21ST EVECC CONGRESS
30 MAY - 1 JUNE 2024
GOTHENBURG-SWEDEN

WWW.EVECC-CONGRESS.ORG

PROCEEDINGSBOOK

EVECC

EUROPEAN VETERINARY EMERGENCY AND CRITICAL CARE CONGRESS

ECVECC

ROYAL CANIN

B|BRAUN SHARING EXPERTISE

BSA ANIMAL BLOOD BANK

VetsNow

Table of contents

VECCUS Symposium, Wednesday 29 May 2024 8
COLOR DOPPLER AND POWER DOPPLER IMAGING TO HELP DIFFERENTIATE SUBPLEURAL CONSOLIDATIONS IN DOGS AND CATS 9
USE OF ULTRASOUND IN ATELECTASIS AND LUNG RECRUITMENT 12
WHAT'S BEHIND THE CURTAIN (SIGN)? 15
KNOBOLOGY; WHY BEING ABLE TO 'DRIVE' YOUR MACHINE IMPROVES IMAGE OPTIMISATION AND HELPS TO SAVES LIVES 19
CASE-BASED POCUS IN ICU PATIENTS 22
MASTERING THE WAVES: COMPETENCY ASSESSMENT IN POINT-OF-CARE ULTRASOUND 25
POCUS APPLICATION IN THE MANAGEMENT AND MONITORING OF CONGESTIVE HEART FAILURE 27
URETERAL OBSTRUCTIONS 30
Main Stream, Thursday 30 May 2024 33
MANAGEMENT OF ACUTE HEART FAILURE 34
MANAGEMENT OF ARRHYTHMIAS 37
MANAGING SEVERE WOUNDS IN THE EMERGENCY CLINIC 41
IS IT SURGICAL? INTERPRETING ABDOMINAL RADIOGRAPHS IN THE EMERGENCY SETTING 44
ELEVATING YOUR EX-LAP: HOW TO GET THE MOST FROM YOUR PROCEDURE 47
IMMEDIATE VS DELAYED SURGERY IN THE ER 49
Advanced Stream, Thursday 30 May 2024 53
CARDIOVASCULAR-RENAL AXIS DISORDERS 54
ELECTROLYTES : WHY NOT ON URINE? 57
KIDNEY REPLACEMENT MODALITIES: WHAT ARE THE OPTIONS? 60
DIAGNOSIS AND MANAGEMENT OF UROABDOMEN IN DOGS AND CATS 62
MANAGING THROMBOSIS: APPLYING THE CURATIVE GUIDELINES 65
VISCOLEASTIC (TEG) - GUIDED TRANSFUSIONS: WHAT IS THE EVIDENCE? 69
URGENT REVERSAL OF ANTICOAGULATION 72
CHALLENGING COAGULATION CONUNDRUMS 74
Nurse & Tech Stream, Thursday 30 May 2024 78
THE ART OF TRIAGE 79
MDB (MINIMUM DATA BASE) AS SIMPLE AS 1-2-3 83

AN INTERACTIVE CASE DISCUSSION: THE ACUTE ADDISONIAN PATIENT 88
TOXICOLOGICAL EMERGENCIES 91
WHEN SUGAR TURNS SOUR: FELINE DKA 96
LIGHTS, CAMERA, ANAESTHESIA! NAVIGATING COMPLICATIONS 99
GDV PATIENT CARE: PREPARATION TO RECOVERY WITH A TWIST! 102
BEYOND THE BLOOD – NURSING CONSIDERATIONS FOR THE HAEMOABDOMEN 104
Main Stream, Friday 31 May 2024 107
COMPLICATIONS OF INTRAVENOUS FLUID THERAPY: PREVENTION, MONITORING AND INTERVENTION 111
DAMAGE CONTROL! MANAGING THE ACUTELY HEMORRHAGING TRAUMA PATIENT 114
FELINE ARTERIAL THROMBOEMBOLISM 117
THE FELINE TRAUMA PATIENT 120
WHAT IS NEW FOR THE MANAGEMENT OF FELINE URETHRAL OBSTRUCTION? 125
FELINE CIRCULATORY SHOCK 128
Advanced Stream, Friday 31 May 2024 133
HYPERTENSIVE EMERGENCIES 134
USING CRITICAL CARE ECHOCARDIOGRAPHY (CCE) TO GUIDE THERAPY IN CRITICALLY ILL PATIENTS 139
MULTIDISCIPLINARY APPROACH TO PULMONARY HYPERTENSION 141
SEPSIS CONSENSUS DEFINITIONS 145
IMMUNOTHROMBOSIS: A NOVEL THERAPEUTIC TARGET? 150
SEPTIC SHOCK- DECISION MAKING FROM THE TRENCHES 156
Nurse & Tech Stream, Friday 31 May 2024 161
MANAGEMENT OF HOSPITALISED INFECTIOUS FELINE PATIENTS 162
THE TECHNICIAN'S GUIDE TO BLOCKED CATS 168
SAVING NINE LIVES: FELINE BLOOD TRANSFUSIONS 172
AND THE BEAT GOES ON 175
MEDICAL MATH FOR THE VETERINARY TECHNICIAN 177
SOOTHING THE STORM: SEDATION STRATEGIES FOR ICU PATIENTS 181
AIRWAY MANAGEMENT IN THE ICU 184
Resident Stream, Saturday 31 May 2024 186
THE THEORY BEHIND YOUR EMERGENCY SURGICAL PROCEDURES 187

FROM MOLECULES TO MEDICINE: CELLULAR PERSPECTIVES ON VASOPRESSORS AND INOTROPES IN THE ICU 189
PHYSIOLOGY OF CARDIAC TAMPONADE 192
THE PHYSIOLOGY OF HAEMODYNAMIC MONITORING 194
Main Stream, Saturday 1 June 2024 199
EMERGENCY MANAGEMENT OF STATUS EPILEPTICUS AND CLUSTER SEIZURES 200
OSMOTHERAPY IN BRAIN OEDEMA - CURRENT EVIDENCE OF PRACTICE 203
DISORDERS OF AMBULATION IN THE ER, PART I: OBSERVATION, EXAM NUANCES AND SORTING OUT SYSTEMS 206
DISORDERS OF AMBULATION IN THE ER, PART II: DIAGNOSTIC AND THERAPEUTIC MANAGEMENT 209
DISORDERS OF AMBULATION IN THE ER, PART III: VIDEO CASE EXTRAVAGANZA! 211
Advanced Stream, Saturday 1 June 2024 214
ERAS® FOR VETERINARY EMERGENCY AND CRITICAL CARE: TIME FOR ACTION 215
PANS : A NEW MEDICAL ENTITY ? 218
VETERINARY ARDS AND REFRACTORY HYPOXEMIA- A REVIEW 220
GARY STAMP MEMORIAL LECTURE: HIGH FLOW OXYGEN THERAPY: BEYOND TREATMENT OF HYPOXEMIA 226
GARY STAMP MEMORIAL LECTURE: VENTILATOR WAVEFORMS: WHAT CAN I LEARN FROM THEM? 229
Nurse & Tech Stream, Saturday 1 June 2024 232
CALM YOUR RAAS DOWN! – UNDERSTANDING THE RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM AND ITS INHIBITORS 233
WHEN THE KIDNEYS GO ON STRIKE: THE GREAT NEPHRON REBELLION 236
PATIENT HANDOVERS 239
WIDENING OUR HORIZONS: HOW VN'S ARE LOOKING TOWARDS HUMAN CENTRED NURSES TO IMPROVE PATIENT CARE 241
NEONATAL NURSING 243
Leadership Stream, Saturday 1 June 2024 247
WELLNESS ACTION PLAN: HOW TO KEEP YOUR TEAM HAPPY AND HEALTHY 248
NON-TECHNICAL SKILLS – HOW CAN WE INCORPORATE THIS INTO RESIDENCY TRAINING? 253
PSYCHOLOGICAL SAFETY 255
Oral Abstracts, Original Study, Thursday 30 May 2024 258

COMPARISON OF RADIOGRAPHS AND ULTRASOUND FOR DIAGNOSIS OF RIB FRACTURES IN CANINE CADAVERS 259

COMPARISON OF POINT-OF-CARE ULTRASOUND AND BLINDED ANATOMICAL LANDMARK THORACOSTOMY TUBE INSERTION IN CADAVER DOGS 261

A LOW-COST SONOGRAPHIC CANINE SIMULATOR FOR PERICARDIOCENTESIS TRAINING 263

POINT-OF-CARE ULTRASOUND OF GASTRIC ANTRUM TO ESTIMATE GASTRIC CONTENT AND VOLUME IN CRITICALLY ILL DOGS 265

NASOGASTRIC FEEDING TUBE POSITION CONTROLLED USING POINT-OF-CARE ULTRASOUND COMPARED TO X-RAYS: A PRELIMINARY STUDY ON 39 CASES 267

THE EFFECT OF PACKED RED BLOOD CELL TRANSFUSIONS ON AMMONIA LEVELS IN DOGS 269

INFLUENCE OF ESTIMATED COSTS ON EUTHANASIA DECISION FOR CATS PRESENTED WITH TRAUMA TO AN EMERGENCY CENTER IN NEW ZEALAND: A RETROSPECTIVE ANALYSIS 271

INTRAVENOUS ADMINISTRATION OF VITAMIN K1 IN DOGS AND CATS PRESENTED FOR ANTICOAGULANT RODENTICIDE POISONING: A RETROSPECTIVE STUDY OF 59 CASES 273

OUTCOME PREDICTION AND SURVIVAL IN CRITICALLY ILL DOGS WITH ACUTE KIDNEY INJURY UNDERGOING CONTINUOUS RENAL REPLACEMENT THERAPY 275

PRELIMINARY EVALUATION OF THE OXYGEN RESERVE INDEX AS A SURROGATE FOR THE ARTERIAL PARTIAL PRESSURE OF OXYGEN IN ANESTHETIZED DOGS 276

EVALUATION OF SERUM MIRNA EXPRESSION AS BIOMARKERS OF BRACHYCEPHALIC OBSTRUCTIVE AIRWAY SYNDROME SEVERITY IN FRENCH BULLDOGS 278

DETERMINATION OF HIDDEN FLUID VOLUME IN CANINE INTENSIVE CARE PATIENTS - HIDDEN FLUIDS IN CRITICALLY ILL DOGS: THE HEROS TRIAL 280

EFFECTS OF ACUTE INTRAVASCULAR VOLUME OVERLOAD ON ENDOTHELIAL GLYCOCALYX THICKNESS IN HEALTHY CATS – PRELIMINARY RESULTS 282

Oral Abstracts, Nurse & Technician Case Reports, Friday 31 May 2024 284

FROM FLAMES TO CARE: MECHANICAL VENTILATION IN A DOG AFTER SMOKE INHALATION 285

TEMPORARY TRACHEOSTOMY TUBE MANAGEMENT IN A DOG WITH BOAS 286

PUPPY WITH COMPLEX TRAUMA TO MULTIPLE BODY PARTS AND SYSTEMS CAUSED BY DOG ATTACK 287

Poster Abstracts 288

Original Study 288

PREVALENCE OF FELV PROVIRUS IN A FELINE BLOOD DONOR POPULATION: A COMPREHENSIVE ANALYSIS 289

NOREPINEPHRINE INFUSION DID NOT IMPACT SUBLINGUAL MICROCIRCULATION IN A PORCINE HEMORRHAGIC SHOCK MODEL 291

PUPILLOMETRY AND PARASYMPATHETIC TONE ACTIVITY INDEX TO DETECT SYMPATHETIC NERVOUS SYSTEM ACTIVATION DURING HEMORRHAGE 293

EFFECT OF FELINE MULTIPLE DONATIONS ON IRON CONCENTRATION AND ITS RESERVES 295

FELINE BLOOD DONATION: DESCRIPTION AND ADVERSE REACTIONS FROM 29201 DONATION EVENTS BETWEEN 2019 AND 2023 297

EVALUATION OF HEMOSTASIS INCLUDING VISCOELASTIC TESTING IN A CANINE HEMORRHAGIC SHOCK MODEL 299

CLINICAL AND POINT-OF-CARE ULTRASOUND FINDINGS IN A CASE SERIES OF CATS WITH HIGH-RISE SYNDROME 301

VALIDATION OF A HUMAN FRET-VWF73 ASSAY FOR ASSESSMENT OF ADAMTS-13 ACTIVITY IN HEALTHY DOGS 303

COMPARISON OF ULTRASOUND PROBE LOCATION AND SONOGRAPHIC FINDINGS USED FOR THE EVALUATION OF PNEUMOTHORAX IN CANINE PATIENTS: A CADAVER PILOT STUDY 305

