is quickly suspected in the absence of a stress leukogram, and in the presence of a decreased Na/K ratio, hypoglycemia, hypercalcemia, and signs of shock, when the animal is in Addisonian crisis. The diagnosis is then confirmed by the demonstration of low baseline and post-ACTH stimulation cortisol (< 1 µg/dL [< 28 nmol/L]). However, because the disease manifests itself with vague and varied signs and electrolyte imbalances are not always present, the challenge is to decide whether to test a patient or not. A baseline cortisol > 2 µg/dL (> 55 nmol/L) is often used as a screening test to rule-out HA. Although studies have shown alterations in urinary sodium concentration and in urinary sodium and potassium fractional excretions, assessment of urinary electrolytes does not appear, at this time, to provide any utility in the diagnosis of HA.[4,5] However, measurement of the urine cortisol-to-creatinine ratio would seem to be a good screening test. It has even been suggested that it could be used to confirm the diagnosis of HA, for example when ACTH stimulation is not possible, but this remains to be supported with larger studies.[6,7] Finally, machine-learning tools that consider objective clinicopathological data have recently been developed and evaluated to aid in the decision to test or not a patient.[8,9]

Acute Management: When the patient is presented in an Addisonian crisis, acute treatment consists of correction of hypovolemia and signs of shock, hypoglycemia, hyperkalemia and its consequences, and acid-base disturbances. In general, balanced isotonic fluids are good choices because they allow improvement of metabolic acidosis and have a lower sodium concentration than 0.9% NaCl, thus reducing the risk of overly rapid correction of natremia. Glucocorticoids should be promptly administered during stabilization of the patient. Only dexamethasone does not interfere with the ACTH stimulation test and is therefore the drug of choice in the absence of a definitive diagnosis. Once the tests have been performed, hydrocortisone or prednisolone can be used. In general, mineralocorticoids are given only after the diagnosis is confirmed. In addition, since fluids help normalize electrolytes, administering mineralocorticoids too rapidly could lead to a sudden and undesirable increase in sodium.

Complications: As mentioned above, rapid sodium changes must be avoided. Indeed, this could lead to the development of myelinolysis, which characterized by neurological signs. Another complication that can occur in hypoadrenocorticism is acute kidney injury most likely secondary to the shock and hypotension seen in Addisonian crisis. Case reports have also described episodes of third-degree atrioventricular block, systolic ventricular dysfunction, non-cardiogenic pulmonary edema, and very severe intestinal hemorrhage requiring blood transfusions in dogs treated for HA.[10-13] Veterinarians must be aware of these possible complications to be able to recognize and treat them accordingly.

Chronic Management: Long-term quality of life is unaffected or minimally affected in most dogs diagnosed with hypoadrenocorticism. However, its management requires a high level of commitment from the owner, and costs can be considerable. It is important for the veterinary team to discuss with the client about the various long-term options for mineralocorticoid supplementation, including the choice between DOCP and fludrocortisone and the associated costs[14]. In a Western European survey, veterinarians seem to prefer DOCP for mineralocorticoid supplementation because it provides better electrolyte control and patient response than fludrocortisone[15]. It is also important to note that a DOCP dose of 1.1 mg/kg, which is lower than the manufacturer's recommended dose, is generally safe and effective.[16] In this presentation, the views of both an intensivist and an internist will be shared to optimize management strategies in the acute and chronic phases of the condition. We will cover life-threatening complications and common pitfalls related to hypoadrenocorticism.

References

- *Peterson ME, Greco DS, Orth DN. Primary Hypoadrenocorticism in Ten Cats. J Vet Intern Med. 1989;3(2):55-58. doi:10.1111/J.1939-1676.1989.TB03080.X*
- *Guzmán Ramos PJ, Bennaim M, Shiel RE, Mooney CT. Diagnosis of canine spontaneous hypoadrenocorticism. Canine Med Genet. 2022;9(1). doi:10.1186/S40575-022-00119-4*
- *Gallego AF, Gow AG, Boag AM. Evaluation of resting cortisol concentration testing in dogs with chronic gastrointestinal signs. J Vet Intern Med. 2022;36(2). doi:10.1111/jvim.16365*
- *Dropkin CA, Kruger JM, Langlois DK. Evaluation of urine electrolytes for the diagnosis of hypoadrenocorticism in dogs. Vet Clin Pathol. 2021;50(4):507-514. doi:10.1111/VCP.13018*
- *Lennon EM, Hummel JB, Vaden SL. Urine sodium concentrations are predictive of hypoadrenocorticism in hyponatraemic dogs: a retrospective pilot study. J Small Anim Pract. 2018;59(4). doi:10.1111/jsap.12792*
- *Moya MV, Refsal KR, Langlois DK. Investigation of the urine cortisol to creatinine ratio for the diagnosis of hypoadrenocorticism in dogs. J Am Vet Med Assoc. 2022;260(9):1041-1047. doi:10.2460/JAVMA.21.12.0538*
- *Del Baldo F, Gerou Ferriani M, Bertazzolo W, Luciani M, Tardo AM, Fracassi F. Urinary cortisol-creatinine ratio in dogs with hypoadrenocorticism. J Vet Intern Med. 2022;36(2). doi:10.1111/jvim.16358*
- *Reagan KL, Reagan BA, Gilor C. Machine learning algorithm as a diagnostic tool for hypoadrenocorticism in dogs. Domest Anim Endocrinol. 2020;72. doi:10.1016/j.domaniend.2019.106396*
- *Reagan KL, Pires J, Quach N, Gilor C. Evaluation of a machine learning tool to screen for hypoadrenocorticism in dogs presenting to a teaching hospital. J Vet Intern Med. 2022;36(6):1942. doi:10.1111/JVIM.16566*
- *Kimura Y, Iwaki S, Kameshima S, Itoh N. A case of canine hypoadrenocorticism needing blood transfusion for severe acute anemia due to gastrointestinal hemorrhage. J Vet Med Sci. 2020;82(1). doi:10.1292/jvms.19-0075*
- *Paulin MV, Snead EC. Case report: Hypoadrenocorticism crisis complicated by non-cardiogenic pulmonary edema in a dog. Front Vet Sci. 2022;9. doi:10.3389/FVETS.2022.1015739*
- *McGrath CA, Borgeat K, Wilson HE. Third-degree atrioventricular block with narrow QRS complexes in a case of hypoadrenocorticism in a dog. J Small Anim Pract. 2022;63(7):559-562. doi:10.1111/JSAP.13464*
- *Gunasekaran T, Sanders RA. Ventricular systolic dysfunction in dogs diagnosed with hypoadrenocorticism. J Vet Cardiol. 2022;41:231-235. doi:10.1016/J.JVC.2022.04.002*
- *Hazuchova K. Optimising the long-term management of dogs with hypoadrenocorticism. Vet Rec. 2022;191(10):414-416. doi:10.1002/VETR.2458*
- *Rebocho R, Domínguez-Ruiz M, Englar RE, et al. Use of Deoxycorticosterone Pivalate by Veterinarians: A Western European Survey. Vet Sci. 2021;8(11). doi:10.3390/vetsci8110271*
- *Vincent AM, Okonkowski LK, Brudvig JM, et al. Low-dose desoxycorticosterone pivalate treatment of hypoadrenocorticism in dogs: A randomized controlled clinical trial. J Vet Intern Med. 2021;35(4):1720-1728. doi:10.1111/JVIM.16195*

Multimodal analgesia

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Learning objectives

- Understand the concept of multimodal analgesia and its use targeting pain
- Explain the alternative definitions of acute and chronic pain
- Identify the five pain pathway stages
- Classify different analgesic drugs and identify their receptors
- Describe how the most common analgesics interrupt the pain pathway

Lecture summary

Pain definition and Multimodal analgesia approach: Pain is defined as an unpleasant emotional and physical experience that can potentially cause tissue damage, and its treatment is still one of the major challenges in veterinary medicine. Pain can be classified as adaptative (acute) or maladaptive (chronic). Adaptative pain comprises both nociceptive and inflammatory pain and is described as protective and reversible, whereas maladaptive pain includes neuropathic and functional pain, is non-protective and results from plastic changes in the pain processing system associated with long-term pain (for more than 3 months) (Adrian et al 2017). The multimodal analgesia approach aims to target a variety of pain receptors using different classes of analgesics, decreasing drug doses and their side effects (Self & Grubb 2019). As to human medicine, many veterinary anesthetists are moving away from opioid anesthesia, based on a multimodal approach and the exponential advances of loco-regional techniques in recent decades. To provide effective multimodal analgesia it is essential to understand the interaction between different analgesics in the pain pathway inhibiting the neurotransmitters' potential. On the other hand, pain scoring needs to be performed to assess pain relief effectiveness and avoid drug overdose.

Pain pathway: The pain pathway is divided into five stages: transduction, transmission, modulation, projection and perception. Transduction is the conversion of a chemical, thermal or mechanical stimulus into nociceptive impulses. When the nerve fibers reach their threshold limit, the nerve impulses travel to the spinal cord generating an action potential, resulting in peripheral sensitization. The primary afferent nerves carry the action potential from the nerve terminals to the dorsal horn of the spinal cord (transmission), where the impulses are inhibited or amplified by the neurotransmitters (modulation) and central sensitization occurs. Once the nerve impulse is modulated in the spinal cord, nociception is projected to the brain through the dorsal horn of the spinal cord, and the sensory information is processed by the brain inducing a nociceptive response (Self & Grubb 2019).
In the anaesthetized patient, we can observe an increase in heart and respiratory rate and blood pressure as a physiological response to pain. Although the patient is not aware of the nociceptive stimulus, when recovering from anesthesia the pain receptors will be activated and the patient will perceive intensive pain (Self & Grubb 2019), which is why pre-emptive, and peri anaesthetic analgesia rescue is so important. Pain relief aims to prevent peripheral and central sensitization, and when it is not adequately provided, a nociceptive stimulus can evolve from an acute to a chronic state due to neuroplastic changes occurring in the central nervous system. (Adrian et al 2017). Neuroplasticity is the ability of the central nervous system to adapt its physiological and behavioral responses to a nociceptive stimulus and can result in hyperalgesia and/or allodynia. Therefore, a patient that experiences continuous painful stimuli or a major physical trauma might be less responsive to a uni-modal analgesic treatment (Goich et al 2019).

Analgesic drugs: For this lecture, different analgesics will be described. Opioids, ketamine, and local anesthetics will be discussed in more detail regarding their interaction with the pain pathway. The analgesic drugs interact with different receptors located in the central nervous system, interrupting the pain pathway. For example, NSAIDs cease the nociceptive stimulus during transduction, whereas local anesthetics interrupt the transmission of the nerve impulses by binding to the sodium channels along the nerve fibers, blocking pain sensation. Both opioids and ketamine have an important role in pain modulation by interacting with the depolarization of the neurons, inhibiting the release of neurotransmitters.

References

- *Adrian, D., Papich, M., Baynes, R., Murrel, J. and Lascelles, B. Chronic maladaptative pain in cats: A review of current and future drug treatment options, UK: The Veterinary Journal, 2017, 230; 52-61.*
- *Bradbrook, C. and Clark, L. State of the art analgesia- recent developments in pharmacological approaches to acute pain management in dogs and cats. Part 1, UK, The Veterinary Journal,2018, 238; 76-82.*
- *Bradbrook, C. and Clark, L. State of the art analgesia- recent developments in pharmacological approaches to acute pain management in dogs and cats. Part 1, UK, The Veterinary Journal,2018, 236; 62-67.*
- *Forget, P. Opioid-free anaesthesia. Why and How? A contextual analysis. Anaesthesia Critical Care & Pain Medicine, 2019, 38 (2); 169-172.*
- *Goich, M., Bascuñán, A., Faúndez. and Valdés, A. Multimodal analgesia for treatment of allodynia and hyperalgesia after major trauma in a cat, UK: Journal of Feline Medicine and Surgery Open Reports, 2019, 5 (1); 1-8.*
- *Gruen, M., Lascelles B., Colleran, E., Johnson, J. and Marcellin-Little, D. 2022 AAHA Pain Management Guidelines for Dogs and Cats, J Am Anim Hosp Assoc, 2022; 58; 55-76.*
- *Grubb, T. and Lobprise, H. Local and regional anaesthesia in dogs and cats: Overview of concepts and drugs (Part 1), Veterinary Medicine and Science, 2020, 6; 209-217.*
- *Pascal, M., Allison, A. and Kaartinen, J. Opioid-sparing effect of a medetomidine constant rate infusion during thoracolumbar hemilaminectomy in dogs administered a ketamine infusion. Veterinary Anaesthesia and Analgesia, 2020, 47 (1); 61-69.*
- *Self, I. and Grubb, T. BSAVA Guide to Pain Management in Small Animal Practice, chapter 2 Physiology of Pain, Gloucester, England; BSAVA, 2019, 13.*

Pain scoring the critical patient

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Learning objectives

- Recognise the physiological effects of pain
- Identify pain behaviours
- Appraise different pain scoring scales available for dogs and cats
- Assess pain using pain scoring systems

Lecture summary

The pain scoring systems are based on physiological, behavioural and/or facial expressions assessment, suggesting guidelines to adjust analgesia if required. The introduction of pain scales in veterinary medicine has changed how pain is currently recognised and treated however, in busy practices and hospitals it can be a challenging routine to implement, often leading to the administration of prescribed analgesia without prior pain assessment or to suboptimal analgesia.

Physiological effects of pain and behavioural changes: Adequate pain relief improves patient care and clinical outcomes preserving the animal's welfare. Poor pain management can result in two scenarios: overdosing and subsequent sedation or dysphoric behaviours, and suboptimal analgesia associated with behavioural and physiological changes related to pain sensation.Pain is described as an unpleasant experience with real or potential tissue damage and has physiological effects on the body such as tachycardia, hypertension, lethargy, inappetence, pupil dilation and hypersalivation. This can lead to catecholamine release, hyperglycaemia, leucocytosis, cytokine production, immune suppression, and respiratory impairment, increasing the risk of further complications (Self & Grubb 2019). It is important to recognise that some abnormal behaviours and body postures can reflect pain. Painful patients may show signs of aggression or fear, self-trauma, vocalisation, and reluctance to move and to be touched. As animals are not aware that pain is temporary, a painful episode can lead to a traumatic experience. Untreated severe pain can result in sensitisation to nociceptive stimulus leading to chronic pain (Janasi 2016).

Pain Scoring Scales: In human medicine, self-report is considered the most reliable and accurate way to assess pain, however, in critically ill patients verbal documentation of pain may not be possible. Severgnini et al (2016) demonstrated that the combination of the Critical Care Pain Observation Tool (CPOT) and the Behavioural Pain Scale improves pain recognition in critical conscious and unconscious patients compared with both scales used separately, highlining that facial expression is a relevant parameter for assessing pain in these patients. In veterinary medicine, we use similar pain-scoring tools to evaluate pain in our patients.

Pain scoring scales validated for dogs: Glasgow Composite Measure Pain Scale (GCMPS) - is validated to assess acute pain and is based on six categories of behavioural descriptions, including the patient's response to gentle palpation of the painful area. It is an objective numerical scale, easy to use but can be affected by the patient's temperament. The maximum score is 24, and the cut-off score to administer rescue analgesia is ≥ 6/24 or ≥ 5/20. For cats includes the evaluation of facial expressions. Colorado State University Veterinary Medical Center Acute Pain Scale - evaluates physiological behaviours and has realistic drawings that help to use the pain scale. It includes gentle palpation of the painful area and has instructions not to disturb the patient if they are asleep. The maximum score is 4 and the analgesic plan should be reassessed when ≥ 2/4. Although it is available for cats is still not validated for feline species.

Pain scoring scales validated for cats: Feline Grimace Scale - is based on changes in facial expressions and does not require close interaction with the patient. Assesses the ear position, orbital tightening, muzzle tension, whisker, and head position. This pain scoring scale is easy to use and requires pre- and post-analgesia assessment. The maximum score is 10 and rescued analgesia must be administered when ≥ 4/10. UNESP Botucatu Multidimensional Composite Pain Scale - evaluates pain expressions, reaction to palpation of the painful area, and psychomotor changes such as patient's posture, comfort, attitude, activity, and physiological variables. Although it is a comprehensive pain scale, it has the advantage of being available in eight languages. The maximum score is 12 and when ≥ 4/12 rescue analgesia must be provided. Glasgow Composite Measure Pain Scale For chronic pain assessment, the following pain scoring systems are available: Feline Musculoskeletal Pain Index, Canine Brief Pain Inventory, Liverpool osteoarthritis in Dogs, Helsinki Chronic pain Index

The pain scoring systems also help to identify patients experiencing pain and not showing signs. Animals submitted to painful procedures should be regularly evaluated for pain, according to the time of the last dose of analgesic received and its duration of action. Care to not leave the postoperative patient without analgesia until experiencing pain. Although pain scoring can be perceived as an extra and time-consuming task, it is a valuable tool to improve pain management. Regular training and consistent pain scoring enhance the ability to identify pain and contribute to greater patient care.

References

- *Adrian, D., Papich, M., Baynes, R., Murrel, J. and Lascelles, B. Chronic maladaptative pain in cats: A review of current and future drug treatment options, UK: The Veterinary Journal, 2017, 230; 52-61.*
- *Evangelista, M.,C., Watanabe, R., Leung, V.,S.,Y., Monteiro, B. P., O'Toole, E., Pang, D.S.J. & Steagall. Facial expressions of pain in cats: the development and validation of a Feline Grimace Scale. Scientific Reports volume 9, 2019, 19128; 1-11.*
- *Janasi, S. Analgesia for the emergency/critical care patient – part 1: Pain assessment. Eur J Comp Animal Practice, 2016, 26(3); 4-7.*
- *Severgnini, P., Pelosi, P., Contino, E., Serafinelli, E., Novario, R. and Chiranda, M. Accuracy of Critical Care Pain Observation Tool and Behavioral Pain Scale to assess pain in critically ill conscious and unconscious patients: prospective, observational study. Journal of Intensive Care, 2016, 4 (68); 1-8.*
- *Gruen, M., Lascelles B., Colleran, E., Johnson, J. and Marcellin-Little, D. 2022 AAHA Pain Management Guidelines for Dogs and Cats, J Am Anim Hosp Assoc, 2022; 58; 55–76.*
- *Self, I. and Grubb, T. BSAVA Guide to Pain Management in Small Animal Practice, chapter 2 Physiology of Pain, Gloucester, England; BSAVA, 2019, 13.*

Invasive blood pressure measurement: curve interpretation and trouble shooting

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Learning objectives

- Understand what blood pressure is
- Understand the components of what we are measuring.
- Learn how to measure
- How to interpret the information obtained
- Learn how to troubleshoot and evaluate abnormal waveforms.

Lecture summary

If you are working with invasive blood pressure measurement it's important that you know what you are measuring, how to monitor it and how you interpret the waveforms.
Blood pressure: Blood pressure (BP) is the pressure of circulating blood on the blood vessel wall. The most pressure is from the heart pumping the blood through the circulation system. "Blood pressure" usually refers to the pressure in large arteries of the systemic circulation. All the pressures are measured in millimeters of mercury (mmHg).

What do we measure with the invasive blood pressure

1. Systolic pressure: The pressure on the blood vessels walls during the heartbeat (contraction of the heart). Normal systolic pressure for dogs are between 90-140 mmHg, for cats 80-140 mmHg.
2. Diastolic pressure: The pressure on the blood vessel walls between two heartbeats (filling of the heart). Normal diastolic pressure for dogs are between 50-80 mmHg, for cats 55-75 mmHg.
3. Mean arterial pressure (MAP):Is the average blood pressure in the arteries (MAP = DAP+(SAP-DAP)/3. Normal MAP for dogs and cats is between 60-100 mmHg

Invasive blood pressure measurement

An arterial catheter must first be placed in an aseptic manner to minimize the risk of infections. Areas with a high risk of contamination should be avoided. For example: areas with damaged skin or pyoderma. The most commonly used artery is the dorsal pedal artery, however, the arterial catheter in the radial, coccygeal, femoral or auricular artery are also used. Some patients may require sedation or a local anaesthetic during placement. The following supplies are needed to perform direct blood pressure measurement.

1. Sterile pressure transducer set, with fast flush device
2. Medical tape
3. Isotonic crystalloids used for flush solution
4. Pressure bag
5. Monitor

When everything is installed and attached to the patient, you will next need to zero and level the systems. Transducer zeroing should be done at initial system setup or when systems components are removed or replaced. The transducer should be levelled to a consistent point, approximately at the level of the right atrium (RA). Your pressure bag containing the flush fluids is pressured to 300 mmHg. A square wave test, which checks if your system has an adequate dynamic response (the ability of your system to accurately display the shape and amplitude of the pulse pressure waveforms) is then performed. The system will have an optimal response when the frequency is as high as possible. One way to maximize the system's natural frequency is to keep the system as short as possible. Frequency refers to the fact that when stimulated, every structure naturally vibrates or moves at a characteristic frequency.
We can also see damping. Damping is the loss of pulse pressure energy between the catheter and the transducer. We see underdamping more commonly than overdamping Overdamping is characterized by a waveform with slurred upstrokes and downstrokes and loss of detail. With overdamping, you will see a falsely decreased pulse pressure with falsely low systolic pressure and falsely high diastolic pressure. With underdamping, you can see waveforms that have points and spikes and extra waves. With underdamping, you can see falsely high systolic pressures and falsely low diastolic pressures. Other tips that improve the waveform are described below.

Troubleshooting abnormal waveforms

1. Check patient clinical status. Are there any changes in patient status that can explain the change in waveform: mentation, heart rate, pulse quality, mucous membrane color, CRT and the extremity temperature. Check ECG for possible change in rate or rhythm.
2. Check your transducer system and make sure that the tubing used between patient and transducer is noncompliant tubing. See if you can shorten the tubing between the catheter and the transducer. Check if the tubing is not kinked or if air bubbles are in the fluid column and the pressure bag is inflated to at least 250 mmHg.
3. Relevelling and re-zeroing the transducer, if the patient position has changed.
4. Check patency of the arterial catheter: aspirate catheter and ensure that blood is easily obtained. Remove air bubbles and cloths from the line. Flush the catheter and ensure its flushed easily, do not force it.
5. Perform a fast flush test (square wave test) to check for system overdamping or underdamping.