BLOOD COMPONENT CONSUMPTION PROFILE OF VETERINARY CENTERS ORDERING FROM A COMMERCIAL BLOOD BANK 307

CLINICAL FINDINGS AND BLOOD GAS ANALYSIS IN COMPANION RABBITS WITH GASTRO-INTESTINAL STASIS: A RETROSPECTIVE STUDY OF 43 CASES 309

DIFFERENCE OF CORRECTED SERUM CHLORIDE AND MEASURED SERUM CHLORIDE IN THE DIFFERENTIATION BETWEEN CARDIOGENIC AND NON-CARDIOGENIC EFFUSIONS IN DOGS 311

MANAGEMENT OF GLASS INGESTION IN 21 DOGS 313

CARDIOVASCULAR EFFECTS OF THREE DIFFERENT INTRAMUSCULAR DOSES OF MEDETOMIDINE-VATINOXAN IN HEALTHY DOGS INDUCED WITH PROPOFOL AND ANESTHETIZED WITH SEVOFLURANE 315

VETERINARY ONCOLOGIC PATIENTS IN ICU: PREVALENCE AND PROGNOSTIC FACTORS 317

A SYSTEMATIC LITERATURE SEARCH AND STRUCTURED REVIEW ON THE MEASUREMENT OF ANTIMICROBIAL RESISTANCE AND SUSCEPTIBILITY TESTING IN URINE SAMPLES OF DOGS AND CATS 318

EXAMINING THE IMPACT OF BLOOD DONATION FREQUENCY ON THE SEDATION DOSAGE ADMINISTERED TO FELINE DONORS 320

RANGE OF REFERENCE FOR IONIZED MAGNESIUM AND IMPACT OF INTRAOPERATIVE FLUID THERAPY IN HEALTHY DOGS 322

ANCILLARY TREATMENTS WITH THROMBOLYTICS OR MEDICATIONS AIMED AT IMPROVING FUNCTIONAL RECOVERY OR BOTH IN ACUTE FELINE AORTIC THROMBOEMBOLISM 324

Poster Abstracts 326

Case Reports 326

ACUTE LINGUAL ARTERY THROMBOSIS IN A CAT WITH HYPERTROPHIC CARDIOMYOPATHY 327

REFRACTORY HYPERKALEMIA IN A CAT WITH PRESUMPTIVE TYPE 4 RENAL TUBULAR ACIDOSIS AND PSEUDOHYPOALDOSTERONISM DUE TO SUSPECTED LIPID-INDUCED RENAL PAPILLARY NECROSIS 328

ACUTE RESPIRATORY DISTRESS SYNDROME IN A DOG: MANAGING LUNG COLLAPSE WITH AIRWAY PRESSURE RELEASE VENTILATION 330

SEVERE ANEMIA CAUSED BY ORAL LIMNATIS NILOTICA LEECH INFESTATION IN A DOG 332

STATUS EPILEPTICUS AFTER ELECTROCUTION INJURY IN A DOG 334

DEXMEDETOMIDINE INFUSION IN THE MANAGEMENT OF A REFRACTORY SEPTIC SHOCK IN A DOG AFFECTED BY PROTEIN LOSING ENTEROPATHY (PLE) 335

SUCCESSFUL OPEN-CHEST CARDIOPULMONARY RESUSCITATION IN A DOG WITH PYOTHORAX 337

ACUTE RESPIRATORY DISTRESS SYNDROME AND TRICUSPID VALVE THROMBUS SECONDARY TO PULMONIC BALLOON VALVULOPLASTY TREATED WITH OXYGEN CAGE THERAPY AND HEPARIN 339

PRESUMPTIVE SEPSIS-ASSOCIATED ENCEPHALOPATHY IN A CAT AND A DOG 341

UPPER AIRWAY OBSTRUCTION FOLLOWING RODENTICIDE INTOXICATION IN THREE DOGS 342

GENERALIZED TETANUS IN A CAT AND ITS MANAGEMENT WITH A PERCUTANEOUS ENDOSCOPIC GASTROSTOMY TUBE 343

FELINE URETHRAL OBSTRUCTION SECONDARY TO A FOREIGN BODY 345

POSTOPERATIVE INTERMITTENT ROPIVACAINE ADMINISTRATION VIA QUADRATUS LUMBORUM PLANE CATHETERS IN A DOG AFTER UNILATERAL LAPAROTOMIC ADRENALECTOMY 346

SPONTANEOUS LUNG LOBE TORSION RESULTING IN A SADDLE THROMBUS IN A 2-YEAR-OLD SIGHTHOUND 348

ERECTOR SPINAE PLANE BLOCK IN A DOG WITH ACUTE PANCREATITIS 349

HAND GRANADE BLAST INJURIES IN A DOG DURING ACTIVE SERVICE IN A WAR ZONE 350

NONCARDIOGENIC ARTERIAL THROMBOEMBOLISM IN A CAT FOLLOWING AORTIC AIRGUN PELLET INJURY 352

PRESENTATION OF HYPOALDOSTERONISM IN A DOG 354

DIABETIC KETOALKALOSIS IN A DOG: A CASE REPORT 356

ACUTE COLLAPSE AND ARDS SECONDARY TO A MULTIPLE AUTOIMMUNE SYNDROME IN A DOG DIAGNOSED WITH ATYPICAL HYPOADRENOCORTICISM, HYPOTHYROIDISM AND POLYMYOSITIS 358

WILDLIFE-INDUCED POLYTRAUMA IN DOGS: A CASE SERIES OF NINE WILD BOAR ENCOUNTERS 360

NEUROLOGICAL DYSFUNCTION AND SEVERE HYPOKALIEMIA IN A CAT AFTER INGESTION OF RHODODENDRON SPP 361

CHYLOABDOMEN IN A DOG WITH ACUTE PANCREATITIS 363

VECCUS Symposium, Wednesday 29 May 2024

COLOR DOPPLER AND POWER DOPPLER IMAGING TO HELP DIFFERENTIATE SUBPLEURAL CONSOLIDATIONS IN DOGS AND CATS

Michał Gajewski [1] Katarzyna Kraszewska [1]

[1] Vet Lus Expert s.c., Warsaw, Poland

Learning objectives:

- To present general principles of applying the color doppler modality in lung ultrasound of dogs and cats

- To describe various types of vascular patterns within subpleural consolidations found in dogs and cats

- To present vascular criteria that help in the differentiation of pathologic processes in the lungs

Proceeding:

Non-cardiac thoracic ultrasonography is increasingly used in veterinary medicine. This imaging modality has been thoroughly studied in human medicine and is considered an important tool for increasing the diagnostic accuracy in dyspneic patients. According to newly published guidelines the first diagnostic approach in patients with acute dyspnea should include thoracic ultrasound. Lung ultrasound (LUS) is important in many fields, from emergency and intensive care to cardiology, internal medicine, pediatrics, and neonatology. Guidelines and publications for adults and children suggest that color doppler analysis can be beneficial to perform when available.

In veterinary medicine LUS is a part of POCUS in various emergency protocols (VETBLUE Ò, TFASTÒ or CALGARY PLUS). The assessment of consolidations in emergency protocols is based on their shape and presence or absence of vertical artefacts, without applying Doppler analysis. Color Doppler analysis enables the assessment of vascular criteria within consolidations, which are one of three criteria used in human internal medicine to describe consolidated lung tissue on LUS. The other two are the pleural and parenchymal criteria. Vascular analysis of the consolidation impacts subsequent diagnostic and therapeutic decisions.

The VetLus protocol utilizes a horizontal sliding technique with the transducer placed at three different vertical locations on each side of the thorax at levels in line with: the middle of the scapula below the rib heads and epaxial muscles (dorsal line); the shoulder joint (at the level of the heart base, middle line); and just dorsal to the sternebrae (ventral line).

Consolidations are described based on their size, shape, margins, echogenicity, and three additional criteria: parenchymal (presence of bronchograms), vascular (using color Doppler sonography), and pleural (presence or absence of lung sliding, effusion, and pleural line irregularities) as defined in recent human lung ultrasound guidelines. The color Doppler analysis is based on the presence or absence of blood flow

within the consolidation, the pattern of blood vessels in the vascularization, and sometimes also the profile of blood flow within the vessel.

Five vascular patterns can be observed within consolidated lung tissue - anatomical (tree-like), residual, chaotic vascularization, penetration of blood vessels from the chest wall into the thoracic cavity, and blood flow amputation (referred to as “vascular sign”). Anatomical tree like vascularization is present in pneumonia. Residual vascularization was found in atelectasis. Residual vascularization or chaotic neovascularization is present in neoplastic consolidations. The fourth type (penetration) is present when a lung neoplasm infiltrates the chest wall, when the intercostal arteries penetrate the consolidation. The last observed patten is a “*vascular sign*.” The “*vascular sign*” is believed to result from the occlusion of a vessel by embolic material. Abrupt cessation of blood flow at the “tip” of the consolidation can be seen using color Doppler. The consolidation is usually triangular or basket-shaped and hypoechoic (a.so called the “wedge sign”).

Color Doppler analysis of vascular patterns withing consolidated lung tissue holds promise as a valuable additional criterion, allowing a more targeted approach to patients with consolidations on LUS and should be included in a comprehensive assessment of patients with dyspnea and lung pathologies.

References:

Pelchat J, Chalhoub S, Boysen SR. The use of veterinary point-of-care ultrasound by veterinarians: A nationwide Canadian survey. Can Vet J. 2020 Dec;61(12):1278-1282.

Demi L, Wolfram F, Klersy C, De Silvestri A, Ferretti VV, Muller M, Miller D, Feletti F, Welnicki M, Buda N, Skoczylas A, Pomiecko A, Damjanovic D, Olszewski R, Kirkpatrick AW, Breitkreutz R, Mathis G, Soldati G, Smargiassi A, Inchingolo R, Perrone T. New International Guidelines and Consensus on the Use of Lung Ultrasound. J Ultrasound Med. 2023 Feb;42(2):309-344. Epub 20220822. doi:10.1002/jum.16088.

Buda N, Kosiak W, Welnicki M, Skoczylas A, Olszewski R, Piotrkowski J, Skoczynski S, Radzikowska E, Jassem E, Grabczak EM, Kwasniewicz P, Mathis G, Toma TP. Recommendations for Lung Ultrasound in Internal Medicine. Diagnostics (Basel). 2020 Aug 16;10(8). The authors declare no conflict of interest. Epub 20200816. doi:10.3390/diagnostics10080597.

Dietrich CF, Buda N, Ciuca IM, Dong Y, Fang C, Feldkamp A, Jungert J, Kosiak W, Mentzel HJ, Pienar C, Rabat JS, Rafailidis V, Schrading S, Schreiber-Dietrich D, Jaworska J. Lung ultrasound in children, WFUMB review paper (part 2). Med Ultrason. 2021 Nov 25;23(4):443-452. Epub 20210303. doi:10.11152/mu-3059.

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 Nov;51(6):1125-1140. eng. Epub 20210914. doi:10.1016/j.cvsm.2021.07.012.

Kraszewska K, Gajewski M, Boysen S. Case report: Application of color Doppler sonography for the assessment of pulmonary consolidations in a dog. Frontiers in Veterinary Science. 2023;10. Epub 08.12.2023. doi:10.3389/fvets.2023.1275929.

Yang P. Applications of colour Doppler ultrasound in the diagnosis of chest diseases. Respirology. 1997 Sep;2(3):231-8. doi:10.1111/j.1440-1843.1997.tb00083.x. Cited in: Pubmed; PMID 9400685.

Yuan A, Yang PC, Chang DB. Pulmonary infarction: use of color Doppler sonography for diagnosis and assessment of reperfusion of the lung. AJR Am J Roentgenol. 1993 Feb;160(2):419-20. doi:10.2214/ajr.160.2.8424369.