The colloid controversy

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Learning objectives

- Attendees will leave understanding the difference between crystalloids and colloids.
- Attendees will learn about potential colloid complications.
- Attendees will learn about both synthetic and natural complications.
- Attendees will learn about the uses of colloids.

Lecture summary

There are three types of hydroxyethyl starches: hetastarch, pentastarch and tetrastarch. Generally the difference between the three is the degree of substitution (the number of substituted glucose molecules divided by the number of glucose molecules present) and the molecular weight (hetastarch has the highest molecular weight, tetrastarch the lowest). Routinely the term hetastarch and HES are used interchangeably, which is incorrect. However, despite the numerous types of HES available, they all perform basically the same job. Most common trade names include: Hespan® (a hetastarch), HAESteril® and Pentaspan® (both pentastarch) and Voluven®, Vetstarch® (both tetrastarchs).

Synthetic colloids have been shown in people to cause several adverse side effects including acute kidney injury, higher morbidity rates, prolonged hospitalization and coagulopathies. These studies have caused the use of synthetic colloids to drop dramatically in human medicine. With the lack of evidence based literature for veterinary patients, the human-based studies were brought over to the veterinary industry. This resulted in many facilities abandoning the use of synthetic colloids all together, including entire countries. Over the years there have been many studies showing that certain drugs react differently in animal patients than in human (theobromine, xylitol, ibuprofen, naproxen, etc) so it is reasonable to deduce that colloids do not react the same way in animal patients as they do in human patients. The question is "are we seeing the same side effects that human medicine is seeing then using HES products in our veterinary patients?"

One of the most well-documented side effects to using HES is its coagulopathy. HES has been shown to decrease factor VIII (an essential blood clotting factor), cause both functional and morphological changes to platelets and decrease both Von Willebrand's factor (vWF) and plasma clotting factors. It appears mainly dose dependent and at high doses (>22ml/kg in dogs) coagulation problems are more likely to occur. Once the product is discontinued the patient's values generally return to normal. The coagulopathy associated with HES is usually insignificant and only poses a problem in pets with prior coagulation issues or where surgery may be needed. Though not as common, anaphylactic reactions can occur (more commonly in the cat than the dog). Vomiting, fever and uritcaria (hives) have been reported. When administering to cats particularly, slow administration (over 15-30 minutes) is important.
The dose of most hydroxyethyl starches is very small. It is important to make sure that any hydroxyethyl starch is not overdosed as it can also lead to fluid overload quickly. The smaller the molecular weight (such as that of Vetstarch®) can likely be given at a higher rate than higher molecular weight HES products (Hespan®). Safe shock doses for most HES are 10-20 ml/kg in the dog (given either rapidly or over 15 minutes) and in the cat, 5-15 ml/kg given over 15 minutes. The same dose is currently used for the tetrastarch Vetstarch®. The constant rate infusion of most HES products is 1-2ml/kg/hr in the dog and cat (not to exceed 20ml/kg/hr in 24 hours in the dog or 10ml/kg/hr in a 24 hours in the cat).

All classes of synthetic colloids have been associated with acute kidney injury though HES is the most cited. HES-associated acute kidney injury is not fully understood. It is thought that HES is taken up in to the renal interstitial system causing damage. HES has been shown to cause a swelling of proximal renal tubular cells within a few hours of administration. Changes to the cells and tubules are reversible once the product is discontinued, but several human patients have had to undergo dialysis to promote reversal.

HES has been shown to cause allergic reactions including anaphylaxis in human patients. Tissue uptake of HES is both dose and time dependent. Pruritus in human patients has a delayed onset (typically 1–6 weeks postexposure) and it has been reported to last up to 24 months. There was some theory that lower molecular weight HES would cause less pruritus, but that claim appears unfounded.

With all the evidence in human literature supporting coagulopathies, acute kidney injury and allergic reactions the use of HES has been challenged in medicine. That said it must still be asked "are we seeing the same side effects in veterinary medicine" and "what evidence based medicine to we have to go on?"
Due to the small word count, please reach out to the author for the full lecture notes. VetTeamTRaining@Gmail.com

POCUS for haemodynamic assessment for nurses

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Learning objectives

- How to interpret normal pleural sliding and abnormal pleural artifacts
- How to use pleural and lung ultrasound to evaluate and monitor pleural effusion
- How to evaluate and monitor pericardial effusion
- Monitoring abdominal effusion

Lecture summary

Point-Of-Care Ultrasound (POCUS) has become an essential tool in the management of small animal emergency and critical care cases. POCUS assists the clinician in the decision-making process, allowing them to choose the most appropriate treatment solution to manage the acute disorder. On the human side, nurses with specific training have enabled POCUS to become a continuous monitoring tool in the ICU.[1] It is can be possible because nurses have the ability to perform POCUS, obtaining and interpreting images with accuracy, helping clinicians in different clinical scenarios.[2]
In veterinary medicine, nurses are beginning to learn POCUS and how to use it, and it will play an increasingly important role in the management and monitoring of several critical conditions. Unstable patients are always tricky. Trauma, shock, sepsis, congestive heart failure are only few examples of scenarios where a continuous monitoring approach can change the management of the patient. POCUS is not limited to a single organ or body cavity, and is used to asses multiple organs and systems, including the pleural spaces, lungs, left atrium size, a visual estimate of left ventricle contractility, and the presence of effusions (pericardial, pleural and abdominal). Scans used for hemodynamic evaluation includes a longitudinal approach of each hemothorax with a S-shaped approach[3], a right parasternal short axis view of the left ventricle, and an evaluation of the abdomen following a previously described technique[4] or simply placing the probe in a longitudinal orientation in the most dependent site of the abdomen from cranial to caudal. Basically, questions are asked during POCUS that generally require a binary answer of yes or no.

Is the sliding sign present? (normal sliding of the visceral pleura moving on the fixed pleura. It generates A-lines which are reverberation artifacts, parallel and equidistant horizontal lines from the pleural line, considered normal)
How many vertical artifacts are present for each hemithorax? More than 3? (vertical artifacts are laser-beam like artifacts arising from the pleural line, directed toward the end of the screen, moving synchronously with breathing, deleting A-lines. A thorax is considered normal if less than 3 vertical artifacts are present in each hemithorax)
Is pleural effusion present? (generally, it is visualized as an anechoic band within the pleural space. Depending on the underlying cause, it can be also a be hypoechoic or corpusculated with hyperechoic floccules)
Is pericardial effusion present? (anechoic band surrounding the heart within the pericardial sac, between the heart and the pericardium). If yes, is cardiac tamponade present? (diastolic collapse of right atrium and right ventricle)
Is the left ventricle contracting normally? (visual estimate of left ventricle contractility, looking at wall dyskinetic movements, reduced contractility or increased contractility)
Is the left ventricle filled or it is empty? (visual estimate)
Is peritoneal effusion present? Is the volume of the fluid increased? (abdominal sites evaluated are between liver lobes, at the poles of kidneys and around the urinary bladder. Depending on underlying cause, the fluid can be anechoic, hypoechoic, corpusculated or hyperechoic and should fill the spaces between abdominal organs and structures. To evaluate the evolution of the amount of fluid present, an abdominal fluid scoring system (AFS) can be used. It consists of assigning a score of 1 if the effusion is present, 0 if isn't present, in one of 4 abdominal sites, with a maximum score of 4. Patients with 1 or 2 can became 3 or 4 and it can mean they may need of a blood transfusion or less often a surgical laparotomy).

References

- *Tulleken AM et al. (2019): Ultranurse: teaching point-of-care ultrasound to intensive care nurses. Intensive Care Med 45(5):727-729*
- *Totenhofer R et al. (2021): POCUS use by registered nurses and nurse practitioners in clinical practice: an integrative review. Collegian 28(4)-456-463*
- *Armenise A et al. (2019): Vet-FAST ABCDE: a prospective observational study in 64 canine trauma patients. JSAP 60: 173-182*
- *Boysen SR and Lisciandro GR (2013): The use of ultrasound for dogs and cats in the emergency room: AFAST and TFAST. Vet Clin North Am Small An Pract 43(4):773-797*

Top reasons for manuscript rejection

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Learning objectives

- To understand the major issues why manuscripts are rejected for publication by journals
- To ensure authors effectively plan the composition of manuscripts and choose the best journal for their submission
- To develop an awareness of writing approaches that effectively conveys the intended message

Lecture summary

While not all aspects of research are perhaps "exciting," or "ground-breaking," being able to answer an unknown question or figure something out should be considered one of most exhilarating roles for clinician/scientist. However, as compelling or titillating your findings may be, as Kathy Barker wrote: "If you can't communicate your data, they don't exist." This is why it is ever so frustrating when a manuscript is rejected for publication. Although most manuscripts will eventually be published, it will certainly require a great deal of time and effort. So getting your manuscript published so that you can tell "your story" is an important skill that must be honed just as many of your clinical skills. And like many skills, the art of publishing takes time - this presentation and others like it should not be viewed as a "step-by-step recipe for publishing" - no such thing exists. So the path to sharing your findings and getting it published begins with the formulation of the research itself. Appropriate experiment design is absolutely crucial but again, beyond the scope of this presentation. The focus of this presentation is about ensuring your manuscript is in the best position to be considered and eventually published by avoiding the major pitfalls that result in manuscripts being rejected.

Unfocused research question
Perhaps the most important aspect of your research is what are you trying to answer? This should be clear and answerable. The question will drive and help you define your research. The data you collect, the population you select, how you analyse your results all centre on your research question. Do not get bogged down and confuse a research question with a hypothesis. These can be related but your research question should be a concise question and it should simply reflect what you are investigating. Keep your question simple as it will allow you to remain focused. In order maximise your impact, you must clearly demonstrate how you have answered your central question to the reader.

Unclear importance: In terms of capturing the attention of your audience, making it crystal clear how there is a significant knowledge gap in a subject area and emphasising the need to fill that knowledge gap is key. The reason to spend any time with such background information is to answer the "why bother" question. Because its "interesting" or "it has not been done before" are usually insufficient.

Study Execution: The second selling point to your audience will be how you propose to answer your research question. The methods should have detail and be clear on how the study was executed. Unclear, vague or incomplete methodology is perhaps one of the most common reasons for rejection or recommendation for major revision. Your audience needs to have a clear idea of the logic of your methods and follow your reasoning.

Unfocused Narrative: Once you have completed your study, you need to package your message (the narrative) to maximise its impact. Your manuscript should be well structured. You want to give details so that your research question is clear, your methods are appropriate, your results are clear and your conclusions are sound. Conclusions can be challenging as you never want to overstate but you don't want to end in the anti-climatic "further research is required…" Remember that the need to do further research was your opening justification – to end on exact same note borders on having wasted your audience's time. And most importantly, how have you answered your research question? This allows you to punctuate how you formulated a question, showed that it was an important question to answer, and how you answered the question.

Don't rush submitting your paper: In order to maximize the chances of getting published - don't rush the submission process - make sure everything is absolutely ready for submission. If you lack attention to detail or is sloppy - expect to have a hard time publishing. Spend the time required to ensure there are no glaring errors in your submission - double check you are following the journal's author instructions, and triple check your numbers! You don't want to lose credibility if you don't pay attention to details.

References

- *Davis M. Scientific papers and presentations. First edition. San Diego: Academic Press; 1997*
- *Day R, Gastel B. How to write and publish a scientific paper. Cambridge University Press; 2012*
- *Get published - Your how to guide - Wiley: https://www.wiley.com/network/researchers/preparing-your-article/get-published-your-how-to-guide*
- *How to get published - What distinguish a good manuscript from a bad one? - Elsevier: https://www.elsevier.com/_data/assets/pdf_file/0004/91165/Get-Published-Quick-Guide.pdf*
- *How to get published - SAGE: https://us.sagepub.com/en-us/nam/how-to-get-published*

Inside the shocked cell

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Learning objectives

- Describe the pathophysiology of the shocked cell
- Relate these abnormalities to shock diagnosis and treatment

Lecture summary

Discussion of shock physiology usually centres on the systemic physiologic derangements that lead to inadequate oxygen delivery and cellular hypoxia. The consequence of this is usually simply described as inadequate adenosine triphosphate (ATP) production. However, a full understanding of shock physiology requires appreciation of the myriad intracellular consequences of ATP depletion. This understanding will aid future shock research and the development of novel diagnostics and treatments, as well as a deeper understanding for the clinical sequelae that occur in a shocked patient. The major intracellular consequences of shock discussed herein are intracellular pH balance, ion flux, adenosine release, oxidative stress, and gene expression. Cells with adequate oxygen supply produce ATP primarily through oxidative phosphorylation in the mitochondria, a process that produces approximately 36 molecules of ATP for each molecule of glucose. In hypoxic conditions, glycolysis proceeds through to pyruvate, producing only two molecules of ATP. This process also leads to depletion of intracellular nicotinamide adenine dinucleotide (NAD^+), which is also usually regenerated during oxidative phosphorylation. Thus, pyruvate is converted to lactate to regenerate NAD^+. There is accumulation of lactate in the cell in higher concentrations than in the extracellular fluid. The production of lactate does not produce acid (H^+), a common misconception.

Whilst lactate production is not an acid-producing reaction, it is true that an intracellular acidosis occurs in the shocked cell. In normal conditions, the hydrolysis of ATP (an acid-producing reaction) is mostly in balance with the oxidative production of ATP (an acid-consuming reaction). In shock, the consumption of ATP is greatly in excess of its oxidative production, leading to net acid accumulation. As intracellular pH is tightly regulated, acid extrusion mechanisms are activated in this setting. One important family of passive cotransporters is the monocarboxylate transporter (MCT) family, such as MCT1. These extrude protons in a 1:1 ratio with monocarboxylates, organic anions with a single $-COO^-$ moiety. As lactate is the most common monocarboxylate in this setting, protons and lactate that have been produced in separate processes leave the cell together, leading to the common extracellular finding of 'lactic acidosis'. Energy-requiring processes are important to maintenance of normal ionic gradients across cell membranes. The core deficit in shock is failure of the sodium/potassium ($3Na^+/2K^+$-ATPase) pump. This leads to intracellular accumulation of sodium. Additional effects on voltage-gated calcium channels and sodium/calcium exchangers also leads to calcium accumulation. These changes lead to cell swelling and dysfunction, as well as maladaptive upregulation of some calcium-dependent enzyme systems.

In shock, ATP catabolism progresses past merely hydrolysis to adenosine diphosphate. Catabolism to the level of adenosine occurs. Adenosine exits cells via facilitated diffusion. This adenosine may act as a signaling molecule, leading to further maladaptive changes in adjacent tissues. It may also be further catabolized in the extracellular fluid, leading to it being unable to re-enter the cell. Thus, even when oxygen supply is restored, the cell may be left with inadequate adenosine stores to regenerate ATP.

Cellular hypoxia alters the function of the xanthine oxidoreductase system, the enzymes responsible for later steps in the catabolism of ATP beyond adenosine. In this setting, the xanthine oxidase form of the enzyme predominates. This enzyme requires oxygen, so cannot function in shock. Large amounts of the metabolite hypoxanthine accumulate, as catabolism of this requires xanthine oxidoreductase. However, when oxygen supply is returned, xanthine oxidase becomes able to function again, and rapidly catabolizes the oversupply of hypoxanthine. This leads to production of a large amount of reactive oxygen species, which cause further intracellular injury and impede recovery from shock.

Expression of several genes in shocked cells is facilitated by reduced breakdown of hypoxia-inducible factor-1 (HIF-1). Whilst some of the stimulated genes are adaptive in recovery from shock, others are vasodilatory and pro-inflammatory.

References

- *Boron, W.F., 2004. Regulation of intracellular pH. Advances in physiology education, 28(4), pp.160-179. Bruegger, D., Kemming, G.I., Jacob, M., Meisner, F.G., Wojtczyk, C.J., Packert, K.B., Keipert, P.E., Faithfull, N.S., Habler, O.P., Becker, B.F. and Rehm, M., 2007. Causes of metabolic acidosis in canine hemorrhagic shock: role of unmeasured ions. Critical Care, 11, pp.1-14.*
- *Dennis, S.C., Gevers, W. and Opie, L.H., 1991. Protons in ischemia: where do they come from; where do they go to?. Journal of molecular and cellular cardiology, 23(9), pp.1077-1086.*
- *Kehrer, J.P., 2000. The Haber–Weiss reaction and mechanisms of toxicity. Toxicology, 149(1), pp.43-50. Kumar, H. and Choi, D.K., 2015. Hypoxia inducible factor pathway and physiological adaptation: a cell survival pathway?. Mediators of inflammation, 2015.*
- *Lumb AB. Hypoxia. In: Lumb AB, editor. Nunn's Applied Respiratory Physiology. 7th ed. London: Churchill Livingstone; 2012, pp. 363-370.*
- *Lumb AB. Hyperoxia and oxygen toxicity. In: Lumb AB, editor. Nunn's Applied Respiratory Physiology. 7th ed. London: Churchill Livingstone; 2012, pp. 377-390.*
- *Rosenstein, P.G., Tennent-Brown, B.S. and Hughes, D., 2018. Clinical use of plasma lactate concentration. Part 1: Physiology, pathophysiology, and measurement. Journal of Veterinary Emergency and Critical Care, 28(2), pp.85-105.*
- *Textoris, J., Beaufils, N., Quintana, G., Ben Lassoued, A., Zieleskiewicz, L., Wiramus, S., Blasco, V., Lesavre, N., Martin, C., Gabert, J. and Leone, M., 2012. Hypoxia-inducible factor (HIF1a) gene expression in human shock states. Critical Care, 16, pp.1-6.*

Lactate: is it all hypoxia?

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Learning objectives

- Recall the principles of lactate production in health, as part of the pathway of normal cellular metabolism under aerobic conditions
- Understand the labile relationship between pyruvate and lactate levels, and the lactate shuttle that exists between organ systems
- Understand A, B1, B2 and B3 hyperlactataemia, and outline drugs, diseases and metabolic contributors to type B lactate generation
- Understand the Krogh cylinder model of oxygen pressure field theory, and the mean tissue oxygen tension established with recent techniques
- Critically consider type B hyperlactataemia in clinical patients where evidence of hypovolaemia/reduced oxygen tissue delivery is not present

Lecture summary

Introduction: Lactate is undoubtedly an interesting molecule, whether you believe it to be the physiologic equivalent of the bogeyman or a critically important metabolic intermediary. One cannot argue with the wealth of data pointing to improved outcomes with normal or normalisation of lactate in various disease processes in both veterinary and human medicine. What is perhaps less clear are the multifactorial contributions to lactate flux, inconsistencies in the data linking cellular hypoxia to lactate generation, and its many other known roles within mammalian physiology. From evidence that failure to elevate lactate in response to catecholamine stimulation is associated with worse outcome, to the improvement in mortality seen in experimental models when lactate is administered in those whose endogenous generation has been blocked, it is clear we have much to learn about the molecule we think we understand.

LactHATE or LactWAIT?: We were surely all taught that strenuous muscular activity causes lactic acid accumulation with the construct this occurs due to anaerobic metabolism and an oxygen deficit (Type A lactate). Glycolysis does not require oxygen which is not the same as stating that it occurs due to the absence of oxygen. Glycolysis occurs aerobically or anaerobically however the fate of glycolytic end products may differ with oxygen tension inside the cell; lactate formed for any reason other than cellular hypoxia is type B lactate. Lactate is the conjugate base of lactic acid (pKa 3.86), the latter being unable to exist at physiologic pH due to its pKa (Roberts *et al.,* 2018) thus we need to reconsider our understanding of lactic acidosis. Both lactate and pyruvate can be formed by glycolysis, the latter being a key intermediatory molecule linking glycolysis and oxidative phosphorylation within the mitochondria. The traditional view is that pyruvate is the major end product of glycolysis although this is being contested with some evidence that lactate is the primary end product instead (Rogatzki et al. 2015; Brooks 2018; Li et al. 2022). The relationship between lactate and pyruvate is bidirectional and facilitated by lactate dehydrogenase; anything affecting activity of LDH or enzymes responsible for metabolism of pyruvate will impact lactate and pyruvate levels within the cell, and the resultant efflux or metabolism thereof. The conversion of pyruvate to lactate consumes H^+: $2\ C_3H_3O_3^- + 2\ NADH + 2\ H^+ = 2\ C_3H_5O_3^- + 2\ NAD^+$. This reaction also regenerates NAD^+ allowing glycolysis to continue in the failure of oxidative phosphorylation (or when the latter may be a saturated metabolic pathway). Pyruvate is known to be a major substrate for the mitochondria but less well known is that lactate can be, given that mitochondria contain both LDH and MCTs, providing evidence of a mitochondrial lactate oxidation complex (Hashimoto *et al.* 2006). So, lactate may be a mitochondrial energy source, as well as being one of the most important precursors for gluconeogenesis (Brooks 2018; Brooks et al 2021) in addition to its roles, via lactate receptor GPR81 amongst others, in signaling numerous physiologic processes (Li *et al*., 2022).

Lactate is vital for life: Lactate is not an offcast byproduct of a metabolic pathway. Instead, it is a well utilized metabolic fuel for many tissues, including brain, heart, liver, kidneys, muscle cells and spermatozoa; evidence even points to lactate being a preferential fuel over glucose in some tissues. Lactate is also a pivotal metabolic intermediary with transporters and a specific receptor with regulatory roles in many processes not related to energy production (such as lactylation which leads to transcriptional regulation), regulation of inflammation, neuronal protection and fatty acid metabolism to name a few (Li *et al.*, 2022). Myriad lactate shuttles both between cellular organelles, tissues and even organisms have been documented and the complex interplay of all these known roles surely elevates this maligned molecule above simply metabolic waste from anaerobic tissue.

Lactate as a treatment: Aside from our use of lactated fluids, in humans half molar sodium lactate improves cardiac index in patients with CHF and in those following coronary artery bypass grafting (Leverve et al. 2008; Nalos *et al.* 2015), as well as having greater reduction in ICP, with greater number of responders, and for a greater length of time than an osmotically equivalent dose of mannitol after TBI (Ichai *et al.* 2009). In an experimental endotoxemic shock model, animals with blockade of skeletal muscle lactate production (via combined b2 blockade and activation of pyruvate dehydrogenase activity) had increased mortality; this was ameliorated with molar lactate addition (Levy *et al*. 2007). Lactate - It is definitively **not** all about hypoxia.

References

- *Brooks, G.A., The Science and Translation of Lactate Shuttle Theory, Cell Metabolism, 2018, 27(4);757-785*
- *Brooks, G.A., Arevalo, J.A., Osmond, A.D., et al., Lactate in contemporary biology: a phoenix risen, The Journal of Physiology, 2022, 600(5);1229-1251*
- *Hashimoto, T., Hussien, R., Brooks, G.A., Co-localization of MCT1, CD147, and LDH in mitochondrial inner membrane of L6 muscle cells: evidence of a mitochondrial lactate oxidation complex, American Journal of Physiology Endocrinology and Metabolism, 2008, 290;E1237–E1244.*
- *Rogatzki, M.J., Ferguson, B.S., Goodwin, M.L., Gladden, L.B., Lactate is always the end product of glycolysis, Frontiers in Neuroscience, 2015, 9:22 Li, X., Yang, Y., Zhang, B., et al., Lactate metabolism in human health and disease, Signal Transduction and Targeted Therapy, 2022, 7(1):305*
- *Levy, B., Mansart, A., Montemont, C., et al., Myocardial lactate deprivation is associated with decreased cardiovascular performance, decreased myocardial energetics, and early death in endotoxic shock, Intensive Care Medicine, 2007, 33(3);495-502*
- *Ichai, C., Armando, G., Orban, J.C., et al., Sodium lactate versus mannitol in the treatment of intracranial hypertensive episodes in severe traumatic brain-injured patients, Intensive Care Medicine, 2008, 35(3);471-479*
- *Nalos, M., Leverve, X.M., Huang, S.J., et al., Half-molar sodium lactate infusion improves cardiac performance in acute heart failure: a pilot randomised controlled clinical trial, Critical Care, 2014, 18(2);1-9*
- *Leverve, X.M., Boon, C., Hakim., T., et al., Half-molar sodium-lactate solution has a beneficial effect in patients after coronary artery bypass grafting., Intensive Care Medicine, 2008, 34(10);1796-1803*

The "business end" of capillaries and cellular energy production - assessment of microcirculation, tissue perfusion and oxygen consumption

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Learning objectives

- Identify challenges with diagnosing "cryptic shock".
- Appraise current methods for evaluating the microcirculation and cellular energy metabolism.
- Classify methods based on local or global assessment

Lecture summary

Introduction: Aerobic cells require oxygen and nutrients in order to survive. Single cell organisms live in aqueous environments where the supply of oxygen and nutrients is "one cell membrane away" and waste can be deposited outside of this cell membrane. The cells of multicellular organisms, however, rely on complex systems oxygen and nutrients and remove waste products. The delivery of oxygen requires a network of bigger (arteries, arterioles or macrocirculation) and then smaller (capillaries or microcirculation) vessels reaching the far end of tissues. Undoubtably, if the supply chain is broken at the level of large vessels due to impairment or obstruction, the small vessels being the continuation of the large vessels will have nothing to deliver to the tissues and their cells will die. However, small vessels can have issues delivering oxygen and nutrients despite adequate work of large vessels. This is conceptualized under the umbrella of "cryptic" shock. To further complicate things, cells suffering from undernourishment do produce cellular energy adequately (the reader is referred to Dr Corrin Boyd's conference paper in the same proceedings (EVECCS congress 2023) "Inside the shocked cell" for more details). This cellular dysfunction also contributes the phenomenon known as "cryptic" shock. Cryptic shock has been associated with development of multi-organ dysfunction in critical patients. Is "cryptic shock" that cryptic, though, in the 21st century? Below summaries with some proof to the contrary.

Local monitoring

Microcirculatory assessment

Mucous membrane color and capillary refill time (CRT) are an essential part of the physical examination. However, in veterinary medicine, they have been popularized as a perfusion parameter, reflecting peripheral vasoconstriction or vasodilation. Empirically, "grey" or "muddy" mucous membranes are considered a sign of decompensated shock. However, veterinary evidence-based association of changes in mucous membrane color with changes in perfusion (e.g., shock) is scarce. "Skin mottling" is a term used to describe poor perfusion in people and might be the equivalent to mucous membrane color change in veterinary patients. It is a result of heterogenous small vessel vasoconstriction secondary to hypoperfusion. Higher pre-hospital skin mottling scores are associated with mortality in septic patients. Capillary refill time (CRT) refers to the time required for return of blood flow (and therefore color) to capillaries after compression sufficient to blanch the area. Popular since WWII, CRT is considered an indicator of peripheral perfusion. It has been associated with increased mortality in septic adults and children. However, in both human and veterinary patients, CRT lacks standardization in threshold values (or reference intervals), sites for measurement, compression time and firmness and necessity for averaging repeated measurements. Therefore, its validity and reliability remain controversial. In veterinary medicine, review articles and book chapters discuss CRT at length, nevertheless only very few studies have evaluated the utility of CRT, with one recent study from Chalifoux et al (JVECC 2019) evaluating the relation of standardized CRT to clinical parameters in hospitalized dogs.

Peripheral perfusion index is a ratio between the pulsatile and non-pulsatile component of absorbed light and calculated by specific pulse oximeters. Given that only arterial vessels are affected by vasoconstriction or dilation, it could be considered as a non-invasive measure of peripheral perfusion. In states of hypoperfusion, constriction of arterioles decreases PPI. Studies have shown the feasibility of measuring the PPI in people with shock. However, there is high inter-individual variability limiting the tool.

Tissue oxygen saturation can be measured via near-infrared spectroscopy. Similarly, to an oximeter, a difference in light absorption between oxy- and deoxyhemoglobin is detected in small vessels and can be measured transcutaneously. There is controversy surrounding the utility of the tool in people from studies in trauma and septic patients. Experimental studies in anesthetized dogs have determined a good correlation between StO_2 and other indices of tissue oxygen delivery. Clinical studies in small groups of dogs with hemorrhage and naturally occurring shock, have determined the feasibility of the technique, and have found the StO2 correlates with other shock parameters, and the severity of illness. The reader is referred to a review by Salcedo et al (JVECC 2016) for further information of the application of non-invasive tissue oxygen monitoring.

Laser Doppler flowmetry utilizes the Doppler shift of laser infrared lighting from moving RBCs to measure tissue perfusion. Laser Doppler flowmetry has been used to determine viability of skin flaps and transplants. Animal hemorrhagic shock and sepsis models as well as small-scale clinical studies have shown that changes in laser Doppler flowmetry flux can be an early indicator for changes in perfusion and is associated with changes in arterial blood pressure. The method has limitations including the necessity to assess trends, rather than absolute value. Laser Doppler flowmetry has been successful in detecting variation in stomach blood flow in dogs with gastric dilation-volvulus and has been validated to use in the ear pinnae in dogs.

Side stream dark field videomicroscopy for real-time visualization of capillaries has been the subject of extensive research in human medicine, with some experimental and clinical studies in veterinary medicine. The device emits light in the hemoglobin absorbance wavelengths for image acquisition. Perfused capillaries appear dark on a bright background. The methodology has come a long way from operators having to manually evaluate images, to the availability of custom software that includes image stabilizers that improve the precision of data acquisition, and automatically calculates the parameter measurements. Furthermore, novel software (Glycocheck™) allows estimation of the endothelial glycocalyx thickness using the same technology. Contrast-enhanced ultrasonography is a novel ultrasound modality allowing assessment of microcirculation of various organs after intra-venous injection of a specific contrast agent consisting of gas-filled microbubbles, enabling depiction of vessels as small as 100 µm. Accurate quantification of perfusion remains challenging, however studies in people are showing promising results.

Interstitial assessment
Microdialysis allows monitoring of various metabolites in the interstitial space. Small soluble molecules equilibrate across the membrane and the perfused fluid can be collected and analyzed. The methodology has been successfully used in animal models and in clinical studies in people to assess tissue metabolism. Microdialysis has been used to detect hormones and cytokines and study pharmacokinetics/dynamics and bioavailability of drugs.

Intracellular assessment
Mitochondrial Redox state evaluates nicotinamide adenine dinucleotide (NAD) and cytochrome C oxidase (COX) concentrations in the mitochondria. Lack of intracellular oxygen or other blockage down the electron transport chain will change unfavorably the reduced/oxidized ratios of both molecules and measurements of those can be made with fluorometry (for NAD) and near-infrared spectrometry (for COX). Both methodologies are at present mostly reserved to the experimental setting, with studies attempting to overcome current challenges with validation, calibration, and interpretation.

General monitoring
Lactate is an established biomarker of hypoperfusion and reduced tissue oxygen consumption. It can be the result of decreased oxygen delivery to the tissues or reflect the inability of tissues to use oxygen. Lactate concentrations are also affected by reduced clearance, drug administration, and the type of disease process itself, which should be factored in result interpretation. The reader is referred to the conference paper in the same proceedings (EVECCS congress 2023) from Tom Greensmith "Lactate - is it still hypoxia?" for more details. Venous oxygen saturation (SvO_2) is widely used as a marker for hypoperfusion and increased tissue oxygen extraction, including in the early goal-directed therapy for sepsis in people. As oxygen delivery decreases, tissues will extract more oxygen, which will result in a decreased SvO_2. While most of the literature focuses on decreases in SvO_2 as a result of increased oxygen extraction, studies in people have demonstrated that increased SvO_2 is associated with higher mortality as a result of the inability of the tissue to extract oxygen (cellular or mitochondrial dysfunction). The reader is referred to a clinical practice review by Walton and Hansen (JVECC 2018) for further information on SvO_2 in critical illness.Magnetic resonance spectroscopy (MRS) uses radiolabelled molecules to determine metabolite concentrations and quantify enzyme kinetics in tissues. In comparison with magnetic resonance imaging (MRI), instead of images, MRS detects chemical composition of the scanned tissue. Magnetic resonance spectrometry can be combined with MRI. The technique has been successfully used in people with congestive heart failure and traumatic brain injury to measure concentrations of ATP and other molecules relevant to tissue metabolism.

Conclusion

Excelling at restoring macrohemodynamics has taken medicine about a century. The beginning of the 21st century is marked by an understanding that cells rely on complex "logistics" for oxygen and nutrient delivery to their "doorstep". Advances in technology allow us to add to "the toolbox" and facilitate the diagnosis and management such derangements. Whether the novel tools presented here have a future is yet to unfold, as some have significant drawbacks. We can only aspire it takes us less than a century to excel at treating "the business end of capillaries".

References

- *Brady CA, Otto CM, Van Winkle TJ, et al. Severe sepsis in cats: 29 cases (1986-1998). J Am Vet Med Assoc 2000;217:531-535.*
- *Brealey D, Brand M, Hargreaves I, et al. Association between mitochondrial dysfunction and severity and outcome of septic shock. Lancet 2002;360:219-223.*
- *Bridges E. CE: Assessing Patients During Septic Shock Resuscitation. Am J Nurs 2017;117:34-40.*
- *Chalifoux NV, Spielvogel CF, Stefanovski D, et al. Standardized capillary refill time and relation to clinical parameters in hospitalized dogs. J Vet Emerg Crit Care (San Antonio) 2021;31:585-594.*
- *Clevert DA, D'Anastasi M, Jung EM. Contrast-enhanced ultrasound and microcirculation: efficiency through dynamics--current developments. Clin Hemorheol Microcirc 2013;53:171-186.*
- *da Costa JC, Coe JB, Blois SL, et al. Twenty-five components of a baseline, best-practice companion animal physical exam established by a panel of experts. J Am Vet Med Assoc 2022;260:923-930.*
- *de Moura EB, Amorim FF, da Cruz Santana AN, et al. Skin mottling score as a predictor of 28-day mortality in patients with septic shock. Intensive Care Med 2016;42:479-480.*
- *Dechant JE, Nieto JE, Le Jeune SS. Hemoperitoneum in horses: 67 cases (1989-2004). J Am Vet Med Assoc 2006;229:253-258.*
- *Ejertsen T, Porsborg PA. Toxic shock syndrome related to simultaneous Staphylococcus aureus epiglottic abscess and group A streptococcal pharyngitis with bacteremia. APMIS 1994;102:956-959.*
- *Ekbal NJ, Dyson A, Black C, et al. Monitoring tissue perfusion, oxygenation, and metabolism in critically ill patients. Chest 2013;143:1799-1808.*
- *Epstein CD, Haghenbeck KT. Bedside assessment of tissue oxygen saturation monitoring in critically ill adults: an integrative review of the literature. Crit Care Res Pract 2014;2014:709683.*
- *Garrard HF. Use of polarized light in color photography of mucous membranes. Arch Derm Syphilol 1949;60:425.*
- *Harrois A, Grillot N, Figueiredo S, et al. Acute kidney injury is associated with a decrease in cortical renal perfusion during septic shock. Crit Care 2018;22:161.*
- *Hiwatashi K, Doi K, Mizuno R, et al. Examiner's finger-mounted near-infrared spectroscopy is feasible to analyze cerebral and skeletal muscle oxygenation in conscious Chihuahuas. J Biomed Opt 2017;22:26006.*
- *Holowaychuk MK, Fujita H, Bersenas AM. Evaluation of a transcutaneous blood gas monitoring system in critically ill dogs. J Vet Emerg Crit Care (San Antonio) 2014;24:545-553.*
- *Ide K, Tokunaga M, Hiratsuka S, et al. Validation of laser doppler flowmetry to measure dermal blood flow of the pinnae in dogs with pinnal alopecia. Vet Dermatol 2023;34:70-76.*
- *Jacob M, Chappell D, Becker BF. Regulation of blood flow and volume exchange across the microcirculation. Crit Care 2016;20:319.*
- *Jouffroy R, Bloch-Laine E, Maignan M, et al. Contribution of Capillary Refilling Time and Skin Mottling Score to Predict ICU Admission of Patients with Septic or haemorrhagic Shock Admitted to the Emergency Department: A TRCMARBSAU Study. Turk J Anaesthesiol Reanim 2019;47:492-495.*
- *Jouffroy R, Saade A, Tourtier JP, et al. Skin mottling score and capillary refill time to assess mortality of septic shock since pre-hospital setting. Am J Emerg Med 2019;37:664-671.*
- *Kaspar M, Partovi S, Aschwanden M, et al. Assessment of microcirculation by contrast-enhanced ultrasound: a new approach in vascular medicine. Swiss Med Wkly 2015;145:w14047.*
- *Koenig G, Tantawi M, Wessner CE, et al. Evaluation of Suspected Small Bowel Ischemia Using Contrast-Enhanced Ultrasound with Computed Tomography Fusion. J Emerg Trauma Shock 2022;15:60-62.*
- *Li T, Ji X, Liu J, et al. Ulinastatin Improves Renal Microcirculation by Protecting Endothelial Cells and Inhibiting Autophagy in a Septic Rat Model. Kidney Blood Press Res 2022;47:256-269.*
- *Liu N, Zhang Z, Hong Y, et al. Protocol for a prospective observational study on the association of variables obtained by contrast-enhanced ultrasonography and sepsis-associated acute kidney injury. BMJ Open 2019;9:e023981.*
- *Manji RA, Wood KE, Kumar A. The history and evolution of circulatory shock. Crit Care Clin 2009;25:1-29, vii.*
- *Marino S, Ciurleo R, Bramanti P, et al. 1H-MR spectroscopy in traumatic brain injury. Neurocrit Care 2011;14:127-133.*
- *Mayevsky A, Walden R, Pewzner E, et al. Mitochondrial function and tissue vitality: bench-to-bedside real-time optical monitoring system. J Biomed Opt 2011;16:067004.*
- *McCown JL, Lechner ES, Cooke KL. Suspected albuterol toxicosis in a dog. J Am Vet Med Assoc 2008;232:1168-1171.*
- *Millham FH. A brief history of shock. Surgery 2010;148:1026-1037.*
- *Monnet E, Pelsue D, MacPhail C. Evaluation of laser Doppler flowmetry for measurement of capillary blood flow in the stomach wall of dogs during gastric dilatation-volvulus. Vet Surg 2006;35:198-205.*
- *Obonyo NG, Fanning JP, Ng AS, et al. Effects of volume resuscitation on the microcirculation in animal models of lipopolysaccharide sepsis: a systematic review. Intensive Care Med Exp 2016;4:38.*
- *Pavlisko ND, Killos M, Henao-Guerrero N, et al. Evaluation of tissue hemoglobin saturation (StO2) using near-infrared spectroscopy during hypoxemia and hyperoxemia in Beagle dogs. Vet Anaesth Analg 2016;43:18-26.*
- *Rajan V, Varghese B, van Leeuwen TG, et al. Review of methodological developments in laser Doppler flowmetry. Lasers Med Sci 2009;24:269-283.*
- *Rimer D, Lerman O, Klainbart S, et al. Lethal acute hemorrhage from an aortoesophageal fistula following endoscopy-assisted esophageal foreign body removal in a dog. J Vet Emerg Crit Care (San Antonio) 2020;30:587-591.*
- *Roberts BK, Aronsohn MG, Moses BL, et al. Bufo marinus intoxication in dogs: 94 cases (1997-1998). J Am Vet Med Assoc 2000;216:1941-1944.*
- *Ruan Z, Li R, Dong W, et al. Laser speckle contrast imaging to monitor microcirculation: An effective method to predict outcome in patients with sepsis and septic shock. Front Bioeng Biotechnol 2022;10:1067739.*
- *Salcedo MC, Tart K, Hall K. A systematic review of human and veterinary applications of noninvasive tissue oxygen monitoring. J Vet Emerg Crit Care (San Antonio) 2016;26:323-332.*
- *Schonberger ZG, Huang SJY, Thakkar RN, et al. Assessments of Perfusion, Blood Flow, and Vascular Structure in Ambulatory Subjects: Guidance for Translational Research Scientists. J Vasc Res 2023:1-57.*
- *Shuster R, Traub-Dargatz J, Baxter G. Survey of diplomates of the American College of Veterinary Internal Medicine and the American College of Veterinary Surgeons regarding clinical aspects and treatment of endotoxemia in horses. J Am Vet Med Assoc 1997;210:87-92.*
- *Sivakorn C, Schultz MJ, Dondorp AM. How to monitor cardiovascular function in critical illness in resource-limited settings. Curr Opin Crit Care 2021;27:274-281.*
- *Sturm T, Leiblein J, Clauss C, et al. Bedside determination of microcirculatory oxygen delivery and uptake: a prospective observational clinical study for proof of principle. Sci Rep 2021;11:24516.*
- *Testuz J, Howard J, Pozzi A, et al. Evaluation of surface blood flow in intact and ruptured canine cruciate ligaments using laser Doppler flowmetry. Vet Comp Orthop Traumatol 2016;29:361-368.*
- *Wang J, Shi M, Huang L, et al. Addition of terlipressin to norepinephrine in septic shock and effect of renal perfusion: a pilot study. Ren Fail 2022;44:1207-1215.*
- *Wang S, Zhao P, Zhang Y, et al. The Therapeutic Effects of Curcumin in Early Septic Acute Kidney Injury: An Experimental Study. Drug Des Devel Ther 2021;15:4243-4255.*
- *Wang XY, Pang YP, Jiang T, et al. Value of early diagnosis of sepsis complicated with acute kidney injury by renal contrast-enhanced ultrasound. World J Clin Cases 2019;7:3934-3944.*
- *Watchorn J, Huang D, Bramham K, et al. Decreased renal cortical perfusion, independent of changes in renal blood flow and sublingual microcirculatory impairment, is associated with the severity of acute kidney injury in patients with septic shock. Crit Care 2022;26:261.*
- *Goodnight ME, Cooper ES, Butler AL. Assessment of microcirculatory perfusion in healthy anesthetized cats undergoing ovariohysterectomy using sidestream dark field microscopy. J Vet Emerg Crit Care (San Antonio) 2015;25:349-357.*
- *Silverstein DC, Pruett-Saratan A, 2nd, Drobatz KJ. Measurements of microvascular perfusion in healthy anesthetized dogs using orthogonal polarization spectral imaging. J Vet Emerg Crit Care (San Antonio) 2009;19:579-587.*
- *Watchorn J, Huang DY, Joslin J, et al. Critically Ill COVID-19 Patients With Acute Kidney Injury Have Reduced Renal Blood Flow and Perfusion Despite Preserved Cardiac Function: A Case-Control Study Using Contrast-Enhanced Ultrasound. Shock 2021;55:479-487.*
- *Yozova ID, Londono LA, Millar KK, et al. Rapid Patient-Side Evaluation of Endothelial Glycocalyx Thickness in Healthy Sedated Cats Using GlycoCheck(R) Software. Front Vet Sci 2021;8:727063*

Effect of hemodialysis and hemoperfusion using cytosorb adsorber on bilirubin in dogs

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Introduction

Hemodialysis has been shown to reduce bilirubin in dogs in a few case reports. Cyosorb hemoperfusion is reported to reduce bilirubin in humans. This study aimed to evaluate the reduction of high serum bilirubin in dogs with hemodialysis and hemoperfusion.

Methods

Hemodialysis and hemoperfusion sessions of dogs with high bilirubin levels (> 50 µmol/l) over a 1.5 years period have been evaluated prospectively. Hemodialysis was performed with a Fresenius 4008 machine with pediatric tubing and a dialyzer adapted to the patient's size. Hemoperfusion was performed in dogs with severe clinical signs of inflammation, using the Fresenius 4008 machine or the vet smart machine. Dogs with multiple diseases were included. Serum levels of bilirubin were analyzed before and after the treatment. Data were analyzed using a paired t-test and Wilcoxon-matched pairs signed rank test. P values < 0.05 were considered significant.

Results

During the study period, 5 dogs during 9 hemodialysis sessions and 4 dogs during 5 hemoperfusion sessions were included. Bilirubin levels decreased from 381 µmol/l (78 – 606 µmol/l) to 285 µmol/l (55 – 545 µmol/l) in the dialysis group and from 548 µmol/l (84 – 1191 µmol/l) to 265 µmol/l (56 – 386 µmol/l) in the hemoperfusion group. The bilirubin reduction ratio was higher in the hemoperfusion group (52%; 28 – 68 %) compared to the hemodialysis group (12%; -10 – 41%; p = 0.0047).

Conclusion

Hemoperfusion using the Cytosorb adsorber, but not hemodialysis, is a promising tool to reduce serum bilirubin in dogs with high bilirubin values.

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Ionized magnesium in critically ill dogs and cats: a retrospective study

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Introduction

A retrospective study to investigate the incidence of dysmagnesemia in critically ill patients.