Yuan A, Yang PC, Lee L, Wu HD, Kuo SH, Luh KT, Chen WJ, Lin FY. Reactive pulmonary artery vasoconstriction in pulmonary consolidation evaluated by color Doppler ultrasonography. Ultrasound Med Biol. 2000 Jan;26(1):49-56. doi:10.1016/s0301-5629(99)00118-0.

Sripathi S, Mahajan A. Comparative study evaluating the role of color Doppler sonography and computed tomography in predicting chest wall invasion by lung tumors. J Ultrasound Med. 2013 Sep;32(9):1539-46. doi:10.7863/ultra.32.9.1539.

Lisciandro GR, Puchot ML, Gambino JM, Lisciandro SC. The wedge sign: A possible lung ultrasound sign for pulmonary thromboembolism. J Vet Emerg Crit Care (San Antonio). 2022 May 6. eng. Epub 20220506. doi:10.1111/vec.13208.

USE OF ULTRASOUND IN ATELECTASIS AND LUNG RECRUITMENT

Angela Briganti [1]

[1] Department of Veterinary Sciences, University of Pisa, Pisa, Italy

Learning objectives:

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

Proceeding:

Atelectasis and Recruiting Maneuvers

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

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

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

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

Recently the use of ultrasonography has been demonstrated to be useful to recognize atelectasis early and to guide the clinician during recruitment maneuvers.

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

Several human studies have shown that lung ultrasound (LUS) and the use of specific scoring systems are able to identify and to measure the extent of atelectasis. The comparison between preanesthetic and postanesthetic LUS and the use of atelectasis scores can detect even minimal alterations in lung aeration. Real time ultrasound lung monitoring during RM allows identification of the opening pressure value of PEEP; in this way it is possible to guide a stepwise incremental PEEP recruiting maneuver and cease the increasing pressure at the value necessary to re-open the alveoli, thus potentially reducing the possibility of lung overinflation. During mechanical ventilation of critical patients, the repeated use of LUS can help the clinician to guide the ventilation mode, the use of PEEP, and of FiO2 and to promptly identify atelectasis areas.

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

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

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

References:

Acosta, C. M., Maidana, G. A., Jacovitti, D., Belaunzarán, A., Cereceda, S., Rae, E., Molina, A., Gonorazky, S., Bohm, S. H., & Tusman, G. (2014). Accuracy of transthoracic lung ultrasound for diagnosing anesthesia-induced atelectasis in children. Anesthesiology, 120(6), 1370–1379. https://doi.org/10.1097/ALN.0000000000000231

Généreux V, Chassé M, Girard F, Massicotte N, Chartrand-Lefebvre C, Girard M. Effects of positive end-expiratory pressure/recruitment manoeuvres compared with zero end-expiratory pressure on atelectasis during open gynaecological surgery as assessed by ultrasonography: a randomised controlled trial. Br J Anaesth. 2020 Jan;124(1):101-109. doi: 10.1016/j.bja.2019.09.040.

Li DK, Liu DW, Long Y, Wang XT. Use of Lung Ultrasound to Assess the Efficacy of an Alveolar Recruitment Maneuver in Rabbits With Acute Respiratory Distress Syndrome. J Ultrasound Med. 2015 Dec;34(12):2209-15. doi: 10.7863/ultra.14.11051.

Monastesse A, Girard F, Massicotte N, Chartrand-Lefebvre C, Girard M. Lung Ultrasonography for the Assessment of Perioperative Atelectasis: A Pilot Feasibility Study. Anesth Analg. 2017 Feb;124(2):494-504. doi: 10.1213/ANE.0000000000001603. PMID: 27669555.

Song IK, Kim EH, Lee JH, Ro S, Kim HS, Kim JT. Effects of an alveolar recruitment manoeuvre guided by lung ultrasound on anaesthesia-induced atelectasis in infants: a randomised, controlled trial. Anaesthesia. 2017 Feb;72(2):214-222. doi: 10.1111/anae.13713.

Sun L, Wu L, Zhang K, Tan R, Bai J, Zhang M, Zheng J. Lung ultrasound evaluation of incremental PEEP recruitment maneuver in children undergoing cardiac surgery. Pediatr Pulmonol. 2020 May;55(5):1273-1281. doi: 10.1002/ppul.24720.

WHAT'S BEHIND THE CURTAIN (SIGN)?

Kris Gommeren [1]

[1] University of Liege, Liege, Belgium

Learning objectives:

- Explain what creates the curtain sign and what it looks like in healthy animals

- Explain how the curtain sign can be used to rapidly diagnose pleural effusion, pneumothorax and lung consolidation.

- Explain how the curtain sign orientates sonographers to where they are on the thorax and can be used to help identify sensitive sites for pleural space pathology to accumulate and to help differentiate some forms of pathology.

Proceeding:

Normal curtain-signs

A normal curtain-sign (C-Sign) is defined as the vertical edge artifact that is created when air-filled lung overlies and obscures sonographically visible soft tissue structures within the thorax. The presence of air-filled lung overlying soft tissues creates a sharp vertical edge artifact demarcating the transition between lung that contains air (visible as reverberation artifact) and the soft tissue structures they overly. The underlying organs disappear as the lung expands across them on inspiration and reappear as the lung contracts on exhalation. In healthy patients, depending on the area the transducer is placed on the thorax, different soft tissue structures can be seen partially covered by air-filled lung. As such, there are two main C-Signs that are often identified in healthy animals:

The abdominal C-Sign which can be seen when air-filled lung overlies the diaphragm and soft tissue structures of the abdomen at the costophrenic recess.

The cardiac C-Sign which is seen when air-filled lung overlies the soft tissue structures of heart at the cardiac notch.

The abdominal C-Sign is the most evident and commonly assessed C-Sign. In healthy animals, the abdominal C-Sign is evident when the transducer is positioned transthoracically, half over lung and half over visible abdomen; air in the lung creates reverberation artifact that obliterates the superimposed soft tissue structures of the abdomen, while the ultrasound beam is transmitted through the soft tissue structures caudal to the lung. The appearance of abdominal structures will vary as the transducer (in

perpendicular orientation to the ribs) is swept ventrally and dorsally along the abdominal C-Sign, and between the left and right sides of the thorax (e.g. the spleen and stomach wall will often comprise the abdominal structures on the left side of the patient while the liver dominates the abdominal organs of the right hemithorax). When only the gastrointestinal wall and luminal gas is visible (e.g. stomach wall or intestinal wall) on the soft tissue side of the C-Sign, it is easy to confuse the normal abdominal structures for lung pathology. When created by air-filled lung of healthy patients, the abdominal C-Sign displays distinct characteristics; during inspiration, as the lung expands into the costophrenic recess the abdominal C-Sign appears to move caudally covering more of the intraabdominal structures. However, the movement of the abdominal C-Sign is caused by lung expansion and therefore is not perfectly synchronous to the movement of the intra-abdominal structures which occurs because of diaphragmatic activity. As a result of these distinct features, there are several criteria that can be distinguished in healthy patients with a normal abdominal C-Sign: 1) there should only ever be one abdominal C-Sign visible at the caudal lung border; 2) it should always be visible throughout the entire respiratory cycle; and 3) although it may move slightly out of sync with the abdominal content and diaphragmatic activity, both the C-Sign and visible abdominal soft tissue structures should move in the same direction during the respiratory cycle. To avoid confusing rib shadows for the C-Sign, the C-Sign should be assessed when it is centred between two ribs, or located just caudal to the more cranial rib, at the end of expiration. Although the normal C-Sign may disappear as it passes under ribs during the respiratory cycle, or extends beyond the caudal border of the ultrasound image, it should not suddenly appear or disappear within the middle of an ultrasound window. It is advisable to assesses the C-Sign and lung sliding together. In healthy animals lung sliding should be visible cranial to the C-Sign, which is not the case with pneumothorax. With pneumothorax lung sliding will be absent in proximity too and give rise to abnormal C-Signs.

Abnormal curtain-signs

Abnormal C-Signs can arise for several reasons. Because the sharp vertical edge artifact of a C-Sign is created by air overlying soft tissue structures, it is not restricted to air-filled lung, and can also be created when free air in the pleural space overlies soft tissue structures (which the authors refer to as abnormal pneumothorax-induced C-Signs). Additionally, when pleural effusion is present C-Signs can also be created when air-filled lung overlies the pleural effusion, expanding and contracting across it (which the authors refer to as an abnormal pleural effusion-induced C-Sign). Although not a true C-Sign pleural effusion and/or consolidated lung that fills the costophrenic recess will obliterate the normal vertical edge artifact of the abdominal C-Sign, allowing the diaphragm to be seen curving away from the chest wall. Therefore, the loss of a normal abdominal C-Sign and visualization of the diaphragm curving away from the chest wall anywhere other than at the pericardio-diaphragmatic (PD) window can be used to diagnose pleural space and lung pathology.

• Abnormal pneumothorax-induced abdominal C-Signs: The absence of lung sliding, B lines/lung consolidation, and a lung pulse, along with supportive history and clinical signs, should raise suspicion of pneumothorax; however, there are two key sonographic criteria that can support its presence; the lung point and abnormal pneumothorax induced C-Signs. In a patient with pneumothorax, the lung point can be defined as the site within the thorax where the visceral pleura of the lung recontacts the parietal pleura of the thoracic wall. Two abnormal pneumothorax-induced C-Signs have been documented in dogs with

pneumothorax; asynchronous and double C-Signs (also referred to as the “accordion” and “peek-a-boo” signs). Although further research is required to determine the sensitivity and specificity of abnormal C-Signs to detect pneumothorax, evidence suggests it may be more sensitive and specific than the lung point for diagnosis of small to medium volume pneumothorax in companion animals. An advantage of assessing the C-Sign in the patient with rapid shallow breathing, often the case in patients with pneumothorax, is the ultrasound image can be frozen. Thus, the stored video loop can be viewed by scrolling back several frames, which allows the C-Sign to be visualized at a slower speed. This often makes it easier to determine if normal and/or abnormal abdominal C-Signs are present. In healthy individuals, the abdominal C-Sign moves caudally on inspiration with other visible abdominal contents at a similar rate; the movement of the vertical edge and abdominal contents is therefore synchronous. Although the abdominal C-Sign may not be visible as it slides under ribs or it slides caudally out of the ultrasound image, it should always present in healthy animals. In the absence of pneumothorax, the lung will slide into the costophrenic recess as the diaphragm contracts, resulting in the C-Sign moving caudally on inspiration. As the lung slides into the costophrenic recess it separates the diaphragm and abdomen from the thoracic wall, preventing abdominal structures from being sonographically visible. In the presence of pneumothorax, free air in the pleural space is present cranial to the costophrenic recess and small pockets of pleural air can be seen within the costophrenic recess. With pneumothorax-induced double C-Signs, two soft tissue-to-air interfaces (a cranial and caudal interface) are visible within the same sonographic window. In contrast to normal abdominal C-Signs which should be present and visible throughout the respiratory cycle, the double C-Sign can suddenly appear and disappear within the ultrasound image. With a pneumothorax-induced asynchronous C-Sign the vertical air edge artifact moves cranially, or fails to move at all, while the abdominal contents move caudally. The C-Sign and the abdominal content are asynchronous to each other. This is believed to occur when pleural air is present lateral and cranial to the diaphragm and there is no lung present to slide into the costophrenic recess when the patient inspires, allowing the soft tissue structures to move laterally into contact with the thoracic wall. Hint: assess both the direction of the C-Sign and abdominal contents with regards to each other to decide if they move in the same or opposite directions during the respiratory cycle. In standing patients with pneumothorax, the C-Sign is often normal in the ventral regions of the thorax, with asynchronous and double C-Signs being identified more dorsally on the thorax. This is helpful to sonographers as sweeping from an area where a subtle asynchronous C-Sign is present to an obvious double C-Sign or ventrally to a region where the C-Sign is normal helps confirm the diagnosis of pneumothorax.

• Pleural effusion and the abdominal C-Sign: When scanned transthoracically the presence of pleural effusion filling the costophrenic recess will appear to track along the diaphragm, often obscuring the mediastinal triangle and permitting the diaphragm to be visible curving away from the thoracic wall. The presence of pleural effusing filling the costophrenic recess may appear before other sonographic signs of pleural effusion can be detected.