Methods

Medical records of dogs and cats admitted to the Veterinary Teaching Hospital of the University of Pisa, Italy, from April 2020 to November 2021, were analyzed. Inclusion criteria were at least a venous blood gas analysis at arrival (Stat Profile Prime Plus Critical Care Analyzer®, Nova Biomedical) to evaluate the ionized magnesium concentration. Normal values were considered 0.5-1 mmol/L for both species, according to machine range. Species, breed, sex, age, weight, reason for hospitalization, presence of comorbidities, and outcome were also recorded.
For statistical purposes, disease categories were created as follows: cardiovascular, respiratory, gastroenteric, urinary, reproductive, endocrine, hematopoietic, immune-mediated, neurological, neoplastic, infectious, and systemic diseases.
Data were analyzed for distribution with a D'Agostino Pearson test; Mann-Whitney or Kruskal-Wallis were used to analyze results; Fisher's test and chi-square test were used to analyze contingency. The chi-square test was used to compare outcomes between dysmagnesemic and normomagnesemic patients. P values <0.05 were considered significant.

Results

Four hundred thirty (430) dogs and two hundred (200) cats were enrolled. The mean value of magnesium in dogs was 0.54±0.12 mmol/L; 153 dogs resulted hypomagnesemic (35.5%), 5 hypermagnesemic (1.1%), and 272 normomagnesemic (62.2%). No differences (p=0.9) were found between survivors and non-survivors for magnesium concentration, while males showed a significantly higher (p= 0.0006) incidence of hypomagnesemia than females. Pathologies with higher incidence of hypomagnesemia were neurological (51%), neoplastic (50%), and endocrinological (42%). Na^+, Ca^{++} and K^+ were significantly lower in hypomagnesemic dogs. In cats, the mean value of magnesium was 0.68±0.19 mmol/L; 12 cats were hypomagnesemic (6%), 17 hypermagnesemic (8.5%), and 171 normomagnesemic (85.5%). Hypermagnesemic cats showed a mortality rate 0.8 times higher (p=0.004) than normo and hypomagnesemic cats. Pathologies with higher incidence of hypermagnesemia were urological (14.5%), cardiovascular (10%), and respiratory (8%). Na^+, Ca^{++} and K^+ were significantly lower in hypomagnesemic cats; BUN, creatinine and lactate were significantly higher in hypermagnesemic cats.

Conclusion

Dogs presented a high incidence of hypomagnesemia, not associated with increased mortality, whereas in cats, despite hypermagnesemia having a low incidence (8.5%), it was associated with increased mortality.

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Body temperature predicts outcome in cats presenting to an emergency service: a retrospective study on 1539 cases (january 2018-december 2021)

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Introduction
To evaluate the association between body temperature and outcome in cats presenting to the emergency room (ER).

Methods
An electronic database of medical records conducted in the study period on cats presented to the institution ER was primarily searched for results of body temperature (BT) and outcome. Patient signalment, additional clinical data, presenting complaint or final diagnosis were recorded when available. BT was categorized as: normothermia (N: 37.8-39.7 °C); hyperthermia (H:≥ 39.8°C); hypothermia (L:< 37.8°C). L was further classified as: mild (MIL: 36.8-37.7 °C); moderate (MOL: 35.6-36.7°C); severe (SL: 33.1-35.5°C); critical (CL:≤33°C). Cats were classified in four disease categories: SIRS (S), based on the reported feline criteria; urinary system (U), cardiovascular (CV), and miscellanea (M). Outcome after ER visit was categorized as death, euthanasia, hospital admission, and discharge. Conventional statistical analyses were used.

Results
1539 cats were included with the following distribution based on BT: H=89 (5.8%), N=916 (59.5%); L=534 (34.7%), further categorized as MIL (n=266, 17.3%); MOL (n=112, 7.3%); SL (n=99, 6.4%); CL (n=57, 3.7%). Disease categories included: S (n=459, 29.8%); U (n=211, 13.7%); CV (n=52, 3.4%); M (n=817, 53.1%). Outcome categories after ER were: discharge (n=468, 30.4%); hospital admission (n=978, 63.5%); death or euthanasia (n=93, 6%). Frequency of death was significantly higher in cats with L (14%) compared to one with N (1.6%) or H (3.4%) (P<0.0001), and increased proportionally in L subgroups: MIL (5.3%); MOL (13.4%); SL (26.3%); CL (35.1%) (P<0.0001).
Reduction of temperature (OR 0.60, CI 0.54-0.65; P<0.0001), systolic arterial pressure (SAP) (OR 0.97, CI 0.96-0.98; P<0.0001), and heart rate (OR 0.98, CI 0.97-0.99; P<0.0001), older age(OR 1.11, CI 1.06-1.17; P<0.0001), and a diagnosis of S (OR 3.24, CI 2.12-4.95; P<0.0001), were independently associated with higher risk of death. BT, SAP, and older age were the only variables retained in the multivariable logistic regression analysis (P<0.0001).

Conclusion
Hypothermia is common and significantly associated with a diagnosis of SIRS and a worse outcome in cats referred to an ER. Reduced SAP and older age might contribute to poor prognosis.

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Effects of high flow oxygen therapy on oxygenation in dogs undergoing diagnostic bronchoscopy

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Introduction

Hypoxemia is a common complication during bronchoscopy and bronchoalveolar lavage (BAL). High Flow Oxygen Therapy (HFOT) has been used to improve oxygenation and prevent periods of hypoxemia in people undergoing bronchoscopy.

Objective

The main objective of this study was to evaluate the effect of HFOT on oxygenation in dogs undergoing diagnostic bronchoscopy in comparison to a traditional oxygen supplementation method (TOT). Secondary objective was to assess the occurrence of complications related to HFOT.

Methods

Prospective randomized trial. Dogs presented for diagnostic bronchoscopy were randomly assigned to receive either HFOT or TOT using nasal cannulas during the bronchoscopic procedure. Oxygenation was evaluated using P_aO_2. Arterial blood gas analyses were obtained for each patient at seven predetermined time points: at baseline (t0), after preoxygenation (t1), after induction of anesthesia (t2), before and immediately after BAL sampling (t3 and t4), at the end of the procedure (t5), and one hour after the end of bronchoscopy (t6). Thoracic radiographs were performed for each patient before and immediately after bronchoscopy to assess the occurrence of air leak syndrome or aerophagia.

Results

20 privately owned dogs presented for diagnostic bronchoscopy were included in the study (HFOT group: n = 10, TOT group: n = 10). There were no significant differences between both groups regarding patient characteristics or physiological baseline parameters. 5 dogs in each group showed hypoxemia (P_aO_2 < 80 mmHg) at baseline with 1/5 in each group having P_aO_2 < 60 mmHg. HFOT improved oxygenation throughout the procedure, with significant values obtained after preoxygenation (P = 0.0007) and at the end of the procedure (P = 0.0131). Additionally, mean individual P_aO_2 values (t1–t5) showed significantly higher P_aO_2 in the HFOT group (P = 0.0114), and only 1/10 dogs in the HFOT group experienced hypoxemia during bronchoscopy compared to 5/10 dogs in the TOT group. There were no serious adverse events related to HFOT, although several dogs in both groups experienced aerophagia to various degrees. However, none of the dogs required medical intervention.

Conclusion

HFOT can improve oxygenation and prevent episodes of hypoxemia in dogs undergoing bronchoscopy compared to traditional oxygen supplementation methods.

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The ability of oxygen reserve index to detect mild hyperoxemia in mechanically ventilated dogs: a preliminary study

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Introduction

Mild hyperoxemia in humans could be detected with the oxygen reserve index (ORi) for arterial partial pressure of oxygen (PaO_2) between 100 and 200 mmHg. This study evaluated the correlation of ORi with PaO_2 and its ability to detect mild hyperoxemia in dogs.

Methods

This prospective observational study enrolled 23 adult anesthetized dogs undergoing elective procedures. The animals were ventilated under pressure-control ventilation with a peak inspiratory pressure of 10-12 cmH_2O and a respiratory rate adjusted to maintain normocapnia. Simultaneous measurements of ORi and PaO_2 were collected, with a multi-wavelength pulse co-oximeter with a probe applied to the dog's tongue, and a blood gas analyzer, respectively. Pearson correlation coefficient (*r*) was calculated between simultaneous measurements of ORi and PaO_2. Youden index was used to identify the ORi cut-off values predicting PaO_2 hyperoxia thresholds ≥110, ≥120, ≥130, ≥140 and ≥150 mmHg with the highest sensitivity and specificity. Diagnostic performances of ORi to detect the same hyperoxia thresholds were estimated using the area under the receiver operating characteristic curve (AUROC). The effects of perfusion index (PI), body temperature, arterial blood pH, partial pressure of carbon dioxide (pCO_2), lactatemia, and hemoglobin on AUROC were evaluated.

Results

A total of 56 paired measurements of ORi and PaO_2 were collected. Mean PaO_2 value was 148.8±32.48mmHg and mean hemoglobin was 12.58±1.84 g/dl. A moderate positive correlation (r=0.53, $p<0.001$) between ORi and PaO_2 was found. The ORi cut-off value with the highest sensitivity (91%) was 0.3, indicating PaO_2 ≥120 mmHg, with 70% specificity and AUROC=0.84 (95%CI=0.68-1.01). The ORI value cut-off with the highest specificity (86%) was 0.6, indicating PaO_2 ≥140 mmHg, with 60% sensitivity and AUROC=0.79 (95%CI=0.67-0.91). The AUROCs of ORi to detect hyperoxia thresholds tended to increase with decreasing blood pH, PI and lactatemia, and with increasing pCO_2 and temperature, while hemoglobin did not affect diagnostic performance of ORi.

Conclusion

ORi may detect mild hyperoxemia with PaO_2 between 120 and 140mmHg and limit excessive hyperoxia in mechanically ventilated dogs, although it does not replace blood gas analysis for assessment of oxygenation.

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A comparison of traditional, fencl-stewart approach and quantitative analysis of acid–base imbalances in dogs with diabetic ketoacidosis.

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Introduction
Complex acid-base imbalances are a common finding in dogs with endocrine emergencies. This preliminary study was designed to compare the diagnostic performance of traditional, semiquantitative, and quantitative approaches for the evaluation of acid–base imbalances in dogs with diabetic ketoacidosis (DKA).

Methods
A restrospective cohort study was performed between November 2020 and November 2022. Dogs admitted to the Emergency Service with DKA were considered for inclusion if venous blood samples for acid–base, lactate, and serum biochemical analysis were all collected on admission. Acid–base analysis was performed using the traditional or Henderson-Hasselbach approach, quantitative (Stewart), and semiquantitative (Fencl-Stewart) approaches.

Results
Fourteen dogs were included in the study. Traditional acid–base analysis identified simple metabolic acid–base abnormalities in 2/14 dogs and a mixed disorder (high anion gap metabolic acidosis and respiratory alkalosis) in 12/14 dogs. The Stewart approach identified metabolic abnormalities in all patients; strong ion difference abnormalities were evident in 6/14 cases, Atot acidosis or alkalosis in 13/14 cases, and strong ion gap acidosis in 9/14 cases. The semiquantitative approach also identified abnormalities in all cases evaluated. Imbalances due to disturbances in water balance were detected only in 6/14 cases, due to the chloride effect in 12/14 patients, and to the lactate effect in 4/14 dogs. Acid-base effect due to abnormalities in the concentration of weak acids (albumin and/or phosphate) was found in 11/14 patients. Of the 14 patients with more than one acid-base en imbalance, all patients showed an acidotic effect associated to the increased concentration of unmeasured anions, mainly ketoacids.

Conclusion
The quantitative and semiquantitative approaches diagnosed more acid–base abnormalities in this population than the traditional approach. Both methods may provide greater insight as to the underlying etiology of abnormalities or comorbidities, which may be of particular relevance in dogs with severe changes in the concentration of strong ions such as ketones, sodium, chloride, and weak acids such as albumin and phosphorus. Further studies are necessary to confirm the clinical utility of using these physicochemical approaches in the decision-making process of dogs with such diabetic emergency.

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A multimodal tissue perfusion measurement approach for the evaluation of the effect of pimobendan, an inodilator, in a porcine sepsis model

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Introduction

Tissue perfusion disorders are major pathophysiologic determinants of sepsis. Thus, early identification and treatment of these disorders remain an important goal to optimize the outcome of the patients. Pimobendan, an inodilator, may improve tissue perfusion. The aims of the study were: 1) to assess the effect of pimobendan on macrocirculation and perfusion and 2) to describe a multimodal approach to the assessment of perfusion in sepsis and compare the evolution of the perfusion parameters.

Methods

Eighteen anesthetized female piglets (*Sus domesticus)* were equipped for macrocirculation monitoring (cardiac output, invasive arterial pressure). Sepsis was induced by an infusion of *Pseudomonas aeruginosa*. After the occurrence of shock (mean arterial pressure < 40 mmHg), animals were resuscitated with fluids, noradrenaline and/or dobutamine. Eight pigs received pimobendan (0.25 mg/kg IV) at the start of resuscitation maneuvers, the others received saline. Tissue perfusion was assessed using temperature gradients measured with infrared thermography (TG = core temperature – tarsus temperature), urethral perfusion index (uPI) derived from photoplethysmography, and sublingual microcirculation, assessed with a Sidestream dark field imaging device, giving the following parameters: De Backer score (DBs), proportion of perfused vessels (PPV), microvascular flow index (MFI) and heterogeneity index (HI). Systemic biomarkers of tissue perfusion (arterial lactate and $ScvO_2$) were also measured.

Results

The infusion of bacteria was associated with a significant decrease in uPI and increase in MFI. Shock was associated with a significant decrease in uPI, PPV and $ScvO_2$ and a significant rise in TG. Only TG could significantly predict an increase in lactate. Pimobendan did not improve tissue perfusion nor macrocirculation. It did not allow a reduction in the amount of noradrenaline and fluids administered. In all animals, resuscitation was associated with a significant increase in uPI, DBs, MFI, lactate and $ScvO_2$. There were fair correlations between the different perfusion parameters.

Conclusion

In this model, pimobendan did not show any benefit. The multimodal approach allowed the detection of tissue perfusion alteration, but only temperature gradients predicted the increase in lactatemia. The variability of the intensity of shock and data obtained precludes any definitive conclusion regarding a potential superiority of one method over the others.

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Evaluation of serum and urinary uric acid in dogs with sepsis

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Introduction

Uric acid (UA) acts as a marker of oxidative stress and tissue hypoperfusion in different critical care settings in humans. In septic people, hyperuricemia has been associated with increased risk of acute kidney injury (AKI) and worse outcomes. The aim of this study was to investigate the diagnostic and prognostic value of UA in dogs with sepsis, and to assess its ability for the prediction of AKI and septic shock.

Methods

Septic dogs were prospectively included according to SIRS criteria and a confirmed source of infection. A control group of healthy dogs was included for comparison. Clinicopathological data, including serum UA (sUA, mg/dL), urinary UA (uUA, mg/dL) and uUa to urinary creatinine ratio (uUA/uCr) were measured at hospital admission. Septic dogs were grouped based on the presence of AKI (as previously reported in IRIS AKI criteria), and/or septic shock, and final outcome. Non-parametric statistics were performed, and significance was set at $P<0.05$.

Results

55 dogs with sepsis and 50 healthy dogs were enrolled. Dogs with sepsis had significantly higher sUA (0.59, 0.0 – 5.68 vs 0.16, 0.0 – 0.31; mg/dL) and uUa/uCr (0.12, 0.0 – 0.55 vs 0.06, 0.0 – 0.14) compared to healthy ones ($P<0.0001$). sUA was higher in dogs with septic shock (n=17) than in those with uncomplicated sepsis (n=38) (0.73, 0.0 – 5.68 vs 0.43, 0.0 – 1.30; mg/dL; $P=0.0144$), and higher in non-survivors (n=21) compared to survivors (n=34) (0.74, 0.0 – 5.68 vs 0.45, 0.0 – 1.30; mg/dL; $P=0.0246$). No difference was documented between septic dogs with AKI (n=31) and without AKI (n=24). A positive correlation was documented between sUA and lactate concentration ($r=0.524$, $P=0.0010$).

Conclusion

In our study, sUA and uUA/uCr behave as biomarkers of critical illness and sepsis severity, likely reflecting an ongoing condition of tissue hypoxia/hypoperfusion. A potential role of UA in predicting AKI in dogs with sepsis was not confirmed by the present data.

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Clinical manifestations, laboratory findings, treatment and outcome of acute aortic thromboembolism in 202 cats: a retrospective study

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Objective
The aim of this large-scale retrospective study was to describe the clinical and laboratory findings, risk factors and outcome of cats presenting with acute Aortic Thromboembolism (FATE).

Methods
Medical records of cats presented on an emergency basis were retrospectively reviewed. Cats were included if FATE was diagnosed based on compatible history and clinical signs (acutely down in any limb/s, pale, hypothermic, pulseless, painful, or lacking motor function). Cats were excluded if FATE could not be definitively identified.

Results
The study included 202 cats diagnosed with FATE (133 males, 69 females) with a median age of 96 months (range 11-294). Hypothermia (87%), tachypnea (85%), paraparesis/paraplegia (82%), and vocalization (52%) were common. In 179/196 (91%) of the cats, ATE caused bilateral hindlimb paresis\plegia, while in 19, 3 and 2 cats, 1, 3 and 4 limbs were involved, respectively. In 94% of cats, an underlying was suspected or diagnosed, with a median left atrium/aorta ratio of 2.27 (range 1-4). Common biochemical abnormalities included systemic hyperglycemia, azotemia, and elevated muscle enzymes concentration. Common coagulation abnormalities included prolongation of prothrombin time and activated partial thromboplastin time, and elevations in d-dimer concentrations. Median glucose concentration difference between affected and normal limbs was 136 mg/dL (range, 27-387). Non-survivors had significantly lower rectal temperatures and higher glucose, potassium, phosphorus, and total protein concentrations, as well as azotemia, compared to survivors (P<0.045 for all). A logistic regression model predicted that for every 1° Celsius drop in rectal temperature, there was a 2.2-time increase in the odds ratio for mortality (95% CI 0.318–0.631). In addition, for every 1 mg/dL increase in serum creatinine or 1 mEq/L increase in serum potassium, the odds ratio for mortality doubled (95% CI 1.156–3.094, and 95% CI 1.230–3.219, respectively). Survival to discharge rate for cats in the present study was 31%; 63% of non-survivors were euthanized.

Conclusion and Clinical Importance
FATE carries a guarded-poor overall prognosis for survival to discharge. Hypothermia, hyperglycemia, potassium, phosphorus and total protein concentrations, as well as azotemia, are negative predictors of survival.

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Evaluation of modified qsofa and news scores in emergency feline patient

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Introduction

Early recognition of critically ill emergency patients is indispensable for outcome and prognosis. The aim of this retrospective study was to evaluate two severity of illness scores: quick Sequential Organ Failure Assessment (qSOFA) and its variants and 2 modified National Early Warning Score (NEWS), and to compare their performances to a validated feline score (APPLEfast).

Methods

Clinical records of cats presented to the Emergency Department of the University of Pisa over six months were analyzed. All cats with a complete clinical examination and blood gas analysis were enrolled; respiratory rate (RR), heart rate (HR), systemic arterial pressure (SAP), mean arterial pressure (MAP), temperature, Glasgow coma scale (GCS), at arrival were used. APPLEfast, qSOFA score (and its variants), and 2 modified NEWS were calculated. Data were assessed for normality using D'Agostino–Pearson test. The receiver operating characteristic curve (AUROC) was calculated for each score. Cut-offs were set, and sensitivity and specificity were calculated.

Results

We enrolled 107 cats: 74 survived (71%) while 33 did not (29%); 14 cats died spontaneously (42%), 19 were euthanized (58%); the latter were excluded from statistical analysis. AUROC analysis was 0.77 (95% CI 0.63-0.92) for APPLEfast score, 0.71 (95% CI 0.54-0.87) for qSOFA (SAP+RR+GCS), 0.72 (95% CI 0.56-0.89) for qSOFA1 (qSOFA + temperature), 0.78 (95% 0.64-0.92 CI) for qSOFA2 (qSOFA + lactates), 0.77 (95% CI 0.63-0.91) for qSOFA 3 (qSOFA + lactates + temperature), 0.57 (95% CI 0.52-0.82) for NEWS (HR, RR, SAP, temperature, mentation score, oxygen administration), and 0.71 (95% CI, 0.55-0.87) for NEWS1 (NEWS + lactate). APPLEfast showed good specificity (71%) and good sensitivity (77%), with a cut-off of 22. A cut-off of 2 for qSOFA2 showed optimal sensitivity (93%) but only moderate specificity 54 %. For NEWS1, a cut-off of 9 resulted in a sensitivity of 86% and a specificity of 61%.

Conclusion

The results confirm the validity of the APPLEfast for cats; qSOFA2 appeared to be a sensitive score useful for identifying clinical deterioration early. A prospective study will be required to validate these modified scores in clinical practice.

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Small animal emergency medicine practice and education in portugal- an online survey

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Introduction

In Portugal, emergency medicine is usually practiced by veterinarians without specific post-graduate training. Teaching and practice of this field has not been previously assessed. The present study aimed to 1) identify how emergency medicine is taught and practiced 2) explore students' expectations versus clinicians' descriptions of job realities, and 3) identify confidence levels with common emergency cases/procedures.

Methods

Two separate surveys were distributed online to veterinary students and veterinarians. The questionnaires evaluated demographics, educational background, working conditions (staffing/hours), self-reported confidence levels with clinical scenarios (i.e., trauma, dyspnea, etc.), procedures (i.e., thoracocentesis, emergent intubation, etc.), and client communication. The surveys were reviewed by the ethics board.

Results

A total of 192 students and 330 clinicians responded. A perception of a lack of specific training in emergency medicine during school was reported by both students (29.5%) and veterinarians (29%), with 78.7% of veterinarians classifying their academic training as insufficient for their daily practice. Eighty-one percent (81.7%) of veterinarians had worked emergency shifts without nurses, although 77% of students considered having support staff for these shifts important. While many individuals reported they felt comfortable with cases such as vomiting/diarrhea (99.1% clinicians, 79% students), lameness (88.4% clinicians, 66.1% students), and urethral obstructions (94.8% clinicians, 55.3% students), both groups selected animals with upper airway obstruction (66.5% clinicians, 76.3% students), collapse (55.8% clinicians, 91.4% students), or dystocia (41.6% clinicians, 75.3% students) as patients they were less confident managing. Students were generally uncomfortable with all procedures examples, while veterinarians considered placement of thoracic drains (75.1%), pericardiocentesis (73.6%), and thoracic point-of-care ultrasound (60.1%) as the most challenging. Additional teaching incorporated into academic (77.6%) and post-graduate training (40.3%) were the top suggestions from clinicians to improve their confidence in managing emergency cases.