• Lung consolidation and the abdominal C-Sign: With trans-lobar (surface to surface) lung consolidation of the caudal lung lobes it may be possible to visualize the diaphragm curving away from the chest wall as consolidated lung allows ultrasound beams to traverse lung tissue due to the absence of air. This will result

in the normal vertical edge artifact of the abdominal C-Sign becoming obliterated and the diaphragm becoming visible, curving away from the chest wall.

References:

Boysen S, McMurray J, Gommeren K. Abnormal Curtain Signs Identified With a Novel Lung Ultrasound Protocol in Six Dogs With Pneumothorax. Front Vet Sci. 2019; 6:291.

Boysen SR. Lung Ultrasonography for Pneumothorax in Dogs and Cats. Vet Clin North Am Small Anim Pract 2021; 51(6):1153-1167.

Hwang TS, Yoon YM, Jung DI, et al. Usefulness of transthoracic lung ultrasound for the diagnosis of mild pneumothorax. J Vet Sci 2018; 19(5):660–666.

Lee, FCY. The curtain sign in lung ultrasound. J Med Ultrasound 2017; 25:101–104.

Martinez AN , Quinn C, Mihm F, Henness e E. 1127: Lung Ultrasound: The fine line between diagnosing and ruling out pneumothorax. Crit Care Med, 2018, 46(1):547.

Goff i A, Kruiss elbr ink R, Volpicelli G. The sound of air: point-of-care lung ultrasound in perioperative medicine. Can J Anaesth, 2018, 65(4):399–416.

Boysen S, Chalhoub S, Gommeren K. PLUS image interpretation: Normal findings In: Boysen S, Chalhoub C, Gommeren K,editors. The Essentials of Veterianry Point-of-Care Ultrasound: Pleural Space and Lung. Zaragoza Spain :Groupo Asis Biomedia (2022). p. 36-62.

Boysen S, Chalhoub S, Gommeren K. PLUS image interpretation: Clinical Application of PLUS: Is there Pneumothorax, Yes/No? In: Boysen S, Chalhoub C, Gommeren K,editors. The Essentials of Veterianry Point-of-Care Ultrasound: Pleural Space and Lung. Zaragoza Spain :Groupo Asis Biomedia (2022). p. 63-90.

KNOBOLOGY; WHY BEING ABLE TO 'DRIVE' YOUR MACHINE IMPROVES IMAGE OPTIMISATION AND HELPS TO SAVES LIVES

Angie Lloyd-Jones [1] Julie Burnage [1]

[1] Aspire Ultrasound Consultancy Services ltd, Ultrasound Training, Flintshire, United Kingdom

Learning objectives:

- The objectives of this lecture are to explain and demonstrate why driving an ultrasound system properly is essential in obtaining well-optimised ultrasound images for all studies undertaken.

- Knowing a particular scan site window or having a particular scan technique is simply not enough when it comes to saving lives. This lecture will highlight the common ultrasound controls, explain why they exist and how to use them to drive any ultrasound system with confidence and consistency and maximise diagnostic outcomes for saving more lives whilst also keeping you safe.

Proceeding:

The objectives of our presentation are to explain and demonstrate how driving an ultrasound system properly is essential if you are to obtain well-optimised ultrasound images for every examination. Knowing where and how to position the probe, the technique for finding a structure and what 'abnormal' looks like are only useful if you can actually see what you are looking at. Having excellent ultrasound system skills will significantly improve your diagnostic potential and help to keep you, your patient and your organisation safe; learning to drive your ultrasound system is just one aspect of confident, competent scanning and is as important as:

Robust competency-based training

Sound clinical knowledge

Understanding of cross-sectional anatomy

Systematic, methodical ultrasound technique

Ultrasound is the most operator dependent form of imaging because it is a dynamic, real-time assessment. Your diagnostic impressions are formed as you scan which is why you need to be sure to give yourself the best chance of seeing what is going on inside your patient. In this presentation we will work through the common ultrasound controls, explain what they do and how using them to drive an ultrasound system with confidence will improve your ability to form your diagnoses. A lack of understanding of the fundamentals of ultrasound beam generation, detection, image formation and tissue interaction can lead to

misinterpretation of ultrasound images, misdiagnosis, incorrect patient management, unnecessary surgery, and in the worst-case scenario, euthanasia.

Benefits of ultrasound image optimisation

Optimising your ultrasound scan images throughout an examination will dramatically increase your chance of recognising both normal and abnormal appearances. A series of well-optimised, archived ultrasound images can go a long way to demonstrate an operator's ultrasound competency, evidence normal or abnormal scan findings, justify subsequent patient management and can serve as a useful training and audit tool.

What is ultrasound image optimisation?

Obviously, the operator's scanning technique plays a very important role in image optimisation but the ability to manipulate the ultrasound system controls and menu functions serves to enhance the diagnostic quality of the dynamic image displayed on the screen and subsequent archived images.

When should you optimise?

It is essential that you optimise the image at the start of the scan and then throughout the examination; structures at different depths and of different reflective properties will require continual adjustment of settings. Good optimisation will also help an operator to recognise image artefacts and why and when they might appear and how to take advantage of them (either use them or remove them so they do not interfere with the image).

Basic 'knobology' In a nutshell

The following ten basic ultrasound system controls are the ones you should use when driving your ultrasound machine (although we admit, some older and some of the very new machines may not have them all or if they do have them, make them difficult to find!)

Probe /Preset option

Frequency

Overall gain

Time gain compensation (TGC)

Depth

Scan width/sector angle

Focus

Magnification/zoom

Dynamic range

Annotation

The presentation will give a useful overview of each control and how it can affect the image as well as the impact incorrect use can have on your diagnostic impression and subsequent scan outcomes. By the end of their talk Angie and Julie, from Aspire Ultrasound Consultancy Services, will have brought their quirky humour and their unique and extensive clinical ultrasound expertise to VECCUS to convince delegates just how important it is to master the ultrasound controls when saving lives. Their favourite phrase is “always see your work as art” because a well optimised image is a thing of beauty!

References:

Further reading

Lloyd-Jones A, Burnage J, Warren-Smith C, Van der Vekens E, Hall a, Dickinson N, Bebbington A, Page N, Kerr A, Bacon R, BMUS and ECVDI Small Animal Veterinary Guidelines for Professional Ultrasound Practice, London, UK, Epub 2022 Dec, version 1. Seiler GS, Cohen EB, d'Anjou MA, French J, Gaschen L, Knapp S, Salwei RM, Saunders HM, ACVR and ECVDI consensus statement for the standardization of the abdominal ultrasound examination, Vet Radiol Ultrasound. 2022 Nov;63(6):661-674. doi: 10.1111/vru.13151. Epub 2022 Oct 3. PMID: 36189784

CASE-BASED POCUS IN ICU PATIENTS

Helen Wilson [1]

[1] Langford Vets, University of Bristol, Small Animal Referral Hospital, Bristol, United Kingdom

Learning objectives:

- Learn how to visually recognise common POCUS findings

- Formulate differentials for these common POCUS findings

- Understand how to logically approach cases with these common POCUS findings

Proceeding:

Free Abdominal Fluid

Free abdominal fluid should always be tapped to ascertain its nature. However, POCUS can narrow down the likely differentials, in conjunction with the history and physical exam findings. Echogenic fluid is more likely to have a high cell content, increasing the index of suspicion for septic peritonitis, haemoabdomen or, less commonly, neoplastic effusions or chyloabdomen. In contrast, anechoic fluid is more likely to have a low cellularity, such as protein-poor transudates due to hypoalbuminaemia or protein-rich transudates due to right-sided congestive heart failure, neoplasia or hepatic cirrhosis.

Hepatised Lung

Consolidated lung tissue can take on the echotexture of liver, hence the term “hepatised lung”. Fluid filled bronchi can appear like bile ducts. The presence of air bronchograms can help to differentiate lung from liver tissue. Differentials for hepatised lung include pneumonia, atelectasis, infiltrative disease and lung lobe torsion. Colour Doppler can help to identify if blood flow is preserved. In the absence of air bronchograms, differentiating hepatised lung from liver can be challenging and therefore the clinician must also consider the possibility of diaphragmatic rupture or hernia. Advanced imaging with CT is often the most definitive way to proceed with confirming a diagnosis when POCUS reveals hepatised lung.

Subpleural Nodules

Subpleural nodules are small mass lesions visible within the pulmonary parenchyma, adjacent to the visceral pleura. Deeper lesions cannot be visualised, as ultrasound waves are only able to penetrate the superficial non-aerated tissue. Subpleural nodules may be neoplastic but can also be septic emboli,

developing abscesses, thromboemboli or granulomas, such as those commonly seen with *Angiostrongylus vasorum.*

E-Lines vs B-Lines

E-lines are emphysema lines. They are linear, vertical, hyperechoic reverberation artefacts that are visible throughout the ultrasound field, with no signal attenuation. They can easily be mistaken for B-lines because they are identical to B-lines, except that they arise from the subcutaneous tissues, whereas B-lines arise from the pleural surface. It is important to consider the possibility of E-lines in trauma cases, to avoid misdiagnosis. Physical exam should usually confirm the presence of emphysema.

Cranial Thoracic Mass

Large, dense areas of soft tissue should not normally be visible on POCUS within the thorax. Regions of soft tissue with rounded margins cranial to the heart often represent mediastinal masses. Masses can be differentiated from mediastinitis, which is typically hyperechoic with poorly defined margins. Mediastinitis is typically associated with free fluid, whereas masses may or may not have free fluid associated with them. Colour Doppler can reveal blood flow within thoracic masses but should be differentiated from tissue wrapping around the great vessels of the cranial thoracic cavity.

Spontaneous Echo Contrast

Spontaneous echo contrast within blood vessels or the heart indicates turbulent blood flow due to interruption of laminar flow, blood stasis, or hyperviscosity, and raises concern that the patient may be at increased risk of thrombotic events.

References:

Cole L, Pivetta M, Humm K, Diagnostic accuracy of a lung ultrasound protocol (Vet BLUE) for detection of pleural fluid, pneumothorax and lung pathology in dogs and cats, Journal of Small Animal Practice, 2021, 62, 178 – 186

Connor-Schuler R, et al. Presence of spontaneous echo contrast on point-of-care vascular ultrasound and the development of major clotting events in Coronavirus Disease 2019 patients, Critical Care Explorations, 2021, 3(1):e0328.

Fernandes Rodrigues N, et al. Comparison of lung ultrasound, chest radiographs, C-reactive protein, and clinical findings in dogs treated for aspiration pneumonia, Journal of Veterinary Internal Medicine, 2022, 36, 743–752

Kraszewska K, Gajewski M, Boysen SR, Application of color Doppler sonography for the assessment of pulmonary consolidations in a dog, Frontiers in Veterinary Science, 2023, 10:1275929 Mannion, P, Diagnostic ultrasound in small animal practice, Oxford, UK, Blackwell Publishing, 2006, 41 – 45.

Pacholec C, et al, Lung ultrasound nodule sign for detection of pulmonary nodule lesions in dogs: Comparison to thoracic radiography using computed tomography as the criterion standard, The Veterinary Journal, 2021, 275, 105727

Roshankhaah R, et al. Detecting pulmonary nodules by using ultrasound multiple scattering, The Journal of the Acoustical Society of America, 2021, 150; 4095 – 4102.

Venco L, et al. Utility of thoracic ultrasonography in a rapid diagnosis of angiostrongylosis in young dogs presenting with respiratory distress, The Veterinary Journal, 2021, 271, 105649

Volpicelli G, et al. International evidence-based recommendations for point-of-care lung ultrasound, Intensive Care Medicine, 2012, 36, 577-591

Ward JL, Lisciandro GR, DeFrancesco TC, Distribution of alveolar-interstitial syndrome in dogs and cats with respiratory distress assessed by lung ultrasound versus thoracic radiographs, Journal of Veterinary Emergency and Critical Care, 2018, 28(5), 415 - 428

MASTERING THE WAVES: COMPETENCY ASSESSMENT IN POINT-OF-CARE ULTRASOUND

Leo Silvén Husu [1]

[1] The POCUS Academy Sus, Skånes University Hospital, Malmö, Sweden

Learning objectives:

- Understanding the significance of competency assessment in POCUS

Recognize the critical role of competency assessment in ensuring the safe and effective use of POCUS in clinical practice, emphasizing patient safety and quality of care.

- Identifying the components of comprehensive competency

Identify the key components involved in a comprehensive competency assessment for POCUS, including theoretical knowledge, hands-on skills, clinical correlation, and ongoing quality assurance.

- Appreciating the role of standardization in training

Appreciate the importance of standardized competency assessment tools and criteria in POCUS training, promoting consistency and fairness in evaluating the skills of new users.

- Exploring the impact on patient outcomes

Understand how competency assessment directly influences patient outcomes by ensuring accurate and timely diagnoses, guiding appropriate interventions, and minimizing the risk of diagnostic errors.

- Recognizing the value of ongoing training and feedback

Acknowledge the significance of continuous education, feedback, and remediation in competency assessment to support the professional development of new POCUS users, fostering a culture of improvement.

Proceeding:

The lecture delves into the subject of competency assessment within the context of training for point-of-care ultrasound (POCUS). The exploration centres on elucidating the foundational importance of this meticulous process in shaping adept and proficient practitioners in the domain of POCUS. Patient outcomes and safety are central for practitioners. POCUS, under the guidance of a proficient practitioner, becomes a tool for accurate diagnoses, informed decision-making, and a discernible improvement in the overall quality of healthcare provision. An incompetent user can be dangerous to the patient because important clinical

decisions can be made based on the POCUS examination. At the forefront of the discussion is an examination of the inherent significance of competency assessment. This analysis transcends the perfunctory nature often associated with assessments, elucidating its critical role as a safeguard for patient safety and a benchmark for the consistent delivery of high-quality care through POCUS. Subsequently, the lecture tries to navigate the intricate landscape of comprehensive competency. This encompasses a thorough integration of theoretical knowledge, hands-on proficiency, and clinical correlation. It is imperative to recognize that this multidimensional approach entails a need for several different assessments that touch on the different parts of the competency. Standardization is necessary for uniformity in assessment tools and criteria. This standardization ensures fairness and consistency in evaluating emerging talents within the dynamic and evolving landscape of POCUS. In conclusion, the lecture reflects on the enduring nature of competency assessment as an ongoing process. It necessitates continuous education, feedback mechanisms, and remediation strategies. By embracing these elements, we advocate for the cultivation of a professional culture committed to perpetual improvement, ensuring that practitioners remain adaptive and responsive to the evolving requisites of POCUS. The lecture exploration unfolds in this academic pursuit, where each facet of competency assessment converges to shape practitioners capable of navigating the multifaceted challenges presented by POCUS.

POCUS APPLICATION IN THE MANAGEMENT AND MONITORING OF CONGESTIVE HEART FAILURE

Teresa DeFrancesco [1]

[1] NC State University College of Veterinary Medicine, Department of Clinical Sciences, Raleigh, United States

Learning objectives:

- To learn the ways that POCUS can provide useful and stress-free assessments for the in-patient and out-patient monitoring of the dog and cat with congestive heart failure (CHF).

- To be knowledgeable of the common POCUS findings associated with left-sided CHF: left atrial enlargement, diffuse bilateral numerous B-lines (vertical artifacts) and that the resolution of these vertical artifacts can lag behind clinical improvement.

- To be knowledgeable that POCUS can provide more useful information than radiographs in dogs and cats with large volume cavitary effusions due to right-sided CHF because it allows a quantification of the fluid volume.

Proceeding:

General considerations

Point-of-care ultrasound (POCUS) examinations of the heart, lungs and pleural space are well accepted for the rapid diagnosis of congestive heart failure (CHF) in dogs and cats. POCUS can also assist with the in-patient and out-patient monitoring of the CHF, optimizing medical management and patient care. POCUS, together with other clinical assessments, allows the clinician to evaluate the response to medical therapy by providing non-invasive physiologic information primarily on the progress of decongestion, but also changes in cardiac chamber dimensions. POCUS provides several advantages to other imaging modalities in the serial monitoring of the hospitalized and outpatient CHF patient. The portability of POCUS allows for cage-side assessments without having to move the patient which can be quite helpful in an unstable or large patient. Because of its lower cost, lower stress and high availability in hospitalized settings, POCUS can be used at more frequent intervals than other imaging modalities. Another advantage of POCUS is its ability to quickly augment the physical exam findings. Physical exam, specifically auscultation, often correlates poorly to the diagnosis of CHF, limiting its clinical utility. And lastly, the author cannot over-emphasize the importance of archiving images when serially using POCUS to monitoring CHF as it provides the best objective data to make clinical decisions.

Monitoring B-lines

Together with serial monitoring of respiratory rate and effort, serial monitoring of B-lines is one of the main POCUS findings used to monitor progress of decongestion of cardiogenic pulmonary edema in the hospitalized patient. Using a consistent lung ultrasound acquisition protocol, changes in sonographic pulmonary edema can be semi-quantitatively assessed serially by the number and distribution of B-lines. In the dyspneic patient with left-sided CHF, numerous B-lines (often coalescing) are typically distributed bilaterally and diffusely throughout the lung fields. The timing and pattern of the resolution of the B-lines in the treatment of congestive heart failure have been studied in both humans and dogs. There have been several human studies showing improvement during the initial hours of treatment in “sonographic pulmonary edema” in patients with acute heart failure. There have been two canine CHF studies showing a decrease in the total number of B-lines and of strongly positive B-line sites to date. Murphy et al showed that total number of B-lines and the number of strongly positive sites improved from initial diagnosis to hospital discharge (median 20 hrs) with continued improvement at first recheck. When the lung ultrasound findings were compared to radiographs, B-line scores reduced more than radiographic edema scores in the cranial quadrants, while the degree of resolution of the caudal quadrants was similar between ultrasound and radiographs. However, the cranial quadrants, despite having a greater reduction in B-line scores, had more residual positive B-line sites at discharge that the more caudal sites, suggesting that the cranial ventral lungs may be the last to resolve. No correlation was found between the B-line scores and 90 days survival or relapse of heart failure. A more recent study, McLaughlin et al attempted to further elucidate more precisely the timing and pattern of resolution of B-lines in hospitalized dogs with cardiogenic pulmonary edema. In this study, B-lines were monitored at 3, 6, 12 and 24 hours after admission. There was a significant decrease in the number of strongly positive B-lines sites over the study time (mostly within the first 6 hours) which lagged behind the decrease in respiratory rates. As with the previous canine study, there was a correlation between the number of positive B-line sites and respiratory rates. However, there was no significant difference between dorsal and ventral clearance of B-lines. This study also compared B-lines to lung auscultation, specifically crackles. There was only a 59.4% agreement amongst all the compared sites further supporting poor correlation between auscultation and B-lines. In an outpatient setting, lung ultrasound exams for B-lines can provide a low-stress and low-cost tool for monitoring for recurrence of CHF. A more comprehensive exam of the lung with either a vertical sliding or similar protocol should be used to evaluate all areas of the lungs.

Monitoring cavitary effusions, caudal vena caval and cardiac dimensions

In addition to monitoring B-lines, POCUS monitoring for the recurrence or resolution of cavitary effusions with treatment can be quite helpful in both the acute and chronic management of dogs with right-sided CHF and cats with CHF. Compared to radiographs and physical exam, POCUS truly functions as the “visual stethoscope” and can easily distinguish between free fluid and enlarged organs in the body cavity. One typically examines the distribution and volume of the fluid in the abdomen and/or chest. The sonographer should evaluate the number of centimeters of fluid that is present between the body wall and organs. Therapeutic decisions regarding medication adjustments or need for centesis can be made based on the POCUS, physical exam and laboratory findings. Caudal vena caval and cardiac dimensions can also be monitored during the management of the CHF patient but require a bit more training and precision to

ensure accurate interpretations. Being consistent with respect to transducer position and anatomic views is super important in serial monitoring of the cardiac chambers to ensure accuracy of measurements.

References:

American College of Emergency Physicians. Policy Statement. Ultrasound Guidelines: Emergency, Point-of-Care, and Clinical Ultrasound Guidelines in Medicine. Accessed on March 4, 2024. https://www.acep.org/patient-care/policy-statements/ultrasound-guidelines-emergency-point-of--care-and-clinical-ultrasound-guidelines-in-medicine

Arvig MD, Laursen CB, Jacobsen N, Gæde PH, Lassen AT. Monitoring patients with acute dyspnea with serial point-of-care ultrasound of the inferior vena cava (IVC) and the lungs (LUS): a systematic review. J Ultrasound. 2022;25:547-561.

Arvig MD, Lassen AT, Gæde PH, et al. Impact of serial cardiopulmonary point-of-care ultrasound exams in patients with acute dyspnoea: a randomised, controlled trial. Emerg Med J. 2023;40:700-707.

Gargani L, Pang PS, Frassi F, et al. Persistent pulmonary congestion before discharge predicts rehospitalization in heart failure: a lung ultrasound study. Cardiovasc Ultrasound. 2015 Sep 4;13:40.

Martindale JL, Secko M, Kilpatrick JF, et al. Serial Sonographic Assessment of Pulmonary Edema in Patients With Hypertensive Acute Heart Failure. J Ultrasound Med. 2018;37:337-345.

McLaughlin C, DeFrancesco TC, Ward JL, Pabon M, Bonagura JD. Patterns and Timing of Resolution of Lung Ultrasound B-lines and Pulmonary Auscultation in Hospitalized Dogs with Cardiogenic Pulmonary Edema. J Vet Intern Med.2024;38:731. (Abstract ESVC-O-20)

Miglioranza MH, Gargani L, Sant'Anna RT, et al. Lung ultrasound for the evaluation of pulmonary congestion in outpatients: a comparison with clinical assessment, natriuretic peptides, and echocardiography. JACC Cardiovasc Imaging. 2013;6:1141-51.

Murphy SD, Ward JL, Viall AK, et al. Utility of point-of-care lung ultrasound for monitoring cardiogenic pulmonary edema in dogs. J Vet Intern Med. 2021;35:68-77.

Tchernodrinski S, Lucas BP, Athavale A, et al. Inferior vena cava diameter change after intravenous furosemide in patients diagnosed with acute decompensated heart failure. J Clin Ultrasound. 2015;43:187-93.

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. 2018;28:415-428.

URETERAL OBSTRUCTIONS

Laura Cole [1]

[1] Royal Veterinary College, Clinical Sciences and Services, Hatfield, United Kingdom

Learning objectives:

- Recall POCUS techniques for the assessment of renal size and architecture
- Describe the technique for measurement of the renal pelvis
- Understand the limitations of renal pelvis measurement on POCUS
- Understand the limitations of POCUS for detecting a ureteral obstruction
- Interpret renal POCUS finding in context of the clinical history and physical examination findings

Proceeding:

Ureteral obstruction is an increasingly prevalent cause of azotaemia, anuria and severe electrolyte derangements in cats (Kyles, et al 2005; Segev et al 2013). The clinical signs are often non-specific and could be compatible with intrinsic acute kidney injury and/or chronic kidney disease. Diagnosis relies on radiographs and ultrasound and/or pyelography, which may require sedation or general anaesthesia. In an unstable, azotaemic and hyperkalaemic patient these diagnostics are often delayed. Unlike acute or chronic kidney disease whereby the cornerstone of treatment is medical management with judicious fluid therapy the treatment of ureteral obstruction requires prompt relief of the obstruction to re-establish kidney function. Abdominal point-of-care ultrasound (POCUS) assessment of the kidneys has a role in expediating diagnosis and management of cats with ureteral obstruction.

Patient preparation

Abdominal point-of-care ultrasound can be performed in whatever position the animal is comfortable in, often standing or sternal recumbency. The fur is often not shaven, instead the fur parted, and alcohol applied directly to skin and ultrasound gel to the ultrasound transducer. For optimal kidney resolution, a high frequency transducer, the linear transducer (8-15MHz) is often recommended in the cat, however, the lower frequency curvilinear transducer can also be used, and is most often used for POCUS.