Conclusion

This is the first descriptive study of current training and practice of veterinary emergency medicine in Portugal, supporting the increased need and enthusiasm for field-specific training. Students may have discrepancies in beliefs about job requirements and support staff available after graduation. While our results may be biased by characteristics of respondents, they suggest that increased emergency medicine training opportunities would be popular in the country.

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A preliminary study to investigate the prevalence and risk factors for the development of chronic kidney disease post acute kidney injury

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Introduction
Chronic kidney disease (CKD) is a recognized sequela to acute kidney injury (AKI). This study aimed to assess the prevalence of CKD in dogs after azotemic AKI utilizing creatinine, SDMA, and direct GFR.

Methods
Client-owned dogs with azotemic AKI were recruited (T0) between January 2021-January 2022 with re-evaluation at 3 (T1) and 12 (T2) months. Dogs with CKD were excluded. Serum creatinine (SCr), SDMA, urine protein-to-creatinine ratio (UPCR), and systolic blood pressure (SBP) were performed at each visit. In non-azotemic dogs (SCr <145µmol/l) at T1 and T2, GFR was determined by iohexol clearance. AKI grade and CKD stage were determined according to IRIS guidelines. CKD staging was based on SCr and SDMA at T1 and T2 unless the dog did not survive until T2. Non-azotemic dogs were considered to have renal dysfunction if GFR was decreased >20% compared to the reference interval for that body weight category.

Results
15 dogs with azotemic AKI were recruited (etiologies: Leptospirosis n=4, non-steroidal (NSAID) toxicity n=3, NSAID and general anesthesia n=6, unknown n=2). Maximal AKI grade was III (n=4), IV (n=8) and V (n=3) at T0. At discharge, 66.6% (10/15) of dogs remained azotemic. At T1 20% (3/15) remained azotemic; this remained stable at T2 in 2/3 dogs. 1 dog was euthanized prior to T2 due to progression of azotemia. 33.3% (4/12) and 36.3% (4/11) of non-azotemic dogs had evidence of renal dysfunction based on GFR at T1 and T2, respectively. 53.3% (10/15) dogs were classified as IRIS CKD stage 1, 26.6% (4/15) stage 2, and one dog that did not survive to T2 was stage 4. Proteinuria (UCPR >0.5) was identified in 71.4% (10/14) dogs at T0. At T1 no dogs and T2 one dog was proteinuric. Hypertension (≥160mmHg) was identified in 73.3% (11/15) at T0. 13% (2/15) and 15% (2/13) were hypertensive at T1 and T2, respectively. 2/4 hypertensive dogs had normal renal function.

Conclusion
Persistent azotemia occurs infrequently in dogs surviving beyond 3 months post-AKI, but GFR measurement may identify renal dysfunction in non-azotemic dogs. Persistent proteinuria and hypertension are uncommon and do not appear associated with CKD progression.

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Prevalence and risk factors for malignancy and hemangiosarcoma in non-traumatic hemoabdomen in dogs

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Introduction
Non-traumatic hemoabdomen is a common medical emergency in veterinary medicine. Once primary coagulopathy has been ruled out, a bleeding mass is suspected. The most common mass diagnosed in these cases is hemangiosarcoma accounting for 63-70% of cases. Since the long-term survival for dogs with hemangiosarcoma is poor, owners may opt for euthanasia rather than pursue surgery. Prognostic indicators known beforehand may help owners make this difficult decision. The aim of the present study was to determine risk factors for malignant versus benign diagnoses and hemangiosarcoma versus other diagnoses.

Methods
Medical records of dogs presented to a veterinary teaching hospital emergency clinic were retrospectively reviewed. Dogs were included if presented with non-traumatic hemoabdomen where a primary coagulopathy was ruled out as the cause, and a definitive diagnosis was made via histopathology.

Results
The study included 95 dogs diagnosed with non-traumatic hemoabdomen (52 males, 43 females) with a median age of 10.5 years (range 2.5-17). Sixty-one cases (65%) had malignant diagnoses, 50 of which were diagnosed with hemangiosarcoma. Thirty-four cases (35%) were benign. There was no difference in age or weight between groups. Hemangiosarcoma dogs had higher occurrences of leukocytosis, anemia, thrombocytopenia, eosinophilia, hyponatremia, hypoglycemia, low total plasma proteins, and an activated partial thromboplastin time (aPTT) below the lower reference range ($P<0.05$ for all). Compared to dogs with benign diagnoses, the dogs with malignant diagnoses had higher occurrences of thrombocytopenia, eosinophilia, hyponatremia, and aPTT below the lower reference range ($P<0.05$ for all).

Conclusion
In contrast to previous studies, age and weight were not found to be risk factors for hemangiosarcoma. New risk factors for hemangiosarcoma found in this study included eosinophilia, hyponatremia, and hypoglycemia.

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Retrospective observational study of acute post-operative complications occurring in the ICU following canine mitral valve repair surgery under cardiopulmonary bypass

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Introduction

The frequency of post-operative complications experienced in the ICU after mitral valve repair (MVR) is unknown. The primary aim was to describe the clinically relevant post-operative ICU complications of dogs undergoing MVR with a secondary aim to analyze their effect on in-hospital mortality.

Methods

Dogs undergoing MVR who survived to post-operative ICU admission at the Royal Veterinary College, London, between May 2019 and January 2020 were eligible for inclusion. Cases with incomplete data for any variable were omitted from that analysis. Demographic data, mortality, length of hospitalization, and clinically relevant complications were collected. Clinically relevant complications were those requiring any intervention that deviated from routine protocol for post-operative care following MVR. Fisher's exact test was used for all stated results and Holm-Bonferroni correction for multiple comparisons was performed with post-correction alpha set at 0.05.

Results

Forty-one dogs comprised the final study population. Common breeds were small crossbreed dogs (26.9%), Cavalier King Charles spaniels (17.1%), and Chihuahuas (12.2%). Age was 10.2 years (1.8-14.4), gender was predominately male (60%, neutered n=19, entire n=5) with 40% female (13 neutered, 4 entire). ICU hospitalization was 43 hours (6-115) with length of hospitalization of 8 days (1-19). Of 38 dogs who survived until ICU discharge, 7 required re-admission. Overall, 36 (87.8%) dogs survived to hospital discharge; three dogs underwent repeat surgery, of whom 1 survived. Twenty-nine (70.7%) experienced at least one clinically relevant complication, and the following were not significantly associated with survival; hyperlactatemia (>2.5mmol/l; n=18), AKI (n=9), hypernatremia (>165mmol/l; n=7), new anti-arrhythmic requirement (n=6), new cardiovascular abnormality (septal hematoma/myocardial infarction/PTE; n=5), calcium or potassium bolus administration (n=2), tranexamic acid administration (n=2), glucose supplementation (n=2), oxygen provision beyond 24 hours (n=2), new antihypertensive requirement (n=1) and hypotension (SAP <90 mm Hg; n=1). Only new-onset neurologic deterioration (n=3; P=0.026) and greater than 1 post-operative transfusion (n=11; P=0.036) retained significance following correction.

Conclusion

Survival after MVR is good, and although clinically relevant complications are common, few have a robust association with survival. The low alpha value increases the risk of type 2 error in this study. Prospective studies are warranted to further explore these associations.

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Nursing of a dog with generalised tetanus: care of the neurological patient

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Introduction

Tetanus is a well-known neuromuscular disease in mammals that can develop after entry of a spore through a contaminated wound. The main clinical manifestation is tetanic muscular contractions. Unlike humans and horses, dogs are considered at low risk for this condition. However, tetanus can be life-threatening in dogs, and excellent nursing care is essential.

Synopsis

A 4-month-old female intact German Shepherd was referred to the ICU for facial swelling, dysphagia and difficulty prehending food, and tremors that did not improve with diazepam and antibiotics given by the referring veterinarian. Upon admission, the dog was presented in lateral recumbency with generalized tremors and spastic tetraparesis. The dog was initially managed with intravenous fluids, supplemental oxygen and a gastrostomy tube (PEG technique) was placed to start early enteral feeding. Daily, physiotherapy was performed to limit muscle contractures and wasting. Intravenous catheters and gastrotomy tube increase risk for nosocomial infections and other complications. Daily protocolized nursing care led to early detection of a subcutaneous abscess next to the gastrostomy tube insertion site. The abscess healed with appropriate wound care. Careful nursing care with strict feeding protocols likely helped avoid aspiration pneumonia in this dog, a frequently reported complication in dogs with generalized tetanus. We also prioritized external stimulation and tender loving care, especially important for the puppy's development. She was discharged after 20 days of hospitalization given, her overall good condition, her ability to stand up and eat unassisted.

Conclusion

Prolonged hospitalization, intense nursing care and enteral feeding are warranted in the management of generalized tetanus. Excellent nursing protocols can improve patient prognosis and limit complications. This presentation will allow me to describe all nursing procedures I performed during hospitalization.

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Locoregional versus systemic analgesia in a dog with a severe wound: nursing inputs in pain assessment

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Introduction

Pain assessment is an essential skill among the different tasks involved in veterinary nursing. Although several pain scales have been developed and used in clinical practice, close interaction with the patients provides information that is difficult to get with intermittent pain evaluation. Furthermore, continuous locoregional analgesia for pain management in hospitalized animals is more commonly used, and nurses should be familiar with these techniques.

Synopsis

A 10-month-old female entire Spanish Mastiff dog was admitted to our hospital with a severe and deep avulsion wound in the dorsal aspect of the lumbosacral area. Bloodwork performed showed anemia and leukocytosis. The patient was anesthetized for surgical debridement and cleaning of the wound. During the same anesthesia, an epidural catheter was placed at the level of L3-L4, and advanced caudally down to L7. The analgesic plan included bupivacaine 0.20% every 6 hours through the epidural catheter and systemic NSAIDs SID. Recovery from anesthesia was uneventful. All the pain score evaluations for the following days were below 3/24, based on the Glasgow (SF-GCPS) scale. The dog was eating, drinking, urinating normally, and walking with a slight ataxia. The bandage was changed and the wound cleaned once a day, without need sedation. Five days after the epidural catheter placement, the filter was dislodged from the catheter, and its removal was necessary to avoid potential complications. A new analgesic plan was set up by adding systemic methadone every 4 hours. The following pain scores remained lower than 5/24, so rescue analgesia was not necessary. However, it was noticed her behaviour had changed slightly: the patient was less active, she turned suddenly to look at her wound sometimes, and was more tense during the bandage changes. Based on this information, paracetamol was added TID, and these slight behavioural changes disappeared.

Conclusion

Pain assessment is always challenging. Close interaction with the patients can add information about patients´ analgesia status even when pain scales are used. Since locoregional analgesia techniques are more commonly used in veterinary medicine, it is crucial to know how to manage them and their potential complications.

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Nuts! The curious case of the missing prostate

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Introduction

Jack is a two-year-old, male neutered Canine who presented to UF ECC department for a diagnosed uroabdomen, three days post-op from a cryptorchid neuter surgery done by the rDVM. It was reported that the surgery was complicated, noting the retained testicle to be significantly adhered and requiring dissection.

Synopsis

Upon presentation, the owner's stated Jack was extremely painful and had not urinated. At their rDVM, earlier that day, diagnosed a uroabdomen (Abdominal centesis BUN 130, creatinine too high to read and azotemic on Chemistry). Jack presented dull, tachycardic (160 bpm), dehydrated, bounding femoral pulses, and tacky mucus membranes. Significant bloodwork abnormalities included: BUN 107 mg/dL, create 7.23 mg/dL, K+ 5.5 mEq/L, Phos 7.1 mg/dL, HCO3- 15 mEq/L. aFAST showed abdominal effusion (no bacteria seen on cytology) and an abdominal drain was placed, along with an indwelling urinary catheter, which had some difficulty/ resistance on placement. Abdominal CT revealed urethral transection, a left inguinal cryptorchid testicle, urinary bladder having medium extravasation, a missing prostate, and both catheters to be in the abdominal cavity (the abdominal drain in place, but the urinary catheter to be exiting the urethra in the transected location.) The patient was checked into ICU where I started ECG Telemetry, sustained tachycardia noted even with boluses of analgesics. Started Lidocaine, Fentanyl, and Ketamine CRIs, placed a PICC line and administered a 5ml/kg LRS bolus after volume underload was noted on tFAST. The cryptorchid testicle was removed, the bladder was repaired, along with a urethral R&A and urethropexy. Surgery placed an indwelling u-cath, NG tube, and Jackson-Pratt abdominal drain. On recovery, multiple USGs, PCV/TP of peripheral blood and abdominal fluid were checked, along with volume of fluid output. Signs of sepsis and hydration status were closely monitored and by three days post-op, the patient started eating, no longer requiring tube feedings, maintained a normal UOP and had an improved mentation.

Conclusion

Jack was discharged after 15 days in hospital with mild urinary incontinence. Serial Weights, fluid outputs from the drains/catheters, and indirect BPs monitoring indicated patient's hydration, volume balance and renal function, aiding in the case's lack of sepsis.

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Ultrasonographic signs of idiopathic lung fibrosis in west highland white terriers

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Introduction

The aim of this study was to find a correlation between pulmonary crackles on physical examination and the presence of various abnormalities on lung ultrasound (LUS) in West Highland White Terriers (WHWT). Our hypothesis was that B-line artifacts are present in increased numbers in patients with pulmonary crackles on auscultation.

Methods

Retrospective study; medical records from 2019 – 2022 were reviewed. We enrolled 57 WHWT dogs treated chronically with clinical signs of cough. All dogs were clinically diagnosed with idiopathic lung fibrosis based on auscultation and excluding other causes; the clinical diagnosis was not confirmed with CT or histopathology. Dogs with mitral valve disease stages B2, C, and D were excluded from the study. LUS was performed by experienced clinicians (two with over 10 years of experience and one with over 3 years of experience in echocardiography and LUS) with a 6 MHz phased array probe, followed by a 12 MHz linear probe. The sonographers were not blinded to clinical examination findings. The probes were placed on the chest wall in three vertical lines (sternal, shoulder and scapular) according to a standardized protocol we created. The assessment included the presence of B- or Z-lines, lung sliding, pleural line abnormalities, and subpleural consolidations. Statistical analysis was performed on the collected data.

Results

The most common LUS abnormalities were irregular pleural line (44, 77%); B-lines (34; 60%); blurred pleural line (28; 49%); decreased lung sliding (12; 21%); Z-lines (10; 17,5%); and subpleural consolidations (8; 14%). Pulmonary crackles were audible in 42 of 57 dogs (74%); 24 of them had B-lines on LUS. Pulmonary crackles were absent in 15 dogs, but 10 of them had B-lines on LUS. The analysis found no significant difference between animals with pulmonary crackles that had B-lines vs those that did not (p=0.558); furthermore, no correlation was found between pulmonary crackles and other LUS abnormalities (p=0.705 – 0.917).

Conclusion

Pulmonary crackles are not correlated with the presence of B-lines in WHWT dogs with idiopathic pulmonary fibrosis. Therefore, pulmonary crackles may appear in dogs with “dry” or “wet” lungs on LUS.

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Acid-base and electrolyte changes in dogs after storaged packed red blood cell transfusion

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Introduction

Transfusion of stored packed red blood cells (pRBC) in infants does lead to changes in biochemical parameters and pH that can have a significant clinical impact. However, these changes have not been correlated to patient outcomes. This study was designed to describe changes in acid-base and biochemical parameters in dogs after transfusion of pRBC and potential correlation with outcome.

Methods

A prospective observational study was conducted between August 2020 and November 2021. Dogs admitted to the intensive care unit were included if they required a transfusion of stored pRBC. Venous blood gas and electrolytes were measured before and within one hour after transfusion. Signalment, underlying disease, days of hospitalization, units of pRBC transfused and outcome were also recorded. Associations between analytical values and all other parameters before and after transfusion were assessed using Wilcoxon test and Spearman's correlation coefficient to assess possible correlations between pairs of variables. Influence of one variable on another was evaluated using univariate linear regression analysis.

Results

Twenty-six dogs were included. Underlying causes of anemia included immune-mediated diseases (9/26), toxic insult (1/26), acute bleeding (9/26) and others (7/26). The minimum range of blood administered was 10 ml/kg/day and the maximum was 20 ml/kg/day. Dogs remained hospitalized for a mean of three days, and the mortality rate in our study was 16% (3/26). Statistically significant differences were found before and after transfusion in the following parameters: heart rate, respiration rate, hematocrit, hemoglobin, HCO_3^-, PCO_2, Anion gap, base excess, tCO_2, chloride and lactate. Higher values were observed before transfusion in heart rate, respiration rate, Anion gap, base excess and lactate, and after transfusion in hematocrit, hemoglobin, HCO_3^-, PCO_2, tCO_2 and chloride. Mortality showed a statistically significant correlation with tCO2, meaning the higher the tCO_2 difference, the higher the mortality was.

Conclusion

Transfusion of storage pRBC does improve physical exam and acid-base parameters. tCO_2 difference might be a new prognostic indicator, but further research is warranted**.**

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A retrospective analysis of venom-induced consumptive coagulopathy following eastern brown snake (pseudonaja textilis) envenomation in dogs and cats in South-East Queensland, Australia

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Introduction
Venom-induced consumptive coagulopathy (VICC) occurs rapidly following snake envenomation and is defined as a coagulopathy caused by venom toxin consumption of clotting factors resulting in elevated prothrombin (PT) and activated partial thromboplastin (aPTT) times, with low or undetectable fibrinogen. Limited reports describe the incidence of VICC following Eastern brown snake envenomation (EBSE). There are no reports of the recovery time or patient outcomes following EBSE VICC in dogs and cats. The objective of this study was to retrospectively describe clinical features, incidence, recovery time and survival of VICC in dogs and cats with EBSE.

Methods
Patient records from a university veterinary teaching hospital from 2017 to 2022 were analysed. Cases were included if EBSE was confirmed. Data on initial clinical presentation, diagnosis, clinicopathologic findings, treatment and outcomes were collected. Descriptive statistics were reported.

Results
240 dogs and 98 cats were included. Dogs commonly presented with lower motor neuropathy (66.2%), pre-paralytic collapse (30.8%), and dyspnea (29.1%). For cats, lower motor neuropathy (93.9%), dyspnea (56.1%), and hypothermia (52.0%) were the most commonly observed clinical signs. Of those tested on presentation, 209/226 (92.5%) dogs and 81/98 (82.6%) cats had evidence of VICC. Median (range) time to normalisation of PT and aPTT was 24 (4-72) hours for dogs and 24 (6-42) hours for cats. Hemorrhage was recorded in 91/240 dogs (37.9%) and 8/98 cats (8.2%), with fatal haemorrhage reported in 8 dogs and 2 cats. Animals euthanised on presentation were excluded leaving 210 dogs and 77 cats for outcome analysis. Overall survival was 87.6% in dogs versus 74.0% in cats. Survival of dogs and cats presenting with VICC and without VICC was 91.8% and 80.3%, and 94.1% and 83.3%, respectively. Of the 90 dogs and 8 cats that suffered hemorrhage, 76.7% and 37.5% survived, respectively.

Conclusion
Most dogs and cats presenting for ESBE have VICC, with over a third of dogs also experiencing hemorrhage. The time to recovery of VICC for EBSE is similar for dogs and cats, and in line with that reported in humans.

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Retrospective evaluation of autotransfusion versus allotransfusion in the perioperative management of acute hemoperitoneum in 43 dogs (2017-2021)

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Introduction

The objectives of this study were to assess the effectiveness and safety of autotransfusion with a cell salvage (CS) device in dogs.

Methods

A retrospective study was conducted in dogs with acute hemoperitoneum of splenic origin and associated surgical treatment between 2017 and 2021. Dogs were grouped according to the cause of hemoperitoneum (malignant vs. benign) and type of blood transfusion received, autotransfusion (CS-group), allotransfusion (AO-group), no transfusion (NT-group), and allo- and autotransfusion (AA-group). Changes from preoperative to postoperative HCT were compared. Results of the initial measurement and the measurement closest to the timepoint 12 hours postoperative were used. The amount of blood required, duration of surgery, and outcome were analyzed.

Results

Forty-three dogs were included. Twenty-seven (62.8%) had a pathohistological diagnosis of hemangiosarcoma, the remaining 16 had a benign cause (37.2%). Eleven dogs (25.6%) were in the AO-group, seven (16.3%) in the CS-group, eleven (25.6%) in the AA-group and 14 (32.5%) in the NT-group. Mean HCT of the AO- and AA-group did not change significantly from the preoperative (AO: 26.8%; SD 7.0, AA: 32.7%; SD 7.4) to the postoperative value (AO: 26.7%; SD 4.9, AA: 33.4%; SD 11.7). In the CS-group mean HCT rose from 29.5% (SD 11.9) to 34.1% (SD 8.9) and in the NT-group mean HCT decreased from 36.6% (SD 9.1) to 30.0% (SD 7.9). A significant difference in the development of HCT was evident only between the NT- and CS-groups ($p=0.04$). The median amount of red blood cell transfusion (autologous and allogenic) given was 47.7ml/kg (range 23-126.5ml/kg) in the AA-group, 7.6ml/kg (range 4.61-13.5ml/kg) in the AO-group and 19ml/kg (range 14-50ml/kg) in the CS-group. The amount of allogenic transfusion did not differ between the AA-group (median 9.4ml/kg; range 5.0-16.1) and the AO-group ($p=0.68$). Dogs treated with CS received more often plasma transfusions ($p=0.03$) and more plasma per kilogram ($p=0.02$).
The use of the CS device had no negative impact on duration of surgery or outcome. No major complications were reported in any subgroup.

Conclusion

The CS device appears to be a safe additional treatment possibility to allogenic blood transfusions in dogs with acute hemoperitoneum.