POCUS views and renal anatomy

The two abdominal POCUS views for assessment of the kidneys are the right and left paralumbar views. The transducer is initially placed longitudinally caudal to the last rib and orientated dorsally to identify the kidney. The left kidney is visualized caudal to the spleen and the right kidney is located more cranially, close to the liver. Once the kidneys are identified the transducer should be fanned dorsally and ventrally to find the longest axis of the kidney as well as surveying the renal architecture (cortex/medulla/renal sinus/pelvis). The transducer should be rotated 90 degrees to achieve the transverse view for assessment of the renal pelvis (Cole, et al 2021).

Renal sonographic findings with ureteral obstruction

In one institution where abdominal POCUS is routinely performed in azotaemic cats, POCUS kidney abnormalities were frequently identified in cats subsequently diagnosed with a ureteral obstruction. Renal asymmetry, renal pelvis dilation and ureteral dilation were seen more frequently in cats with obstructive disease than those without (Beeston et al 2023). POCUS in an azotaemic cat should therefore focus on assessment on renal size, structure and presence or absence of renal pelvis dilation and/or ureteral dilation.

Measurement of the kidneys should be performed in the longest axis. Normal ranges cited for kidney length in cats span from 2.9-5.1cm (Cole et al 2021). It is therefore preferable to compare the left and right kidney lengths rather than focus on absolute values. Reliable assessment of the renal pelvis requires identification of the renal crest in the transverse view which enables the identification of the V shaped renal pelvis (D'Anjou, et al 2011). Measurement of renal pelvis diameter can be challenging, especially in an awake standing patient and POCUS measurement only has moderate reliability when compared to specialist ultrasound (Beeston, et al 2023). Although ureteral dilation can be identified on POCUS, a complete assessment of the ureter from the kidney to the ureterovesicular junction is technically challenging and requires a highly skilled ultrasonographer.

All of the POCUS findings identified in cats with ureteral obstruction have also been identified in cats without obstructive disease and some cats with ureteral obstruction have no kidney POCUS abnormalities. Although guidelines for renal pelvis measurements have been published for diagnosis of a ureteral obstruction, a range of renal pelvis dimensions have been reported in cats with and without ureteral obstruction and the degree of renal pelvis dilation cannot readily differentiate pyelonephritis and ureteral obstruction in isolation (Lulich, et al 2016; Quimby et al 2017; Lemieux et al, 2021, D'Anjou et al 2011, Fages, et al 2018). As for all POCUS, these sonographic findings should be interpreted in light of the history, physical examination and diagnostic tests to guide further management.

References:

Beeston D, Dirrig H, Cole L. The utility of clinicopathological findings and point-of-care ultrasound in increasing the index of suspicion of ureteral obstruction in azotaemic cats presenting to the emergency room. J Small Anim Pract. 2023;64(12):781-787.

Kyles, A. E., Hardie, E. M., Wooden, B. G., et al. (2005) Clinical, clinicopathologic, radiographic, and ultrasonographic abnormalities in cats with ureteral calculi: 163 cases (1984-2002). Journal of the American Veterinary Medical Association 226, 932-936

Segev, G., Nivy, R., Kass, P. H., et al. (2013) A retrospective study of acute kidney injury in cats and development of a novel clinical scoring system for predicting outcome for cats managed by hemodialysis. Journal of Veterinary Internal Medicine 27, 830-839 Lulich JP, Berent AC, Adams LG, Westropp JL, Bartges JW, Osborne CA. ACVIM Small Animal Consensus Recommendations on the Treatment and Prevention of Uroliths in Dogs and Cats. J Vet Intern Med. 2016;30(5):1564-1574.

Lemieux C, Vachon C, Beauchamp G, Dunn ME. Minimal renal pelvis dilation in cats diagnosed with benign ureteral obstruction by antegrade pyelography: a retrospective study of 82 cases (2012-2018) [published online ahead of print, 2021 Jan 27]. J Feline Med Surg. 2021;1098612X20983980. doi:10.1177/1098612X20983980

D'Anjou, M. A., Bédard, A. & Dunn, M. E. (2011) Clinical significance of renal pelvic dilatation on ultrasound in dogs and cats. Veterinary Radiology & Ultrasound 52, 88-94 Quimby JM, Dowers K, Herndon AK, Randall EK. Renal pelvic and ureteral ultrasonographic characteristics of cats with chronic kidney disease in comparison with normal cats, and cats with pyelonephritis or ureteral obstruction. J Feline Med Surg. 2017;19(8):784-790.

Fages J, Dunn M, Specchi S, Pey P. Ultrasound evaluation of the renal pelvis in cats with ureteral obstruction treated with a subcutaneous ureteral bypass: a retrospective study of 27 cases (2010-2015). J Feline Med Surg. 2018;20(10):875-883.

Cole L, Humm K, Dirrig H. Focused Ultrasound Examination of Canine and Feline Emergency Urinary Tract Disorders [published correction appears in Vet Clin North Am Small Anim Pract. 2022 May;52(3):xiii]. Vet Clin North Am Small Anim Pract. 2021;51(6):1233-1248.

Main Stream, Thursday 30 May 2024

MANAGEMENT OF ACUTE HEART FAILURE

Teresa DeFrancesco [1]

[1] NC State University College of Veterinary Medicine, Department of Clinical Sciences, Raleigh, United States

Learning objectives:

- Review the common drugs used for the acute management of heart failure (HF)

-To be knowledgeable about the expected timing and how to monitor for clinical improvement after giving initial doses of these drugs (furosemide and pimobendan).

- To be knowledgeable about when a patient may need additional therapies or dose escalation in a case of initially refractory heart failure.

Proceeding:

Initial considerations

The immediate goals of HF therapy are to reduce abnormal fluid accumulations and restore comfort in breathing while providing an adequate or improved cardiac output. As with any severely decompensated patient, assessments and treatments often occur simultaneously beginning with the evaluation of the ABC's (airway, breathing, and circulation). The initial evaluation should quickly identify those patients with severe respiratory distress and potential respiratory failure. In rare cases, this may involve immediately securing an airway and mechanical ventilation. Support with conventional oxygen therapy or high flow nasal oxygen therapy are more often used for those patients in respiratory distress while medical therapy alleviates congestion.

Initial treatments typically include mild sedation, oxygen, furosemide, pimobendan (in dogs and some cats) and centesis of pleural or peritoneal effusions if needed. Recognizing that there are several "phenotypes" of acute HF in which tailoring of drug therapies and treatments are important considerations. For example, cardiogenic pulmonary edema can be due to chronic circulatory overload as in a patient with chronic heart disease or can be due to acute fluid redistribution as in a patient with an acute chordae tendineae rupture. The chronic circulatory overload patient will benefit from aggressive diuresis whereas the acute chord rupture patient is not hypervolemic and would benefit more from vasodilator therapy and less aggressive diuresis. Minimizing stress is also important. Anxiolysis with low dose butorphanol may be beneficial to decrease the stress of handling. Thoracocentesis in an HF cat with large volume pleural effusion will be the most effective maneuver to quickly restore comfort to breathing. Often, additional sedation is needed to accomplish the centesis. Alfaxalone is the author's current preferred additional sedation drug if butorphanol

alone is insufficient. Dogs with large volume ascites will also benefit from abdominocentesis. Optimizing heart rate and rhythm are also important goals of HF therapy but beyond the scope of this lecture.

Diuretics

Furosemide is the mainstay in the management of fluid overload in acutely decompensated HF. In patients with severely decompensated HF, the best route of administration is IV. However, if IV access is not possible IM administration can be used. The typical starting dose is ~ 2mg/kg IV bolus, that can be repeated 2-3 times in hourly intervals. A combination of repeat IV bolus dosing and CRIs (0.33 – 0.66 mg/kg/hr) of 4-8 hrs in duration, in the author's opinion, results in an efficient and effective diuresis. IV and IM furosemide have a peak onset of action of approximately 30 and 60 minutes, respectively. After initial dosing and progress in decongestion, one will switch to a scheduled dosing at q 6-8 hr until hospital discharge. Choosing the optimal dose of furosemide is challenging. The dose is titrated to respiratory rate/effort and renal perfusion parameters (BUN/creatinine). Combination or alternative diuretics are also other important strategies to improve diuretic response especially in the refractory cases. The author will switch to torsemide, a more potent loop diuretic, if oral furosemide dosages are 8-10 mg/kg/d. Torsemide has a longer duration of action and more consistent bioavailability. Other diuretics that may help in decongestion are spironolactone, hydrochlorothiazide or acetazolamide. Spironolactone has been shown to improve survival and delay recurrence of HF in dogs as compared to placebo when added to conventional HF treatment. Spironolactone is used together with the loop diuretic.

Inotropes

Pimobendan, a dual action drug with positive inotropic and balanced vasodilatory actions ("ino-dilator") is extremely helpful in the management of acute HF. If given orally, peak blood levels are achieved within 1-2 hrs for both pimobendan and its active metabolite. Many dogs in our experience are already on pimobendan when they present in HF as the drug is often used in the preclinical management of dilated cardiomyopathy and mitral valve disease. In these cases, we routinely escalate the dose and/or frequency of pimobendan. Pimobendan is primarily eliminated in feces via bile (95%) and only 5% of the drug and its metabolites are renal excreted, resulting in its safe use in dog with concurrent renal disease and HF. The use of pimobendan in cats has been somewhat controversial. Schober et al found no clear benefit of pimobendan vs. placebo in cats with HF due to hypertrophic cardiomyopathy. However, this study showed that cats with non-obstructive disease benefitted more from the pimobendan than cats with obstructive disease. Ideally an echocardiogram is needed to distinguish cats with and without obstruction. However, if an echo is not possible, cardiac auscultation may help; cats with obstructive cardiomyopathy typically have loud heart murmurs that get louder with increasing heart rates.

Vasodilators

In most acute HF cases, vasodilators (aside from pimobendan) are often not needed but may be indicated in chronic management. Vasodilators are helpful in the refractory acute HF case as they decrease the preload and afterload to the heart. Vasodilators are indicated in the management of an acute chordae tendineae rupture case as these dogs are not classically fluid overloaded but have cardiogenic pulmonary edema due to severe acute increase in left atrial and pulmonary venous pressure. Vasodilator options include

transdermal nitroglycerin, amlodipine, hydralazine, isosorbide dinitrate and sodium nitroprusside. Blood pressure monitoring is advised to help titrate dosages of the vasodilators.

References:

Deniau B, Costanzo MR, Sliwa K, et al. Acute heart failure: current pharmacological treatment and perspectives. Eur Heart J. 2023;44:4634-4649.

Keene BW, Atkins CE, Bonagura JD, et al. ACVIM consensus guidelines for the diagnosis and treatment of myxomatous mitral valve disease in dogs. J Vet Intern Med. 2019;33:1127-1140.

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;46:100596.

Luis Fuentes V, Abbott J, Chetboul V, et al. ACVIM consensus statement guidelines for the classification, diagnosis, and management of cardiomyopathies in cats. J Vet Intern Med. 2020;34:1062-1077.

McDonagh TA, Metra M, Adamo M, et al. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. Eur Heart J. 2021;42:3599–3726.

Peddle GD, Singletary GE, Reynolds CA, et al. Effect of torsemide and furosemide on clinical, laboratory, radiographic and quality of life variables in dogs with heart failure secondary to mitral valve disease. J Vet Cardiol. 2012;14:253-9.

Ponikowski P, Voors AA, Anker SD, et al. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: the Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) developed with the special contribution of the Heart Failure Association (HFA) of the ESC. Eur Heart J. 2016;37:2129–2200.

Salvador DR, Rey NR, Ramos GC, Punzalan FE. Continuous Infusion versus bolus injection of loop diuretics in congestive heart failure. Cochrane Database Syst Rev 2005;(3): CD003178.

Schober KE, Rush JE, Luis Fuentes V, et al. Effects of pimobendan in cats with hypertrophic cardiomyopathy and recent congestive heart failure: Results of a prospective, double-blind, randomized, nonpivotal, exploratory field study. J Vet Intern Med. 2021;35:789-800.

Suzuki S, Fukushima R, Ishikawa T, et al. The effect of pimobendan on left atrial pressure in dogs with mitral valve regurgitation. Journal of Veterinary Medicine 2011;25:1328-33.