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Retrospective study of perioperative parameters in dogs undergoing splenectomy

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Introduction

The objectives of this study were to investigate the most common splenic pathologies leading to splenectomy in dogs, patients' characteristics, final diagnosis, perioperative parameters, complications, and hospital stay/mortality rate, trying to find prognostic factors for the immediate postoperative period.

Methods

Retrospective observational study on 98 dogs presented for splenectomy between 2003 and 2021. Dogs presenting concomitant diseases or other surgery in the same operating time were excluded. Normality was assessed with Shapiro-Wilk test. Variables were analyzed with Student's paired *t*-test, Wilcoxon test, Chi-Square and/or Fisher's exact test. Correlations were examined with Spearman's or Pearson's correlation coefficient. ROC curves were generated for hematocrit (Htc) and platelet number in relation with hemoabdomen. Significance was set at $p < .05$. All statistical tests were performed using the SAS 9.4 package.

Results

79.8% (75/98) of the dogs underwent splenectomy due to splenic masses; 57.3% of all biopsies were diagnosed as neoplasms, with 60.8% of them being hemangiosarcomas (35.2% of total biopsies). The prevalence of splenectomy was higher in males ($p=0.0047$), older than 9 years ($p<0.0001$) and big-sized (≥17kg, $p=0.0012$). Hemoabdomen was a common complication (40.5% of patients) and significatively associated with thrombocytopenia ($p<0.0001$), anemia ($p=0.0026$), leukocytosis ($p=0.0098$), hemangiosarcoma ($p=0.0463$) and longer hospital stay ($p=0.0225$). The optimal cut-offs that better discriminated between the presence or absence of hemoabdomen were Htc 33.55% (70% sensitivity, 60% specificity, AUC 0,704) and 181×10^3/µL platelet count (84.6% sensitivity, 65.4% specificity, AUC 0.808). 80% of anemic dogs and 73.5% of dogs with presurgical leukocytosis presented a malignant neoplasm ($p=0.0002$, $p=0.007$). A highly significant association was found between the presence of anemia and/or leukocytosis with hemangiosarcoma diagnosis ($p=0.0037$, $p=0.0094$). 10% of the dogs needed erythrocyte transfusion, which in turn was highly associated with the presence of hemoabdomen ($p=0.0128$) and thrombocytopenia ($p=0.0078$). Only 2 dogs died (2.04%) during the perioperative period.

Conclusion

Intra-hospital mortality rate after splenectomy is very low. Thrombocytopenia and anemia could be considered as negative prognostic factors as they are related to complications (such as hemoabdomen and increased need for transfusion) or biopsy results with a poor prognosis (such as hemangiosarcoma).

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Retrospective review of pericardiocentesis in dogs and cats (2016-21): disease prevalence and long-term outcome

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Introduction

Different diseases can cause pericardial effusion or cardiac tamponade. Pericardiocentesis can be a therapeutic and/or diagnostic procedure. The aim of this retrospective study was to review disease prevalence and outcome in dogs and cats undergoing pericardiocentesis.

Methods

The institution's archive was searched for patients that underwent pericardiocentesis (August 2016-September 2021). Cases were included if they had complete case record and full echocardiogram. Repeat pericardiocentesis, final diagnosis (where available), outcome and additional diagnostics were collected.

Results

Sixty-one (61) patients (55 dogs and 6 cats) were included. The most common breeds were crossbreeds (14) and domestic shorthairs (5). No sex difference was found. Mean age at presentation was 10 years (0.9-19). All dogs presented with cardiac tamponade. Pericardiocentesis was performed once in all cats and 39 dogs, 10 dogs had repeat pericardiocentesis, 6 dogs had ≥3 pericardiocentesis. Echocardiography identified a cardiac mass in 38 dogs (27 right atrium, 9 heart base and 2 of complex anatomical location), 7 dogs had an intrathoracic mass. Left atrial tear causing tamponade was identified in one dog. An idiopathic cause was presumed in 9 dogs. Two cats had pyopericardium, 2 multiple systemic and cardiac disease, one each had right atrial or a pericardial mass. Pericardiocentesis was performed successfully in all patients. Two dogs were euthanized briefly after pericardiocentesis, and six additional dogs were euthanized within 24 hours for a similar reason (relapse of cardiac tamponade resulting in euthanasia). Three cats died for non-cardiac reasons (>3mo after pericardiocentesis). Cytological analysis was performed in 40 patients; the most common diagnosis was hemopericardium, but cytology also identified neoplastic cells (9) or neutrophilic effusion. Median survival was 135 days (dogs) and 184 days (cats). Relapse of tamponade requiring repeat pericardiocentesis in dogs surviving > 24hr occurred 118 days (12-368) since the first event. The presence of a mass was associated with reduced survival (median survival cardiac/thoracic mass 17 days vs 437 no mass, $p<0.05$). Only a minority of dogs underwent pericardiectomy (5).

Conclusion

In most dogs with cardiac tamponade, a mass was identified (both intrathoracic or cardiac). The main complication leading to cardiac-related euthanasia was recurrence of cardiac tamponade.

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Viscoelastic test results in dogs with adrenal tumors

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Introduction

Although an association between neoplasia and hypercoagulable states is suggested, there are no studies documenting its occurrence. Dogs with pituitary-dependent hyperadrenocorticism have been shown to be hypercoagulable, but hypercoagulability in dogs with primary adrenal tumors has not been reported. This pilot study aimed to evaluate the incidence of hemostatic abnormalities in dogs with adrenal tumors.

Methods

A prospective observational study was performed. Results of a viscoelastic coagulation test (VCM Vet™) were recorded on all patients before surgery for adrenalectomy. The VCM was performed according to the manufacturer's instructions. Computed tomography was performed on all patients before surgery. Histopathological exams from the adrenal mass (es) were performed for dogs submitted to surgery. Descriptive statistical analysis was used.

Results

Twenty-three dogs were enrolled, of which 9 had pheochromocytoma, 7 adrenal adenoma, 4 adrenal carcinoma, and 3 without a final diagnosis. The median age was 12 years. Fifteen patients (65%) had one or more abnormal viscoelastic test results indicating a hypercoagulable profile, while no dogs had results suggesting hypocoagulability. The amplitude after clot time (A10 and A20) plus the clot formation time (CFT) were the most commonly abnormal results, followed by the clot time (CT). Three dogs (3/15 or 20%) had concomitant impaired fibrinolysis due to decreased lysis index (LI) results at 30- and 45- minutes after clot time. Two dogs (2/15) had mildly increased CT results despite increased A10, A20, and maximum clot firmness (MCF) results. Pheochromocytomas (7/15; 47%) were most commonly associated with hypercoagulability, followed by adrenocortical adenoma (5/15; 33%) and carcinomas (1/15; 7%). The overall mortality rate was 17% (4/23). All deceased dogs were hypercoagulable; adenoma (1), carcinoma (1), and pheochromocytoma (2). Interestingly, 6/23 (26%) had gross vascular invasion (phrenicoabdominal vein and vena cava), of which only 3 had hypercoagulability. The tumor size or vascular invasion was not associated with hypercoagulability in this small number of cases.

Conclusion

Hypercoagulability seems to be common in dogs with adrenal tumors. A larger number of patients will be necessary to detect an association between hypercoagulability and more specific tumor characteristics such as tumor type, size, and vascular invasiveness.

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Effects of room temperature on stored canine and feline packed red blood cells quality control analysis

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Introduction

After processing, packed red blood cells (pRBC) units are stored at temperatures between 2-6°C. The percentage of hemolysis is an indicator of cellular integrity. The purpose of the study is to assess the effect of room temperature exposure in pRBC units, based on the percentage of hemolysis and the risk of bacterial contamination.

Material and Method

Forty pRBC units (20 canine and 20 feline) discarded for clinical use due to positive results in serology and/or PCR of infectious agents were used. After processing, pRBC units were stored in a dedicated fridge at 2-6°C. After 2 days of storage, units were placed at controlled room temperature (18-22°C) for 3 days and then returned to the fridge to be stored for 42 days. Hemolysis percentage was tested at T0(after processing), T1(21 days after donation(AD)), T2(28 days AD), T3(35 days AD) and T4(42 days AD). Blood cultures were performed at T0 and T4.

Results

In canine pRBC units, the average percentage of hemolysis was 0.20%(0,11-0,58%) at T0; 1.51%(0,74-2,81%) at T1; 2.75%(1,01-6,96%) at T2; 5.74%(2,33-7,26%) at T3; and 6.02%(5,07-9,38%) at T4. Of the 20 canine pRBC units, 18 units would have been discarded for clinical use at T1 due to presenting a percentage of hemolysis >0.8%, and 2 units at T2.
In feline pRBC units, the average was 0.27%(0,05-0,73%) at T0; 3.94%(0,39-12,95%) at T1; 7.64%(0,55-16,07%) at T2; 9.55%(0,74-16,36%) at T3; and 10.72%(079-16,51%) at T4. Of the 20 feline pRBC units, 11 units would have been discarded for clinical use at T1 due to presenting a percentage of hemolysis >1.5%; and 8 units at T2. Blood cultures were all negative.

Conclusion

The results of this study suggest that, after a break in the cold chain and exposure at room temperature for three days, the percentage of hemolysis increases significantly, and these units are unsuitable for clinical use. It is not expected an influence of infectious agents on the results of the present study, as all units presented an acceptable percentage of hemolysis on T0. New studies are needed to evaluate the effect in pRBCs of shorter exposure to room temperature.

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The effect of leukodepletion on canine pRBC contaminated with infectious agents

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Introduction

Pre-storage leukodepletion of blood products is used to prevent leukocyte and platelet-induced transfusion reactions and is a widespread and mandatory technique in humans. Leukodepletion has been shown to reduce the risk of transfusion-associated cytomegalovirus disease in human medicine. To assess the effect of pre-storage leukodepletion on the presence of *Leishmania spp*., *Ehrlichia spp*., and *Anaplasma spp*. in canine pRBC units.

Methods

From all screening tests for infectious agents in potential canine blood donors performed at a veterinary blood bank, 60 samples with positive real-time PCR results (LightCycler 480II, Roche) for *Leishmania spp*., *Ehrlichia spp*., or *Anaplasma spp*. were selected for this study. After leukodepletion of the blood unit, the quantitative PCR to detect the positive agent was repeated. Positive PCR results were recorded as "quantification cycle" (Cq), indicating the number of amplification cycles needed until a positive PCR result was obtained. Negative result was considered when Cq>37. In addition, an automatic WBC count was performed on the pre- and post- leukodepletion count samples (XN-550, Sysmex).

Results

Sixty blood samples of potential canine donors with positive real-time PCR results were included: *Leishmania spp*. (n=4; mean Cq 36.11 (SD=1.78)); *Ehrlichia spp.* (n=11; mean Cq 29.99 (SD=3.81)) and *Anaplasma spp*. (n=45; mean Cq 31.50 SD= (4.02)). After leukodepletion, all units previously positive for *Leishmania spp.* became negative; of the previously positive units to *Ehrlichia spp*., 5 became negative and 6 remained positive (mean Cq=34.86; SD=2.72); and of the previously positive units to *Anaplasma spp*., 28 became negative and 17 remained positive (mean Cq=32.91; SD=1.83). All units showed a decrease in the number of WBCs after leukodepletion: pre- leukodepletion 10.32x10^3/µL (SD=3.26 x10^3/µL) and post- leukodepletion 0.02 x10^3/µL (SD=0.01 x10^3/µL).

Conclusion

Leukodepletion could decrease the risk of transfusion-mediated transmission of *Leishmania spp*. For the post-leukodepletion units that remained positive to *Ehrlichia spp*. or *Anaplasma spp.,* the Cq values increased after leukodepletion, demonstrating a decrease in the parasitic load.

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Retrospective evaluation on the outcomes of dogs and cats with tick paralysis requiring mechanical ventilation in new South Wales, Australia

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Introduction

The primary objectives of this study were to describe the indications and outcomes for patients with tick paralysis (*Ixodes holocyclus*) requiring mechanical ventilation and whether rates are similar to previously reported in other regions of Australia.

Methods

A retrospective observational study was conducted using medical records to identify dogs and cats diagnosed with tick paralysis that required mechanical ventilation between January 2017 to December 2021. Diagnosis was based on the presence of a progressive ascending neuromuscular paralysis with either identification of a paralysis tick, or the absence of a tick with or without a tick crater if from endemic geographical areas. Indication for mechanical ventilation was categorized as hypoventilation, hypoxemia unresponsive to oxygen therapy, impending respiratory fatigue, or respiratory arrest. Historical, biochemical and ventilation data were analyzed to evaluate risk factors for survival.

Results

A total of 36 patients were identified; 32 dogs and 4 cats. The indication for mechanical ventilation was hypoventilation for 19.4% (n=7), hypoxemia in 16.7% (n=6) and respiratory fatigue with impending respiratory arrest in 63.9% (n=23) of patients. Overall survival rate to discharge was 69% (n=25). A total of 83.3% (n=30) patients were diagnosed with aspiration pneumonia based on radiographs or positive microbial growth from an airway culture with cytological evidence of intracellular bacteria. Pneumonia was present in 73.9% of survivors and 84.6% of non-survivors and was not associated with survival (OR 0.51, 95%CI 0.35-10.62, p=0.68). The number of ventilation days did not differ between survivors (median 3, IQR 2-4) and non-survivors (median 3, IQR 2-3.5, p0.68). Overall, 66.7% (n=24) of patients failed initial attempts at liberation. Successful liberation on the first attempt was significantly associated with survival, with 92.3% of non-survivors failing the first attempt, compared to 52.2% of survivors (OR 0.09, 95% CI 0.01-0.063, p=0.025).

Conclusion

The results of this study show that survival rates for animals diagnosed with tick paralysis requiring mechanical ventilation are similar between different geographical regions in Australia. However, the indication for mechanical ventilation differed, with an unsustainable respiratory effort more common in this study compared to hypoxemia and hypoventilation.

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Heterologous plasma segments for ocular use produced in a commercial animal blood bank - a pilot clinical study

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Introduction

Ulcerative ocular lesions are difficult to treat conditions. Topical and systemic broad-spectrum antibiotics and surgical debridement are frequently applied in these treatments. Serum or plasma are valuable compounds to use as adjunctive therapy. Serum is applied topically using multidose bottles; however, its long-term use may increase the risk of bacterial contamination. The goal of this study was to verify the efficacy and potential side effects of a new heterologous plasma single-dose (HPSD) presentation produced in an animal blood bank for adjuvant treatment of different ocular ulcers in companion animals.

Methods

A total of 10 animals with a diagnosis of corneal ulcers were included in the study. To complement the conventional treatment, a complementary treatment using HPSD every 4 hours during 4- 5 days was applied to each animal. All animals were evaluated by a veterinary ophthalmologist. Photograph records were obtained at the beginning and end of the treatment with owners' informed consent.

Results

This study included the clinical records of 7 dogs and 3 cats, from several breeds and ages ranging from 5 to 144 months. All animals showed an improvement (n=5/10) or total resolution (n=5/10) of their ocular condition at the end of the treatment. The strategy of a product used in a single dose, led to high owners' adherence to the treatment. Also, there were no reported adverse reactions during HPSD treatment.

Conclusion

The HPSD presentation showed to be an effective and safe product to be used in ocular ulcers and was associated with a favorable clinical course. This presentation was easy to apply by veterinarians and owners with medical indications. Its potential ability to reduce secondary bacterial infections and product waste, as well as to apply a standard volume in each treatment, reinforces HPSD potential to be used commercially by professionals. No patient suffered adverse effects associated with the application of HPSD. The main limitations of this work are the low number of animals included and the lack of a control group to assess the efficacy of the treatment. In addition, the use of plasma in the treatment of dry eye has already been described.

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Validation of a new qualitative color scale to determine the percentage of hemolysis in feline and canine packed red blood cells

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Introduction

The percentage of hemolysis is an important indicator of the cellular integrity of packed red blood cell (pRBC) units, being used to determine pRBC's shelf life. This study aims to validate a color scale that relates the color of supernatant in centrifuged pRBC and their percentage of hemolysis.

Methods

A sample of pRBC was transferred to a microhematocrit capillary tube and centrifuged. The color of the supernatant was compared to a color scale developed by Banco de Sangue Animal (BSA), classified from 1 to 10 (transparent to dark red), where 1 represents a low degree of hemolysis and 10 a high degree. The units were classified into group 1, colored between 1-5; and group 2 between 6-10. The graduation of the color scale of each unit was compared with the real percentage of hemolysis, calculated using the formula: *"% hemolysis = Supernatant HGB(g/L)x(100-PCV)/Total HGB(g/L)".*

Results

A total of 2539 different samples were evaluated: 2168 were evaluated by members of the BSA staff, and 371 by Hospital's staff. From the visual evaluations performed by BSA staff, 94.1% (2041/2168) were classified in group 1 and 5.9% (127/2168) in group 2. From group 1, 95.1% (1941/2041) had a percentage of hemolysis < 1%. From group 2, 92.9% (118/127) had a percentage of > 1%. Regarding the Hospital's staff evaluations, 83.8% (311/371) were classified in group 1 and 16.2% (60/371) in group 2. From group 1, 94.5% (294/311) had a percentage of hemolysis < 1%. From group 2, 40% (24/60) had a percentage of > 1%.

Conclusion

This color scale could be a useful tool to determine the viability of stored pRBC's units according to the percentage of hemolysis. Units with a score from 1 to 5 very likely presented hemolysis < 1%, which indicates the unit is suitable for transfusion. Units with score >5 may be incorrectly discharged if the score is evaluated by Hospital staff. Since the samples evaluated were not from the same pRBC's units, the scale assessment may depend on the operator's experience. Further prospective study must be made to analyze this statement.

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Modeling the relationship between arterial blood pressure and sublingual microcirculation in anesthetized piglets

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Introduction

Videomicroscopy devices can perform bedside sublingual microcirculation measurements. Although the sublingual microcirculation is practical, our understanding of its circulatory physiology, such as perfusion autoregulation, is limited. Our objective was to assess the relationship between arterial pressure and sublingual microcirculatory blood flow in an anesthetized piglet model. We hypothesized this relationship would be linear if the sublingual vessels do not exhibit autoregulation, or bilinear if they exhibited autoregulation.

Methods

Sublingual microcirculation was assessed using a Sidestream Dark Field (SDF) handheld imaging device in seven anesthetized piglets that underwent pharmacologically-induced - using sevoflurane or noradrenaline - blood pressure variation: mean arterial pressure (MAP) ranged from 30 to 120 mmHg. Microcirculation was filmed at different MAP values. Microvascular flow index (MFI) represents a semi-quantitative approach of sublingual blood flow. To evaluate the hypothesis of a linear relationship, we performed a linear mixed model with MFI as a response variable and MAP as an explanatory variable. The individual was included as a covariate (random effect). The bilinear model was built by assuming a "MAP-threshold" value beyond which the relationship between MAP and MFI changed slope. For MAP values below the "MAP-threshold", the relationship was: MFI=constant1*MAP+constant2. For MAP values above the "MAP-threshold", the relationship was: MFI = constant (called "MFI-threshold").

Results

For each MAP recording, there was a large intra- and inter-individual MFI variability. Graphically, no model appeared satisfactory. For the first hypothesis, the model showed a significant linear relationship (coefficient = 4*10-3, 95% confidence interval = [4*10-4; 7*10-3], P = 0.03). For the second hypothesis, the model identified a point of inflection for a MAP-threshold of 55 mmHg (95% confident interval = [32; 78], P < 0.0001) and an MFI-threshold of 2.6 (95% confident interval = [2.5; 2.7], P < 0.0001).

Conclusion

Large variability in videomicroscopy recordings of the sublingual microcirculation and the semi-quantitative assessment of local blood flow precluded assessment of any relationship between MAP and MFI, particularly whether there is a linear or bilinear relationship based on the presence or absence of local autoregulation. Further work is needed to improve the performance of sublingual videomicroscopy.

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Association between the risk of hemoparasites infection and the blood type, sex, and breed in canine blood donors

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Introduction

Emerging data in human medicine suggests an association between blood type and susceptibility to certain microorganisms, such as *Plasmodium falciparum*, *Helicobacter pylori*, *Noroviruses* and *SARS-CoV-2*. An association between ethnic groups and infectious diseases has also been suggested.

Objective

To assess the prevalence of infectious agents considering the blood type, sex, and breed in a population of blood donors in Portugal and Spain.

Methods

All potential canine blood donors from a veterinary blood bank in Portugal and Spain who underwent analysis for infectious agents (*Leishmania spp., Ehrlichia spp., Babesia spp., Anaplasma spp.,* and *Dirofilaria immitis*) between January and December 2022 were considered. Three possible associations were investigated: 1) presence of infectious agents and DEA 1 blood group; 2) presence of infectious agents and sex; and 3) presence of infectious agents and breed. Statistical analysis was performed using the SPSS 28.0 software. The Chi-square test was applied.

Results

A total of 8690 potential blood donors were considered. 1) 286 DEA 1 negative donors and 527 DEA 1 positive donors were positive for some infectious agent. No association was found between the blood type and the presence of infectious agents ($p > 0.05$). 2) 507 males and 306 females were positive for some infectious agent. No association was found between sex and the presence of infectious agents ($p > 0.05$). 3) More than 40 canine breeds were tested, and an association was found between the breed and the presence of infectious agents with $\chi 2 (168) = 293.530$; $p < 0.001$. Canine mixed breeds were the group that showed the greatest susceptibility to the presence of hemoparasites (n=203), followed by Portuguese Podengo (n=178) and Spanish Podenco (n=167).

Conclusion

The results obtained in this study suggest that the presence of infectious agents in potential canine blood donors is not influenced by the blood type or sex, while this could be influenced by the breed.

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Pain-related syndrome of inappropriate antidiuretic hormone secretion in a kitten

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Objective

To describe a clinical case of syndrome of inappropriate antidiuretic hormone secretion (SIADH) associated to pain due to an orthopedic trauma in a kitten.

Case summary

A 2-month-old, intact male, domestic shorthair kitten was presented to the emergency service for evaluation of weakness, gait abnormalities, and marked unilateral left hind limb lameness after accidental trauma. On orthopedic examination, severe pain, crepitus, and swelling of left hind limb were detected. On neurological exam, no abnormalities were detected. Results of diagnostic work-up (CBC, biochemistry panel, blood gas, T_4 levels, abdominal ultrasound, and thoracic X-rays) were all consistent with only severe hyponatremia, hypochloremia, and a Salter-Harris type I fracture of the left femoral head and distal epiphysis. The kitten was initially treated with balanced isotonic crystalloids, buprenorphine, and maropitant. Twelve hours after admission, the patient's condition worsened. The kitten showed severe depression and evidence of uncontrolled pain on manipulation. Several samples of blood and urine were collected to go further in the diagnostic workup revealing worsening hyponatremia (113 mmol/L; [RI: 146,2-156,2]), severe plasma hypo-osmolality (218.2 mOsm/kg; [RI: 287-307 mOsm/kg]), high natriuresis (Na: 74.9 mmol/L; [RR: <40 mmol/L]), and urinary hyper-osmolality (630 mOsm/kg; [RR: <150 mOsm/kg]). Based on such results, the hyponatremia detected on admission, and lack of evidence to support any underlying thyroidal or adrenal disease, SIADH was diagnosed.
Emergency treatment with hypertonic saline was then instituted, and a bolus (2 ml/kg) of 3% hypertonic saline to initially increase plasma sodium of 4-6 mmol/L was administered. Once the target was achieved, the fluid therapy was changed to achieve a controlled rise of plasma sodium concentration (<10 mmol/L in the first 24h). To promote the excretion of positive body water balance, loop diuretic was instituted to force diuresis. Two additional boluses of furosemide (1 mg/kg/IV) were administered 12h apart. Buprenorphine was discontinued; instead, metamizole was started as analgesic therapy throughout the hospitalization period. Discharge occurred 4 days after admission as the patient was clinically stable and the hyponatremia resolved.

PACS

New or unique information provided

To the author's knowledge, this is the first report of a kitten developing SIADH presumably associated to pain.

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Cardiac defibrillation as emergency treatment of ventricular fibrillation in a sphynx cat with hypertrophic cardiomyopathy and acute congestive heart failure

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Background

Ventricular arrhythmias are identified in cats with hypertrophic cardiomyopathy (HCM), but life-threatening ventricular tachycardia (VT) is seldomly reported. Sudden cardiac death is an uncommon complication of HCM, affecting approximately 2.2% of cats. This is presumed to be caused by malignant ventricular arrhythmias, like in people. The authors aimed to report a case of sustained monomorphic VT refractory to emergency IV antiarrhythmics, which required external cardiac defibrillation due to acute deterioration.

Case presentation

A 10-year-old, male neutered, 8kg Sphynx cat was referred for emergency treatment of acute congestive heart failure. The cat was found in lateral recumbency, tachypneic and was transferred to our hospital by the referring veterinarians, which administered an IM furosemide bolus. On presentation the cat was quiet, alert but responsive. A fast heart rate with poor femoral pulses was appreciated (HR 300bpm). Respiratory rate was mildly increased (44 breaths/min). Emergency cardiac assessment identified sustained monomorphic VT associated with HCM phenotype and minimal B-lines. 4 boluses of IV lidocaine (0.5mg/kg) and subsequently IV esmolol (0.1 mg/kg) were administered with no success. The VT acutely deteriorated into ventricular fibrillation, at which point the patient lost consciousness, with absent pulses. Emergency biphasic defibrillation was performed at 50 J and was successful in restoring sinus rhythm and return to spontaneous circulation did not require additional chest compressions. Oral sotalol and congestive heart failure treatment were started. A complete echocardiogram identified an area of presumed chronic myocardial infarction. Troponin I was elevated in the acute setting and substantially decreased over time. The patient did well for 4 months, after which he developed refractory congestive heart failure.

New/ Unique information

VT is an uncommon clinical presentation in cats with HCM, which can acutely worsen and cause cardiac arrest. Lacking response to IV antiarrhythmics, cardiac defibrillation can be resolutive, preventing arrhythmic death. In this patient, echocardiography identified an area of presumed left ventricular myocardial infarction, which could have favored VT. Oral medications were effective in controlling the arrhythmia long-term and no recurrence of VT was noted. Although acute prognosis seemed poor, adequate quality of life was achieved for 4 months since emergency presentation.

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Case report: percutaneous insertion of small-bore central line to treat an hepatic abscess in a dog

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Background

The traditional therapeutic approach for an abdominal or thoracic abscess includes invasive surgical procedures that may not be the best option for unstable patients. This report describes conservative management of a hepatic abscess using a small bore central catheter inserted through the modified Seldinger technique.

Case Presentation

A twelve-year-old male Jack Russell dog was presented with signs of compensated shock five days after a hepatic lobectomy for nodule removal. On admission, the patient was markedly tachycardic (160bpm) and had a fever (41ºC). Bloodwork revealed moderate regenerative anemia (28% - 96Kreticulocytes/uL) and mild leukocytosis (19,2Kleukocyte/uL). A cavitated hypoechogenic lesion (2,5 x 3 cm) was found at the previous hepatic surgical site, together with a small amount of echogenic free fluid during the abdominal ultrasound. A sample was taken from both sites; a bacterial culture and cytology were performed, revealing the presence of bacilli bacteria in the liver. Due to the age of the patient and a chronic cardiac illness, the owner declined the surgery. The patient received fluid therapy, analgesics, and antibiotics initially. A small-bore 22G central catheter was placed through the modified Seldinger technique under ultrasound guidance to drain the abscess. The catheter remained in place and was used to wash the lesion every 6 hours allowing recording the amount and aspect of secretions. On day four, the catheter was removed without signs of leakage to the abdominal cavity. On day five, the dog was fully recovered and discharged uneventfully with oral amoxicillin-clavulanate continuation (20mg/Kg three times a day) due to sensible *Escherichia coli* within the culture.

New / Unique information

To our knowledge, this is the first veterinary report describing the use of a percutaneous approach to treat an abdominal abscess. This could represent a novel non-invasive way to treat localized cavitary infections with better results than the traditional surgical approach. However, more veterinary trials are required to confirm the clinical benefits. In conclusion, this technique could be recommended to owners with financial constraints or unstable patients as an alternative to more invasive procedures.

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Pericardial effusion in two dogs with suspect eastern brown snake (pseudonaja textilis) envenomation in Queensland, Australia

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Background

Venom-induced consumptive coagulopathy (VICC) can occur in dogs with Eastern brown snake envenomation (EBSE). Pericardial effusion has never been reported in any domestic species with VICC. This report describes pericardial effusion in two dogs with VICC secondary to suspected EBSE.

Case presentations

A 7-year-old male neutered Border Collie cross dog presented for acute collapse. Presenting clinical signs included tetraparesis, hyperthermia, increased respiratory effort, and muffled heart sounds. EBSE was presumptively diagnosed based on positive snake venom detection test (brown snake immunotype), geographical location, and season (spring/summer). Initial prothrombin time (PT) was 19 seconds, and activated partial thromboplastin time (APTT) was prolonged out of range. Viscoelastic testing reported a prolonged clotting time, reduced clot strength and increased clot lysis. Thoracic Point-of-Care Ultrasound (POCUS) identified a small pericardial effusion without evidence of cardiac tamponade. The dog was treated with 8100IU tiger-brown antivenom. The pericardial effusion resolved over three days without the need for pericardiocentesis.

A 7-year-old male neutered American Staffordshire Terrier was referred for management of acute onset tetraparesis and hypoventilation requiring emergency intubation. Presenting clinical signs included muffled heart sounds, and POCUS identified a 3-4cm pericardial effusion without evidence of cardiac tamponade. PT and APTT were normal. A small bleeding wound on the lip was noted and based on history had been present for 24 hours. Pericardiocentesis was performed and 505mL of hemorrhagic fluid removed (PCV/TP similar to peripheral blood). A presumptive diagnosis of EBSE was made due to clinical signs (tetraparesis and suspicion of transient coagulopathy), geographical location and season. A vial of 8100IU Tiger-Brown antivenom was administered. The patient received supportive care and was mechanically ventilated for 24 hours before resumption of spontaneous ventilation. The tetraparesis gradually improved and the patient was discharged after four days. The pericardial effusion did not reoccur.

New / Unique information

This is the first report of pericardial effusion occurring secondary to suspected EBSE in dogs. Management of EBSE should include investigation for pericardial effusion, and snake envenomation should be a differential in cases presenting with a pericardial effusion.

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Reduction of serum ibuprofen concentration in a dog with hemoadsorption using the cytosorb adsorber and therapeutic plasma exchange

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Background

Therapeutic plasma exchange (TPE) is an effective treatment strategy for life-threatening ibuprofen intoxications in dogs. However, the amount of required plasma products is not always available. Hemoperfusion using the Cytosorb adsorber may be used as an alternative.

Case presentation

A 14 kg, 10.5-year-old, female spayed mixed-breed dog was presented after ingestion of 260 mg/kg ibuprofen six hours prior to presentation. Successful emesis was initiated, and charcoal was administered at the referring veterinarian two hours after ingestion. The initial clinical examination revealed mildly reduced general condition, and dry mucous membranes with normal capillary refill time. Body temperature was 37.8°C. Neurologic examination was unremarkable.

The patient received supportive medical therapy with IV fluids, gastrointestinal protectants and antiemetics. An 80-minute cycle of therapeutic plasma exchange (Vet Smart, PEX 400 filter), with additional hemoperfusion using a Cytosorb adsorber, positioned before the plasma filter, was initiated 8 hours after ingestion. During the extracorporeal treatment, 7000 ml of blood (500 ml/kg) were processed by hemoperfusion. A total of 1100 ml plasma (78 ml/kg; 1.5 times patient's plasma volume) were removed and substituted with 800 ml 5 % human albumin and 260 ml donor plasma.
Serum samples for ibuprofen concentration analysis at a commercial laboratory were collected before and after the treatment, as well as during the treatment before and immediately after the Cytosorb adsorber. Pre-treatment ibuprofen concentration of 170 µg/ml was reduced by 82 % during and decreased to 5 % 30 hours after extracorporeal therapy. Ibuprofen reduction ratio achieved by the Cytosorb adsorber alone ranged from 67 % at the beginning of the treatment to 15 % at the end of the treatment.

The dog was discharged 34 hours after presentation with no gastrointestinal symptoms and normal concentrations of urea and creatinine. Five months later, the owner reports no abnormalities since discharge.

New/unique information

Hemoperfusion using Cytosorb adsorber is effective in reduction of serum ibuprofen concentration in combination with TPE. Further research is warranted to determine the effectiveness of the Cytosorb adsorber alone as an alternative to TPE.

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A case of hypoventilation after placement of an external splint in a cat with severe pectus excavatum

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Background

Pectus Excavatum (PE) is a rare condition characterized by a dorsal deviation of the sternum, which potentially causes respiratory compromise. Recent studies in cats have shown that computed tomography (CT) can better assess the severity of PE by calculating several indices such as Vertebral Index (VI), Correction Index (CI), and Asymmetry Index (AI). Correction of PE is usually achieved with external splinting. Complications of this procedure include pneumothorax, puncture of the lungs/heart, re-expansion pulmonary edema, and pyoderma.

Case presentation

A twenty-week-old, male, Maine Coon cat (BW 3,5 kg) was presented with a two-day history of depression, dysorexia, and increased respiratory effort. At physical examination, a mild dyspnea and a marked depression of the caudal sternum were observed. CT scan diagnosed a severe caudal PE with left mediastinal shift associated with severe vertebral lordosis (VI: 2,43; CI: 68%; AI: 98,4). An external splint was surgically placed two weeks after the presentation. After postoperative extubation, persistent hypoventilation occurred ($PaCO_2$: 73 mmHg, PaO_2: 123 mmHg, oxygen supplementation with nasal cannula) and mechanical ventilation (MV) was needed. Total intravenous anesthesia (ketamine/dexmedetomidine/midazolam) was used, and the patient was maintained alternating assisted pressure-control and pressure-support ventilation. Daily spontaneous breathing trials (SBT) were performed during the next two days, but tidal volume was inadequate. On day 3, a successful weaning attempt was made and the patient was extubated. $PaCO_2$ was normal the following day. The external splint was removed after six weeks, with improved radiographic indices (VI on X-rays: 11,8) and complete resolution of clinical signs.

New/Unique information

Given the severity of the malformation of the caudal aspect of the chest, the authors suspect that the cat relied predominantly on intercostal and accessory respiratory muscles for respiration. Atrophy of the diaphragmatic muscles and presence of an external splint could have caused hypoventilation during the postoperative recovery. A mild sedative protocol was used to allow assisted MV in order to encourage the activity of the primary inspiratory muscle. Daily SBT were performed to evaluate ventilatory improvement. To the authors' knowledge, this is the first description of hypoventilation as post-surgery complication of correction of PE in a cat.

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Human recombinant factor VIII treatment for severe gingival hemorrhage and dental surgery in a dog with Hemophilia A

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Background

Hemophilia A (HA), a genetic coagulation factor VIII deficiency, commonly causes spontaneous bleeding. Recombinant human factor VIII (rhFVIII) is administered to treat bleeding in HA human patients, but was not reported in hemophilic dogs.

Case presentation

A 7-year-old intact male poodle dog was admitted with marked oral hemorrhage of 4-day duration. HA was diagnosed 5 years before and the dog was previously hospitalized several times due to bleeding. Two months prior, molar and pre-molar teeth were extracted due to severe ongoing gingival hemorrhage and dental disease, and major intra- and post-operative hemorrhage occurred despite pre-operative cryoprecipitate, tranexamic acid (TXA) and desmopressin treatment, requiring 4 FFP units and 1 packed RBC unit post-op. Physical examination showed pale mucous membranes, marked gingival hemorrhage, tachycardia, and a left apical systolic cardiac murmur. The packed cell volume was 21% (reference interval, 37-55%). Oral examination under anesthesia revealed bilateral oronasal fistulas in both maxillary teeth. Due to ongoing bleeding, dental extractions were planned. Immediately prior to induction, an IV bolus of rhFVIII (Advate® 5 mg/kg) was administered, followed by a 20-hour IV constant rate infusion (CRI; at 4 mg/kg/hour). TXA was administered upon induction (30 mg/kg slow IV, followed by 3-hour CRI at 30 mg/kg/hour). During surgery, 1 packed RBC unit was administered. Thromboelastometry, performed prior to treatment, revealed intrinsic-pathway hypocoagulability, typical of HA, which resolved upon repeated thromboelastometry, 12 and 36 hours post-commencing rhFVIII therapy. Citrated plasma samples from the dog and 2 control dogs taken before, during, and after treatment were sent to a human laboratory for quantification of factor VIII activity; however, failed to document a measurable increase. The dog recovered uneventfully with no evidence of bleeding and was discharged 36 hours post-op.

New / Unique information

This first report of successful rhFVIII treatment for HA-induced clinical bleeding supports this therapy in hemophilic dogs.

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Xenotransfusion of canine fresh frozen plasma and canine fresh whole blood for the treatment of feline coagulopathy

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Background

Xenotransfusion is the transfer of blood from one species to another. Successful administration of fresh whole blood (FWB) or packed red blood cells (pRBC) from dogs to cats has been well documented. There are no reports describing xenotransfusion for the treatment of feline coagulopathy.

Case presentation

A two-year-old, domestic short hair cat was presented due to acute gastrointestinal hemorrhage. Physical examination was unremarkable, besides moderate fever and rectal bleeding. CBC revealed severe thrombocytopenia, and severe prolongations of prothrombin time (PT), and activated partial thromboplastin time (aPTT). Initial presumptive diagnosis was anticoagulant intoxication: The cat was treated with type-specific feline FFP at 10ml/Kg, vitamin K1, and tranexamic acid. Following initial treatment, PT remained severely prolonged and aPTT was mildly prolonged. Thromboelastometry (TEM) demonstrated severe hypocoagulability, with no evidence of effective clot formation. Due to lack of typed feline FFP and owner financial constraints, the use of canine blood products was suggested. The cat received cFFP (10ml/kg) and canine pRBC (5ml/kg). Repeated blood work showed mild overall improvement; thus, the treatment was repeated. Coagulation panel performed following the second xenotransfusion was suggestive of DIC, with D-dimer levels exceeding the measurable capacity, mild prolongations of PT and aPTT, hypofibrinogenemia, and decreased antithrombin activity. CBC demonstrated severe thrombocytopenia and moderate anemia. TEM demonstrated mild improvements but was still consistent with severe hypocoagulability. Additional 10ml/kg cFFP were administered, followed by 18ml/kg of cFWB. Repeated CBC revealed normal hematocrit, and persistent thrombocytopenia. Significant improvements were noted in coagulation profile parameters. TEM demonstrated substantial improvement with first evidence of platelet recruitment and stable clot formation. Clinically, the cat had no evidence of active hemorrhage. The following day the cat deteriorated, exhibiting melanotic diarrhea, tachypnea, mental depression, and general weakness. Repeated blood work showed recurrent moderate anemia and severe coagulopathy. The owner chose to withdraw treatment and euthanasia was performed. Necropsy revealed acute pancreatic necrosis, and per-acute multifocal pulmonary thromboembolism with histopathological evidence of DIC.

Unique information

Canine to feline xenotransfusion using cFFP and cFWB for treating severe coagulopathies has not been previously described. Further studies are needed to establish the efficacy and safety of this treatment.

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Ozonized saline solution lavages in a dog with multiresistant bacterial peritonitis due to a gunshot

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Background

Septic peritonitis is a life-threatening condition. Management includes surgical debridement, treatment of the septic source and appropriate antibiotic therapy. Post-surgical drainage of the abdominal cavity facilitates the primary action of peritoneal defense, removing bacteria and inflammatory mediators. Saline lavage through the drainage may help to remove bacteria and inflammation. Ozone is a gas with a high oxidative potential, it has affinity for carbon double bonds and can interact with bacterial cytoplasmic membranes affecting osmotic stability, promoting oxidation of aminoacids and nucleic acids, and causing bacterial cellular lysis. Ozonated saline solution (O_3SS) has been shown to possess antibacterial and anti-inflammatory effects and has been used successfully in human wound healing, chronic pelvic pain and in experimental peritonitis in rats.

Case presentation

A female mongrel dog, seven months old, 30 kg, was received in the ER/ICU service due to septic peritonitis induced by a gunshot. Five intestinal perforations and an abscess with the bullet inside were observed during laparotomy. The affected area was resected, and an anastomosis was performed; a closed active drain was placed. Blood and peritoneal fluid cultures were performed. Post-surgical treatment included intravenous antibiotics (amoxicillin/clavulanate, metronidazole and marbofloxacin). While waiting for culture results, daily O_3SS lavages were made. A medical ozone generator (Ozonobaric P- Sedecal®) was used. Ozonation of 250 ml 0,9% saline solution was performed by using the Dual-Kit System® bubbling ozone through the solution (ozone concentration: 50 µg/NmL; 20 minutes). A total of 5 lavages with 250 ml of O_3SS were done, and peritoneal fluid production decreased from 1000 ml to 58 ml/day, with an improvement in clinical signs. A multiresistant *Enterococcus faecium* was isolated in blood and peritoneal fluid only sensitive to sulfamethoxazole/trimethoprim and rifampin. Oral therapy with Sulfamethoxazole/trimethoprim was started after the 5 lavages, and the patient was discharged after 2 days.

New / Unique Information

O_3SS presumably contributed to control multiresistant septic peritonitis due to its bactericidal effect since bacteria were resistant to intravenous antibiotics administered to this patient. This is the first report of O_3SS peritoneal lavages used successfully as a coadjuvant antimicrobial treatment in a dog with multiresistant bacterial peritonitis.

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Multiple organ dysfunction syndrome induced by a rupture of an unclassified large hepatobiliary cyst in a 10-month-old dog

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Background

While hepatobiliary neoplasia is uncommon in dogs (<1.3% of all cancer), liver cystic lesions are sparsely described.

Case presentation

A 10-month-old, 48.2kg, male intact American Bulldog was referred due to suspected anaphylactic shock. At the referring veterinarian (rDVM), acute onset of lethargy, weakness, and vomiting after playing in the backyard 12-hour before presentation were the main complaints. A weak, quiet-alert-responsive, and dehydrated patient was observed. Blood work revealed hematocrit (Hct) of 73%, leukocytosis (29.1K/µL), thrombocytosis (353K/µL), creatinine of 1.5mmol/L, and ALT of 214U/L. Thoracic and abdominal radiographs revealed microcardia, mild pleural effusion, and decreased abdominal serosal detail. Abdominal point-of-care ultrasound (POCUS) diagnosed free fluid; both cavitary effusions had PCV<5% and TS≤3g/dL. Finally, hypotension (unmeasurable blood pressure) was diagnosed. rDVM treatment consisted of hypertonic saline (given once), Normosol-R, fresh frozen plasma, epinephrine by continuous rate infusion, and intermittent injections of diphenhydramine, dexamethasone-SP, maropitant, ondansetron, ampicillin/sulbactam, and pantoprazole. The patient was referred 24 hours later. On admission, tachycardia (150bpm), tachypnea (32rpm), dullness, abdominal distention, and unmeasurable blood pressure were noticed. Ascites plus gallbladder wall edema were observed on POCUS. Venous blood gas showed Hct of 70%, hyperlactatemia (3.9mmol/L), and base excess of -8.4mmol/L. No lactate, blood glucose, and potassium level differences were noticed between the abdominal free fluid and peripheral blood; fluid cytology revealed hemodiluted sample without infectious organisms. Leptospirosis' SNAP test was negative. Urinalysis showed brown urine, blood 3+, bilirubin 2+, and few bilirubin crystals, whereas serum biochemistry showed creatinine of 4.8mmol/L, BUN of 60mmol/L, ALT of 316U/L, and mild hyperbilirubinemia (0.9mg/dL). Similar treatment for anaphylaxis was started, but patient persisted effusing into the abdomen, being vasopressor-dependent and declining; acute kidney injury was diagnosed. Abdominal ultrasound was performed but inconclusive; a small liver and spleen were visualized. However, abdominal and pelvic computed tomography revealed a large ruptured right cystic hepatobiliary mass. New free fluid cytology showed bilirubin levels of 6.2mg/dL. Patient progressed to respiratory arrest before further treatment. Humane euthanasia was elected followed by necropsy.

New / Unique Information

This is a rare case of an unclassified insidious hepatobiliary cyst that progressed for spontaneous rupture and consequent development of MODS.