MANAGEMENT OF ARRHYTHMIAS

Teresa DeFrancesco [1]

[1] NC State University College of Veterinary Medicine, Department of Clinical Sciences, Raleigh, United States

Learning objectives:

- To be knowledgeable about the common cardiac and non-cardiac conditions commonly associated with arrhythmias.

- To understand the treatment considerations and indications for using ant-arrhythmic therapy to suppress an arrhythmia.

- To review the common drugs used to manage important ventricular and supraventricular tachyarrhythmias.

Proceeding:

General considerations

Simply stated, arrhythmias result from abnormalities in impulse formation, impulse conduction, or both. Arrhythmias can occur with a wide range of diseases and vary notably in their clinical signs from no clinical signs to hemodynamic collapse. The importance of the arrhythmia depends on the rate, frequency, and complexity of the arrhythmia as well as the severity of any underlying cardiac dysfunction or other non-cardiac disease. In general, the approach to the management of arrhythmias involves understanding the underlying cause of the arrhythmia as well as being knowledgeable about when to suppress the arrhythmia and with which drug. Many cardiac and non-cardiac diseases, intoxications/envenomations and electrolyte disturbances are associated with arrhythmias. Electrolyte disturbances, such as severe hypokalemia, hypocalcemia, hypomagnesemia or hyperkalemia, can cause myocardial ionic abnormalities and arrhythmias. Cardiac causes of tachyarrhythmias include cardiomyopathies, infectious endocarditis, myocarditis, severe congenital heart disease, advanced mitral valve disease, and cardiac neoplasia. Trauma resulting in blunt chest or ischemia-reperfusion injury, may also result in ventricular arrhythmias. Non-cardiac disease conditions include sepsis/SIRS, pancreatitis, hemolytic/acute anemias, pheochromocytoma, gastric-dilation-volvulus, splenic diseases and end-stage organ failures. Arrhythmias are identified typically on cardiac auscultation and then characterized with an ECG. Hemodynamically significant arrhythmias typically occur as sustained or paroxysms of cardiac impulses that are too fast, tachyarrhythmias, or too slow, bradyarrhythmias.

Tachyarrhythmias

Tachyarrhythmias are rhythms at rates of 160-300 per minute. An ECG is necessary to further characterize the tachycardia as to its origin – supraventricular (SVT) or ventricular (VT). The duration of the QRS complex in the tachyarrhythmia is the most useful feature of the ECG to differentiate SVT from VT. Narrow QRS tachycardias (QRS duration < 70 ms in dogs, < 40 ms in cats) are generally supraventricular in origin, whereas wide QRS tachycardias are usually ventricular in origin. However, uncommonly SVT can have a wide QRS if it is conducted with a bundle branch block (typically a right bundle branch block), making the distinction between a SVT and VT more challenging. Sinus tachycardia, technically a supraventricular tachyarrhythmia, is a physiologic rhythm that occurs in response to increased need for cardiac output or increased sympathetic tone. The distinction between sinus tachycardia and pathologic SVT can also be challenging.

Pathologic SVTs include atrial fibrillation, atrial flutter, multifocal atrial tachycardia, junctional tachycardia and reentrant accessory pathway. Aside from atrial fibrillation and multifocal atrial tachycardia, most other underlying mechanisms of supraventricular tachycardias have regular QRS intervals. Atrial fibrillation, the most common SV tachyarrhythmia, is typically sustained, while regular QRS interval SVTs and multifocal atrial tachycardias are typically intermittent. Most dogs and cats presenting with atrial fibrillation have severe underlying heart disease, often in congestive heart failure. Diltiazem is a common drug used to manage rapid atrial fibrillation and most other SVT because of its action to slow atrioventricular (AV) nodal conduction. In dogs, the combination of digoxin and diltiazem is also commonly used as the combination is superior to either drug alone at reducing the atrial fibrillation ventricular response rate. Treatment of other supraventricular arrhythmias depends on the frequency of the rhythm disturbance and the presence of any underlying myocardial dysfunction. If the intermittent SVT is frequent, occurring in long runs (several minutes) or is causing symptoms, treatment is recommended. It is important to emphasize that chronic, sustained SVT may result in tachycardia-induced cardiomyopathy and congestive heart failure. A vagal maneuver (ocular or carotid sinus massage) may be successful in transiently breaking a SVT. However, medical treatment will usually be necessary to chronically control the SVT. For management of sustained, symptomatic SVT, diltiazem is usually successful in both dogs and cats. IV esmolol, a rapid-acting beta blocker, may also be helpful in managing SVT acutely. For oral maintenance therapy or non-urgent control of SVT, treatment with oral diltiazem or sotalol can be used. Sotalol may be a good choice if an animal has both ventricular and supraventricular arrhythmias as this dual action drug has effects on both the AV node and myocardium.

The management of VT is importantly different than SVT. However, like SVT, the need and urgency of treatment of VT depends on the hemodynamic status of the patient, the severity of the arrhythmia and underlying myocardial dysfunction. Some ventricular arrhythmias may not require specific anti-arrhythmic therapy and would benefit from supportive care and treating the underlying condition. For example, an accelerated idioventricular rhythm, a ventricular rhythm with rates of 70 – 140 per minute, is a relatively benign rhythm and requires no specific drug therapy. For acute management of severe and symptomatic ventricular arrhythmias in the dog, treatment recommendations include lidocaine IV bolus (2 mg/kg) , repeated to effect up to 8 mg/kg, or less if an adverse effect is observed. If the bolus injections are successful in controlling the VT, then a continuous rate infusion of lidocaine is initiated. If lidocaine is unsuccessful, supplementation with magnesium and ensuring adequate serum potassium concentrations

are advised. Other drugs useful in the management of VT include procainamide, sotalol and amiodarone, contingent of drug availability. For severe medically refractory VT, electrical cardioversion of VT can be performed.

Bradyarrhythmias

Bradycardia is generally defined as a heart rate of < 60 per minute in a dog and < 140 per minute in a cat. An ECG is necessary to further characterize the bradycardia into sinus bradycardia, a sinus node dysfunction/sick sinus syndrome, atrial standstill, or high-grade AV block. Most animals with clinically significant bradyarrhythmias present for evaluation of syncope or weakness. Occasionally some animals may be asymptomatic and are identified on a routine exam. Sinus bradycardia can be due to high vagal tone or due to medications that lower heart rate such as sedative, analgesic, or negative chronotropic cardiac drugs. In cats, severe sepsis, congestive heart failure and low body temperatures can also produce sinus bradycardia. Treatment usually is directed at the underlying cause or disease; however atropine can be given if the sinus bradycardia is hemodynamically significant. When sinus bradycardia occurs with excessively long pauses (several seconds), sinus node dysfunction (preclinical) or sick sinus syndrome (clinical) is suspected; this bradyarrhythmia is an idiopathic dysfunction of the sinus node that most commonly affects Miniature Schnauzers and West Highland White Terriers. For the symptomatic dog, medical therapy may help if the atropine response test resulted in an increase in the heart rate. Bronchodilators, such as terbutaline or theophylline, or a vagolytic drug, such as hyoscyamine, could be used for their chronic positive chronotropic effects. Many dogs may not respond to medical therapy and will require a pacemaker to resolve their syncope. Asymptomatic dogs with sinus node dysfunction do not require any treatment, however, may need to be supported with temporary pacing if they were to undergo a general anesthesia event. Atrial standstill is a slow supraventricular rhythm in which the atria fail to contract with no discernible p waves noted on the ECG. Severe hyperkalemia is the most common cause, resulting from acute renal failure, urethra obstruction, uroabdomen or addisonian crisis. Urgent management of hyperkalemia is recommended. Another cause of atrial standstill, known as persistent atrial standstill, is an uncommon condition in dogs with cardiomyopathy that primarily affects the atria. This causes severe atrial enlargement and fibrosis that does not allow electrical conduction, leading to a junctional escape rhythm at about 60 beats per minute. Pacemaker implantation is recommended. Neither hyperkalemic nor persistent atrial standstill will respond to atropine. Complete or third degree and high-grade second-degree AV block is a common pathologic bradyarrhythmia identified in dogs. Most dogs with high grade AV block have no substantial structural heart disease, while about 50% of cats with high grade AV block have underlying myocardial disease. All dogs and cats with high-grade AV block should have a diagnostic echocardiogram to rule out endocarditis, myocardial disease, and neoplasia. Dogs with high grade AV block have a high risk of death regardless of presence of symptoms and pacemaker implantation is the only effective treatment to improve survival and symptoms. Pacemakers are usually only recommended in cats that are syncopal with paroxysmal high grade AV nodal block.

References:

Bartoszuk U, Keene BW, Baron Toaldo M, Pereira N, Summerfield N, Novo Matos J, Glaus TM. Holter monitoring demonstrates that ventricular arrhythmias are common in cats with decompensated and compensated hypertrophic cardiomyopathy. Vet J. 2019;243:21-25.

Cunningham SM, Dos Santos L. Arrhythmogenic right ventricular cardiomyopathy in dogs. J Vet Cardiol. 2022;40:156-169.

DeFrancesco TC. Supraventricular tachyarrhythmias. In Small Animal Critical Care Medicine 3rd Ed. St. Louis: Elsevier; 2023:pp 283-290.

Gelzer AR, Kraus MS, Rishniw M, et al. Combination therapy with digoxin and diltiazem controls ventricular rate in chronic atrial fibrillation in dogs better than digoxin or diltiazem monotherapy: a randomized crossover study in 18 dogs. J Vet Intern Med. 2009;23:499-508.

Greet V, Sargent J, Brannick M, Fuentes VL. Supraventricular tachycardia in 23 cats; comparison with 21 cats with atrial fibrillation (2004-2014). J Vet Cardiol. 2020;30:7-16.

Michael AE, Grimes JA, Rajeev M, Wallace ML, Schmiedt CW. Perioperative ventricular arrhythmias are increased with hemoperitoneum and are associated with increased mortality in dogs undergoing splenectomy for splenic masses. J Am Vet Med Assoc. 2023;261:1-6.

Pariaut R. Ventricular tachyarrhythmias. In Small Animal Critical Care Medicine 3rd Ed. St. Louis: Elsevier; 2023:pp 291-295

Santilli R, Moïse NS, Pariaut R, Perego M. Electrocardiographic changes secondary to systemic disease and drugs. In: Electrocardiography of the dog and cat DIAGNOSIS OF ARRHYTHMIAS 2nd ed. Milano: Edra S.p.A., 2018.

Verschoor-Kirss M, Rozanski E, Rush JE. Use of esmolol for control of tachycardia in 28 dogs and cats (2003-2020). J Vet Emerg Crit Care. 2022;32:243-248.

MANAGING SEVERE WOUNDS IN THE EMERGENCY CLINIC

Richard Coe [1]

[1] University of Glasgow, , Glasgow, United Kingdom

Learning objectives:

- Even very large/significant wounds can be managed successfully.

- Initial wound management in the first 6-8 hours after injury is crucial to a good outcome.

- Good wound management involves a series of straightforward steps - clipping, lavage, debridement, dressing

- Dressings for the acute wounds are readily available, inexpensive and effective

Proceeding:

The main goal of this lecture is to show a number of case examples of very severe, extensive wounds managed successfully, and to encourage technicians and practitioners working in both specialty emergency and general practice to recognize how treatable such wounds can be with good wound management and reconstruction. Severe wounds are often shocking to both owner and practitioner on presentation, and amputation for limb wounds and euthanasia are sometimes recommended by practitioners without full consideration of treatment options.

Although most wounds are treatable, the costs involved in managing severe wounds are usually substantial, and it is usually financial constraints alongside clinical factors which are the key limiting factor to successful wound management. As with many other conditions, it is vital that emergency practitioners are able to give a realistic idea of cost and prognosis to owners on initial presentation, even if they will not be responsible for the majority of the management of the case.

Emergency practitioners have two key roles to play in the management of major wounds.

INITIAL WOUND MANAGEMENT

All traumatic wounds should be considered “contaminated” or “dirty”. That is not to say that they are “infected” – (bacteria are established and actively dividing). Early experiments demonstrated that it takes approximately 6-8 hours for a contaminated wound to become infected –until that time you have the opportunity to physically wash away contamination, and antibiotic therapy at this point will also be highly effective. This early period is often known as the “Golden Period”.