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Severe hemorrhage due to erosion of the inferior alveolar artery caused by severe periodontitis in 2 dogs

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Background

Periodontitis is a common chronic disease that is typically not considered as differential for oral hemorrhage significant enough to cause severe anemia or hemorrhagic shock. This case report describes the presentation, diagnosis, and management of two dogs presented for profuse oral hemorrhage and severe anemia due to erosion of the inferior alveolar artery (IAA) caused by severe periodontitis.

Case Presentation

A 12-year-old female entire Bearded Collie (case 1) and a 9-year-old female neutered Jack Russel Terrier (case 2) were referred for further investigation of oral bleeding, melena, and hematemesis. Both case 1 and 2 were anemic (Packed cell volume 21% and 20%, respectively) on presentation, with case 2 exhibiting signs of hypovolemic shock. Profuse hemorrhage from the right mandibular arcade originating at the first molar and severe periodontitis were identified on oral exam. Platelet count, activated partial thromboplastin time, and prothrombin time were normal in both cases. Viscoelastic testing was normal in case 1 and showed evidence of hyperfibrinolysis in case 2. Initial management included fluid resuscitation, packed red blood cell transfusions and tranexamic acid, but both cases eventually required surgery to control the hemorrhage. In case 1, surgery was elected the next day due to progressive anemia with failure of resolution of the oral hemorrhage. In case 2, severe continuous hemorrhage required emergent surgery for control of the bleeding shortly after presentation. In both dogs, a laceration of the right IAA was identified and tooth extractions and ligation of the artery were performed. Additional diagnostics included a preoperative computed tomography scan of the head in case 1 and intraoperative dental radiographs in case 2. Surgery was curative for oral hemorrhage in both cases. Case 2 did not survive to discharge due to progression of multiple other comorbidities.

New / Unique Information

This is the first published report of severe anemia and hemorrhagic shock secondary to IAA hemorrhage due to advanced periodontitis in any species. This case report shows that IAA erosion needs to be considered as a differential diagnosis for severe oral hemorrhage and has to be recognized as a condition requiring emergency surgery in veterinary medicine.

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Acute abdomen secondary to intestinal leiomyositis in a dog

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Background

Intestinal leiomyositis is a rare disease scarcely described in dogs. Patients show signs of acute abdomen and initial tests commonly suggest emergency surgery. However, its treatment is mostly medical.

Case summary

A 10-year-old neutered male Dalmatian presented with a two-month history of chronic intermittent gastrointestinal signs, which had acutely worsened. The dog was previously diagnosed and treated for hypothyroidism.
On admission, the dog was hemodynamically stable but had severe abdominal distention and was painful on palpation. Blood tests, abdominal radiographs, and ultrasound performed by a board-certified radiologist were performed. Results showed moderate hyperlactatemic metabolic acidosis, leukocytosis, mild hyponatremia, and hypokalemia. Imaging suggested an obstructive process with absent peristalsis, but the underlying cause could not be identified. The dog was admitted to the intensive care unit for close monitoring and was started on intravenous fluid therapy, antiemetics, and analgesia. Twelve hours later, the abdominal ultrasound showed a moderate amount of free fluid around several loops of small intestine. Cytology of the abdominal fluid, reviewed by a board-certified clinical pathologist, was consistent with a non-septic exudate with a marked population of degenerative neutrophils. Given the patient clinical deterioration and concern for potential intestinal obstruction, an exploratory laparotomy was performed. During surgery, all loops of intestine were markedly distended, but no underlying cause or perforation could be identified. Biopsies were taken and the dog recovered uneventfully. After surgery, the dog was started on amoxicillin-clavulanic, prednisone 2 mg/kg/d, and vitB12 pending biopsy and culture results. Forty-eight hours after surgery, the dog's condition declined, and developed a moderate amount of free fluid compatible with septic peritonitis. A revision exploratory laparotomy was performed: no areas of perforation could be identified either, and primary septic peritonitis was suspected. Unfortunately, the dog experienced a cardiopulmonary arrest and died during the procedure. Necropsy was performed and multiple areas of the gastrointestinal tract were sampled. Histopathological findings were consistent with gastrointestinal leiomyositis; therefore, diagnosis of chronic intestinal partial obstruction CIPO was confirmed.

New / Unique Information

Intestinal leiomyositis should be suspected in dogs with chronic gastrointestinal signs. Primary peritonitis could be related to intestinal leiomyositis.

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Thromboelastography trace in a cat with DIC secondary to Hemangiosarcoma

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Background

Disseminated intravascular coagulation (DIC) has been traditionally diagnosed by a combination of clinical and laboratory findings. Viscoelastic tests such as thromboelastography (TEG) have been proposed as useful diagnostic methods for DIC. Few TEG studies have been performed in dogs with DIC, and there are no publications describing TEG in cats with DIC. TEG has been successful in diagnosing and classify the hypocoagulable stage with hyperfibrinolytic phase of overt DIC in dogs. Our objective is to describe the findings observed in TEG of one cat with DIC secondary to hemangiosarcoma.

Case summary

A 9-year-old, male neutered, domestic short-haired cat was presented with a hematoma and a mass in right limb with no reported history of trauma. Initial blood tests revealed anemia (PCV 11.3%), severe thrombocytopenia (39 K/µL), prolonged aPTT (> 300s; RI: 65-119) and PT (29s; RI: 15-22). Fresh frozen plasma, packed red blood cells, and antifibrinolytics were administered. Laboratory tests were sent to determine TEG trace, D-dimer, and antithrombin III (AT-III). The results showed increased fibrinolysis in the TEG tracing (LY 30 2%; RI: 0-0.7), no alteration on D-dimers, and decreased levels of AT-III (80%; RI: 85-145%). The owners finally decided to euthanize the patient due to the lack of favorable response despite treatment. A histopathologic postmortem study of the mass diagnoses a muscular hemangiosarcoma. Our conclusion is that the patient presented a DIC secondary to hemangiosarcoma.

New/Unique information

In this case, clinical and laboratory criteria were met to reach a diagnosis of DIC, and we proved that TEG was successful in diagnosing hyperfibrinolysis. We found many differences between our TEG results and those previously reported in dogs with DIC; however, these results could have been altered by the transfusions and antifibrinolytics.
This case report demonstrates that haemangiosarcoma in cats can lead to DIC, and TEG can be a useful tool to diagnose the hyperfibrinolytic phase of DIC in feline patients.
Further studies in cats are required to characterize the changes in TEG tracing in patients with DIC as well as the impact of certain therapies.

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Application of fluorescent light energy (FLE) therapy in a septic dog with an extensive necrotic skin lesion

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Background

Fluorescent light energy therapy (FLE) is a novel type of photobiomodulation. FLE has been shown to improve wound healing, accelerate skin recovery, minimize fluid losses, and reduce antibiotic therapy time.

Case presentation

A 7-month-old Collie was presented with a history of 5-day lameness of the right forearm, anorexia, and vomiting. The dog was vaccinated against rabies the previous week. The dog had altered mentation, tachycardia, tachypnea, icteric mucous membranes, increased capillary refill time, and hypotension on clinical examination. Blood tests showed hypoalbuminemia, anemia, thrombocytopenia, azotemia, hyperbilirubinemia, and hyperlactatemia. Extended necrotic skin lesions, involving 30% of the dog's body, were distributed along the ventral and right lateral thorax, right lateral and ventral cervical region, and medial aspect of the forelimbs. Oxygen supplementation, intravenous fluids, constant rate infusion of noradrenaline, blood products, antibiotherapy, analgesia, and supporting treatments were administered during the first four days to manage the septic shock. The initial approach to the wounds was with proteolytic agents. As soon as the dog was stable enough to be anesthetized, surgical debridement was carried out, and the FLE therapy started. The FLE was applied twice a week for the first fifteen days and then once a week. The chromophore gel was extended over the debrided tissue, and the LED was applied. In between FLE therapy sessions, the wounds were managed by cleaning them daily and applying wet bandages. The patient stayed in ICU for nine days and then moved to the surgery ward. At that point, treatment focused on the wounds since the dog ate and drank normally, and the blood tests were within the average values. The dog was discharged seventeen days after admission with analgesia, antibiotherapy, an antithrombotic agent, and outpatient wound management. After six months, the dog remains clinically stable and has only a small skin lesion covering the upper aspect of the right forearm.

New / Unique information

This is the first report in the veterinary literature of the use of FLE as an adjunctive treatment of septic shock in a dog. FLE might be an excellent option to avoid systemic complications of necrotic skin lesions.

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Development and treatment of pyothorax after esophagostomy tube placement in a dog

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Background

Esophagostomy tubes are essential for providing regular food intake in patients unable or unwilling to feed adequately. Complications include vomiting, tube displacement, peristomal infection, and abscess formation, appearing in 13- 71% of cases, while major complications are less commonly described.

Case presentation

A three-year-old, male neutered Standard Poodle with a history of epilepsy was referred for lethargy, anorexia, and vomiting associated with a suspicion of an intestinal foreign body. Clinical examination, hematology, and serum biochemistry were unremarkable. Abdominal palpation revealed a 5 cm round mesogastric mass, painful on palpation. Abdominal radiographs confirmed the intestinal foreign body and exploratory celiotomy was performed. An esophagostomy tube was placed to ensure fast and adequate feeding. Proper tube positioning was radiographically confirmed.

Postoperative therapy included antibiotics, analgesics, antiemetics, and infusion of prokinetics. Six days after the esophagostomy tube placement, signs of peristomal infection were noted. Clinical signs and laboratory results worsened progressively, suggesting the development of systemic inflammatory response syndrome. Due to the large amount of purulent dark-red discharge with presence of food at the tube site, the esophagostomy tube was removed. Esophagoscopy revealed a 3 cm-long defect at the tube entrance in the cervical portion of the esophagus, and surgical repair ensued. Thoracic ultrasonography showed bilateral pleural effusion. Samples were taken for microbiology and cytology, followed by chest tube placement. Cytology revealed a septic effusion, while bacteriology discovered multi-resistant *Klebsiella pneumoniae*. Thoracic lavage was performed daily via chest tubes, and antibiotics were adjusted according to antibiogram. Due to continuous discharge and peristomal tissue infection, wound dehiscence developed. Surgical closure of esophagus was repeated, leading to resolution of the infection. The dog made a complete recovery and was discharged after fifteen days of hospitalization.

New / Unique information

To the authors' knowledge, development of pyothorax after esophagostomy tube placement has not been previously described. We assume that, despite the use of prokinetics, regurgitation was still present, as evidenced by food coming out of the tube site. That led to local infection, difficulties in esophageal healing, and pyothorax development, probably due to leakage of contents along the wall of the esophagus into the thorax.

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Short-term severe polyuria responsive to vasopressin following hypoglycaemia and hypotension in a cat

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Background

Central diabetes insipidus (CDI) is a rare disorder in cats which is characterized by a complete or partial deficiency of ADH. Diabetes insipidus in cats has been reported in association with trauma, congenital conditions, or neoplasia. Findings include low urine osmolarity, hyposthenuria, increased serum osmolarity, hypernatremia, and polyuria.

Case presentation

An 8-month-old, male neutered, domestic short-haired cat, presented to our hospital for further investigations of acute vomiting. The patient was tachycardic (230 bpm), normotensive (SYS 116 mmHg), and hypoglycemic (2.1 mmol/L [RI 5.5-10.27 mmol/L]) on presentation. Following initial stabilization, an abdominal ultrasound revealed a jejunal foreign body which was removed via enterotomy. Intraoperatively, a dopamine CRI was required to maintain MAP > 60 mmHg. On recovery, the patient was found to be severely hypoglycemic (1.3 mmol/L [RI 4.4-6.6 mmol/L]) despite previous supplementation. In the first 24-hour post-surgery, despite normalization of the cardiovascular status and euglycemia, the patient developed progressive polyuria (up to 14 ml/kg/h). This was associated with neurological signs suggestive of diffuse brain disease (non-ambulatory, obtunded, mydriatic pupils, absent bilateral PLR and menace response) and absence of azotemia or signs of overhydration. During the first four days of hospitalization, any attempt to decrease intravenous fluid therapy was associated with the development of hypotension, weight loss, and clinical dehydration. USG during this time was 1.005-1.010 and failed to increase during fluid challenges. Due to a suspicion of CDI, the patient was administered desmopressin (1 mcg/cat SC) on day 5 of hospitalization. Following this, his urinary output decreased (from 14 to 6 ml/kg/h), and his weight increased (from 3 to 3.2 kg) within 4 hours. He required a total of 4 doses of desmopressin during the 13 days of hospitalization, but no further doses since discharge (UOP 3 ml/kg/hr). At follow-up 3 months later, his neurological signs and polyuria had resolved, and his USG was >1.050.

New/Unique information

In this case, a presumptive diagnosis of CDI was supported by clinical progression, neurological signs, and response to desmopressin. To our knowledge, reversible diabetes insipidus following diffuse brain injury secondary to hypotension and hypoglycemia is not reported in veterinary patients.

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Emergency case of dog intoxication by the fire salamander

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Background

Spread across Europe, the fire salamander (*Salamandra salamandra*) is an amphibian capable of using specialized skin glands to secrete toxic alkaloid substances as a defensive mechanism. It is known that these compounds, when in contact with mammals' mucous membranes, can cause severe neurological implications that may lead to seizures, respiratory paralysis, and death. Despite that knowledge, and even though salamanders are a common sight in wet green spaces like public parks and gardens, very few intoxication cases have been reported (both formally and empirically).

Case presentation

A 5-year-old male mixed breed dog was admitted to AniCura CHV Porto Hospital Veterinário presenting a seizure cluster after reportedly having bitten a salamander in a public park in Porto, Portugal. The dog's owner photographed the salamander, which had the unmistakable distinctive black-yellow skin. Upon controlling the cluster using propofol intravenously *ad effectum*, a thorough examination was performed, and the dog was hyperthermic (42.0ºC) and with hyperlactatemia [18.0 mmol/L (<2 mmol/L)], which were promptly addressed using cooling techniques and fluid therapy. Haematomas were easily formed by iatrogenic puncture. Blood work revealed no clinically significant changes except for the blood coagulation times, which were increased [PT 19s (14-19s), aPTT 121.6s (75-105s)]. Since secondary disseminated intravascular coagulation was suspected, a plasma transfusion was performed. Lactate level normalized (1.7 mmol/L) after stabilization. Two days later, the dog presented a normal mental status and physical examination, with discrete haematochezia. Despite the altered blood coagulation tests (PT 22.5s, aPTT 120.3s), the dog was discharged from the hospital. On a follow-up appointment three days later, it was reported that the dog was completely normal at home. Physical examination was unremarkable and blood coagulation test results normalized (PT 16.4s, aPTT 77.8s).

New / Unique information

Very little information has been published as it is believed that most of the dogs affected are not presented to the hospital on time or they die soon after arrival. This case report is unique because the dog completely recovered and it brings unique information regarding blood coagulation times and lactate levels and how they are affected, directly or indirectly, by the fire salamander toxins.

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Broken tail syndrome in a cat trapped in dryer machine

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Background

In dogs, limber tail is a condition that makes their tail appear as broken. It is typically caused by overworking tail muscles (acute caudal myopathy). Here we describe a case of 'broken tail Syndrome' in a cat, after trapping in a dryer machine.

Case presentation

A domestic, shorthair, 9 years old, neutered male, outdoor/indoor cat, presented in an emergency setting after being trapped in a dryer machine for almost one hour. The patient was in severe respiratory distress with increased respiratory effort and air-hunger position. Crackles were audible on thoracic auscultation. Rectal temperature was 36.5 °C and heart rate was 150 bpm with sinus rhythm. Cat was alert and responding. A mild ambulatory paraparesis was observed, with a good sensitivity on both limbs. Spinal reflexes and femoral pulses were normal. A complete inability to move the tail, with normal sensitivity and pain evocation, was also detected. Thoracic and abdominal POCUS were negative for pleural or abdominal effusions. No vertebral luxation, fractures, or dislocations (spinal, coccygeal vertebrae) were observed on radiographic examination. The cat presented sialorrhea and multiple small cuts and ulcer-like lesions were observed on the tongue. The venous blood gas analysis showed a normal acid/base status with hyperglycemia (255 mg/dL) and slight hyperlactatemia (2.7 mmol/L). CBC, total solids, BUN, and creatinine were normal. The cat was hospitalized and the following therapies were started: Ringer lactate 3ml/kg/h IV, buprenorphine 20µg/Kg IV q8h, maropitant 2mg/kg IV q24h, N-Acetylcysteine 70mg/Kg IV q12h, oral 0.21%chlorhexidine gel locally applied q6h. The patient was discharged after three days uneventfully, with a slight improvement in tail movement. After one week of rest at home, the cat was able to move the tail with a complete resolution of signs.

New/Unique Information

To the author's knowledge, 'limber tail syndrome' is not documented in cats. In this case, we assumed an acute myopathy, due to the 'fatigue' of keeping in balance during the rotation of the machine within the absence of tail, pelvic, or spinal fractures or luxations. This, in addition to high temperature followed by cooling, could explain the "broken" (reversible) tail condition.

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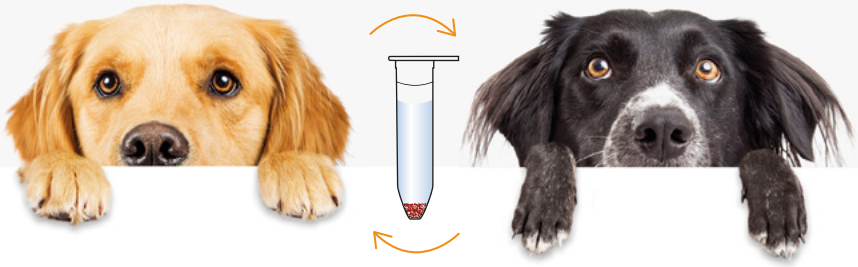

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Alvedia

Alvedia is a company specialized in the veterinary diagnostics field with their most advanced and innovative immunochromatographic technology for Blood Typing. With a committed quality control department that tests and validates all products before supplying to more than 70 worldwide distributors, Alvedia aims to manufacture products of the highest standard from France. Alvedia is also providing a NEW Gel Test technology known as the Gold standard in the human field. The Gel Test technology will allow the vets to Crossmatch without any washing step procedure with a very high sensitivity. The Gel technology will allow you to perform a fast and reliable Direct Antiglobulin Test (Or Coombs Test) without washing the red blood cells. Alvedia is also very involved in supporting numerous research projects that could have significant benefits in the veterinary transfusion field.
www.alvedia.com

BSA

The Animal Blood bank provides quality blood components, as a treatment aid in clinical practice, following inhouse good practice and strict quality criteria guided by specialized human medicine protocols. Our mission is to provide a significant platform connecting donor animals and critical patients in veterinary clinics and hospitals who require blood transfusions. The Animal Blood Bank is a specialized organization that aims to help cats and dogs who have a critical illness and for whom a blood transfusion can be life saver. Our gool is to help as many animals as possible, ensuring none of them dies due to difficult access to blood transfusions or lack of reliable sources of blood componentes. BSA has haemocomponent safety and donors welfare high standards.
www.bsanimal.co.uk

Nova Biomedical

Nova manufactures point of care meters for glucose, ketone, creatinine, lactate, hemoglobin and hematocrit testing, and the Stat Profile Prime Plus® VET blood gas/critical care analyzer that provides a comprehensive menu of blood gases, electrolytes, chemistry, and hematology for veterinary emergency or critical care testing. Prime Plus VET measures pH, PCO2, PO2, Na, K, Cl, iCa, iMg, Glu, Lac, BUN, Creatinine, Hct, Hb, SO2%, and Co-oximetry, and features ZERØ Maintenance cartridge technology, simple operation, results in about a minute, and low-cost testing. Prime Plus VET provides improved practice profitability through in-house diagnostics.
www.novabiomedical.com

Unavets

UNAVETS is a leading veterinary healthcare platform with over 105 practices and labs and more than 1,000 people focused on veterinary healthcare excellence, training, and continued innovation. The group is comprised of reference/specialized centers, 24-hour hospitals, primary opinion clinics and veterinary offices, which guarantee a complete range of services. UNAVETS is distinguished by its strong investment in scientific clinical training, equipment, facilities and business support for clinics as well as its commitment to pushing the boundaries in favor of advanced veterinary care. The group also invests in veterinary-adjacent verticals, which are directly relevant to the veterinary healthcare sector.

Our commitment is to our people and is evident in how we support our practices and clinical team members:

- We have over 60 people in our corporate headquarters focused on HR, Training, Operations, Finance & Accounting, Marketing, IT and Network Development.
- UNAVETS also offers company wide health insurance and other benefits to all team members.
- The group continues to make significant investment in Clinical training and professional development, as well as access to OneAcademy, an extensive clinical training platform powered by UNAVETS.

UNAVETS' growth strategy began by focusing on Iberia, but has since expanded to other European countries and the United States, as well as adjacent verticals synergistic with veterinary healthcare. In order to fund its continued growth strategy, the Company has raised significant capital from financial partners Oaktree Capital and Ares Management.
www.unavets.com

VECCS

VECCS was founded in 1974 to promote the advancement of knowledge and high standards of practice in veterinary emergency medicine and critical patient care. To fulfill this purpose, we host a membership community, medical journal, monthly webinars, facility certification, wellness initiatives, a charitable arm and two annual educational conferences that offer progressive, innovative, and comprehensive CE to members and non-members alike.
www.veccs.org

Vet's Choice Radiology

Vets Choice Radiology is a full service teleradiology company with board certified radiologists and highly experienced staff dedicated to supporting veterinary hospitals. Our team is committed to offer the premier experience through guaranteed turnaround times 24/7/365 as well going above and beyond for customer satisfaction.
www.vetschoiceradiology.com

Woodley

Woodley Equipment Company is a market leading supplier of specialist laboratory equipment, diagnostic and critical care products to the veterinary industry worldwide. Woodley are committed to helping veterinary practices and laboratories provide the very best patient care by supplying and supporting the latest laboratory diagnostic products, supported by a focussed Customer Service and Warehouse team. Woodley also supplies worldwide veterinary distributors with laboratory diagnostic products including the InSight Veterinary Lab System (haematology, chemistry and immunoassay), coagulation, critical care, blood gas/electrolyte analysers and handheld meters Woodley maintain their position by a team of 14 Biomedical Scientists committed to Product Development and Technical Support. Passionate About Laboratory Diagnostics.
www.woodleyequipment.com

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AND CRITICAL CARE CONGRESS