The first team to see the wound have the best chance of establishing the best possible wound environment for primary repair (in minimally contaminated wounds) or of setting the wound on the right path for successful open wound management and delayed closure or reconstruction.

Anaesthesia or sedation?

Generally I would advise that a patient is anaesthetised for the first exploration/debridement of a very severe wound, if it is safe to do so from the perspective of other injuries. Anaesthesia facilitates more thorough clipping, debridement and lavage. Trying to perform these tasks in a poorly sedated patient is unfair for the animal and is likely to be less effective. Anaesthesia with intubation also ensures that if wounds are found to communicate with a body cavity (especially thorax) then the situation is much more controlled.

In some cases an animal's other injuries may preclude anaesthesia or sedation for proper assessment and initial treatment of the wounds. However, early wound management is a vital part of ensuring a good recovery for the traumatised animal. If the animal cannot be sedated or anaesthetised, try to do whatever is possible with the conscious animal (if the animal is so unstable as to preclude sedation/GA it may well be recumbent) to clip hair from the area and remove as much dirt and debris as possible.

LAVAGE TECHNIQUE

Hartmann's solution, delivered at 4-15 psi is the ideal lavage solution. Saline and tap water have theoretical disadvantages (saline is mildly toxic to fibroblasts in vitro because of its lower pH and lack of a buffering system) but in practice I would use tap water delivered by a shower head for initial copious lavage of grossly contaminated wounds. A convenient way to deliver lavage is to use a bag of fluids as a reservoir and connect a 20-30cc syringe and needle (19-21G) to a three way tap. This enables you to refill the syringe easily without disconnecting and delivers the lavage at about the right pressure.

If there are substantial quantities of dead or devitalised tissue then consider early surgical debridement. Removing all that dead tissue means the macrophages and neutrophils will not be overwhelmed trying to phagocytose large volumes of dead tissue. Don't be afraid to surgically debride on several occasions over the first few days. If you're still debriding tissue on the third or fourth day – consider whether there is some other reason why tissue is continuing to die (e.g. vascular compromise/infection).

In general I would not add anything to lavage fluid e.g. iodine, chlorhexidine, or use any alternative flushing solutions. Prontosan® (B.Braun) is a combination of Polihexanide and Betaine which is marketed as a wound flushing solution for preventing and treating the formation of biofilms. A bottle (350mls) costs in the region of 10-20 Euros, so it is best considered as an additional step to isotonic lavage.

INITIAL DRESSINGS

Following a severe trauma, the wound is likely to be heavily contaminated even after surgical debridement and lavage. Therefore a dressing which will continue to manage contamination and non-viable tissue is recommended for initial wound management. My preference is to use wet-to-dry dressings as they are inexpensive and highly effective. Wet-to-dry dressings are out of favour for the treatment of wounds in people, because they are very uncomfortable to remove. However, in our patients, who will almost always

be sedated or anaesthetised for dressing changes in the early stages of wound management, this is not such a big concern. The alternative to wet-to-dry dressings are hydrogels (e.g. Intrasite, Citrugel) which liquefy dead material so that it can be removed by lavage.

Where bony or joint/ligament injuries are present in combination with severe wounds, then immobilisation is an important part of the initial bandage. Until an external fixator or other form of fixation can be applied, a Robert Jones bandage or splinted bandage should be used to immobilise the limb and maintain the dressings. When changing a bandage like this, it is vital to minimise disruption of the soft tissues, so manipulate the limb with great care. This is the great advantage of external fixation, in that the dressings can be changed without destabilising the bone/joint injury.

IS IT SURGICAL? INTERPRETING ABDOMINAL RADIOGRAPHS IN THE EMERGENCY SETTING

Anna Frykfors von Hekkel [1, 2] Thom Watton [3, 4]

[1] Royal Veterinary College, Royal veterinary college, small animal surgery , South Mimms, United Kingdom
[2] Coastal Veterinary Specialists , Royal veterinary college, small animal surgery, London , United Kingdom
[3] Royal Veterinary College, Small Animal Diagnostic Imaging, South Mimms, United Kingdom
[4] Coastal Veterinary Specialists , Small Animal Diagnostic Imaging, London, United Kingdom

Learning objectives:

- Understand how best to acquire diagnostic abdominal radiographs

- Be able to perform a systematic evaluation of abdominal radiographs

- Recognize radiographic indicators of surgical abdominal disease

Proceeding:

Radiographic Acquisition

Always obtain three radiographic views of the abdomen as the intestinal gas pattern varies in left vs right recumbency. VD is preferable (cf DV) as organ crowding is reduced. In cases of equivocal pneumoperitoneum, horizontal beam radiography can be performed to help to reach a diagnosis.

Contrast Radiography of Urinary Bladder

Plain radiography is an essential first step. Pulling the pelvic limbs forwards allows inclusion of the entire canine male urethra. General anaesthesia is necessary to facilitate patient manipulation and positioning.

Pneumocystography

A pneumocystogram allows assessment of urinary bladder position, wall thickness and as part of a double contrast cystogram, evaluation of intraluminal changes. It is a simple procedure that requires only basic equipment (urethral catheter, syringe, +/- three way tap). Potential complications include fatal air embolism and urinary tract rupture – avoid in very traumatised/inflamed bladders or those with mural pathology.

Positive Contrast Cystography

Useful for identifying bladder position, shape and evaluating for leakage. Small filling defects, such as calculi, may be obscured by contrast material.

Double Contrast Cystography

Useful for evaluating the bladder wall thickness, mucosal wall irregularities and allows detection of small filling defects. A relatively small volume of contrast medium is instilled (5 - 20 ml), the patient is rolled and the bladder massaged then a pneumocystogram is performed.

Retrograde (Vagino-) Urethrocystogram

A bolus of non-ionic iodine-based contrast (e.g. iohexol) is diluted in a 1:1 solution with sterile saline is injected via a urethral catheter during exposure. In a male dog or cat the urethral catheter is inserted into the most distal portion of the urethra to ensure a thorough study is performed. In a female dog or cat the catheter is inserted into the vagina. A foley catheter is used and the bulb in inflated to help secure the catheter. This allows filling of the entire length of the urethra. When assessing for urolithiasis, ensure that the catheter has been **pre-filled** with contrast to avoid instillation of air bubbles which can be difficult to distinguish from uroliths.

Radiographic Indicators of Abdominal Disease

Loss of serosal detail

Pneumoperitoneum

Abnormal size, shape, opacity or location of the gastrointestinal tract

Loss of Serosal Detail Loss of serosal detail describes how easily abdominal organs can be distinguished. Recognition of loss of serosal detail should prompt the clinician to perform a ‘free fluid check’ using ultrasonography (and if appropriate obtain a sample via abdominocentesis).

Pneumoperitoneum

The most common reason for a pneumoperitoneum is prior abdominal surgery. Appreciable free peritoneal gas can remain present on radiographs for 3-4 weeks post-operatively. It is important not to misinterpret this finding for a more concerning diagnosis. When looking for a pneumoperitoneum, evaluate the cranial abdomen particularly carefully. Frequently, the presence of gas caudal to the diaphragm highlights the diaphragm and cranial hepatic margin.

Small Intestinal Obstruction

Sometimes radio-opaque objects can be readily apparent on plain radiographs. It is important to remember that radio-opaque foreign bodies are occasionally incidental findings and do not always warrant surgery. In particular, foreign bodies which are situated within the colon are unlikely to require surgical intervention. A diagnosis of small intestinal obstruction is made based on an increased diameter of the small intestine. Index of suspicion for a **mechanical obstruction** is increased if the distension ends abruptly or if there is a dual population of small intestine.

Linear Foreign Bodies

Linear foreign bodies tend to lodge under the tongue in cats and at the gastric pylorus in dogs. Linear foreign bodies are potentially life-threatening problems that can be overlooked because they do not cause the typical 'obstructive pattern'.

Radiographic signs suggestive of a linear foreign body:

- Bunching of intestines in the cranial abdomen

- Plication of intestines

- Geometrically shaped gas bubbles, often 'comma' shaped

ELEVATING YOUR EX-LAP: HOW TO GET THE MOST FROM YOUR PROCEDURE

Anna Frykfors von Hekkel [1, 2]

[1] Royal Veterinary College, Royal veterinary college, small animal surgery, South Mimms, United Kingdom
[2] Coastal Veterinary Specialists , small animal surgery, London, United Kingdom

Learning objectives:

- Be aware of the steps required for systematic and thorough abdominal exploration

- Understand normal abdominal anatomy

- Know how to pursue abdominal organ biopsy

Proceeding:

Duodenal and colonic manoeuvre

Allows easy access to and inspection of the right and left paralumbar gutters, respectively i.e. adrenal glands, kidneys, ureters, ovaries and uterine horns, and ventral sub-lumbar musculature (invaluable for a bleeding ovarian pedicle). The long mesenteries of the descending duodenum and the descending colon are recruited so that they cradle the intestinal tract, facilitating retraction and inspection.

Avascular Peritoneal Reflection of Caudal Duodenal Flexure

The caudal duodenal flexure is attached to the right caudal abdominal wall by an avascular peritoneal reflection. This can be transected to improve mobility of the duodenum in this region. This is particularly beneficial if an intestinal resection and anastomosis is required at this site.

Proximity of Ureters to Urogenital Tract

At their distal extent, the ureters leave their retroperitoneal position to enter the trigone of the urinary bladder. The ureters flank the urogenital tract and colon in their passage into the pelvic canal. This means that inadvertent trauma or ligation of a ureter during ovariohysterectomy is most likely to occur at this location.

Left Lobe of the Pancreas

The left lobe of the pancreas is often overlooked during exploratory laparotomy. It is most easily accessed by retracting the double leaf of the greater omentum ventrally and cranially to allow visualisation of the most cranial aspect of the dorsal leaf of the omentum.

Abdominal Wall Closure

Closure can be performed using a simple continuous pattern provided the correct suture type and size are used. Bites of at least 5mm are taken in the external rectus sheath, at appropriate intervals (around 1 cm). Care should be taken not to damage the suture with instrument handling, which can predispose to it snapping. The strength holding layer is the external rectus sheath and passage of the needle and suture through the rectus muscle itself is unnecessary. The external rectus sheath can be appreciated as a discreet layer from the umbilicus to the pubis, therefore in the caudal half of the abdomen, it is onlynecessary to include the external rectus sheath in suture bites. Cranial to the umbilicus the external rectus sheath is not visible as a separate layer.

Performing the Exploratory Laparotomy

Patient preparation: A large area from the caudal third of the thoracic wall to 5 cm caudal to the pubis is clipped. Consider whether drains or feeding tubes will be needed and ensure the clip is wide enough to facilitate this.

Skin incision from xiphoid to pubis: In a male dog the caudal extent of the laparotomy is continued parapreputially. A branch of the caudal superficial epigastric artery and vein are ligated and divided during this approach. Note that although the skin incision curves away from the midline, the abdominal wall incision continues midline along the linea alba. Take care - the cranial extent of the approach can result in a defect in the diaphragm resulting in a pneumothorax. Ensure that you notice this by inspecting the diaphragm as the first step of every exploratory laparotomy ensures that this is identified immediately allowing appropriate ventilatory support, drainage and repair as necessary.

Perform a systematic abdominal exploration:

1. Diaphragm
2. Oesophagus
3. Liver, gall bladder, biliary tract
4. Stomach
5. Descending duodenum and right lobe of pancreas
6. Right paralumbar gutter
7. Small and large intestinal tract
8. Left paralumbar gutter
9. Spleen
10. Left lobe of pancreas and dorsal side of stomach
11. Urogenital tract
12. Body wall

IMMEDIATE VS DELAYED SURGERY IN THE ER

Jethro Forbes [1]

[1] Cornell University College of Veterinary Medicine, Ithaca, United States

Learning objectives:

- Review the literature and outcomes around immediate vs delayed surgery for abdominal conditions such as pyometra, GDV, intestinal foreign bodies, and hemoabdomen.

- Develop an evidence based confidence advocating for patients around timing of emergency surgery for abdominal conditions.

Proceeding:

This panel discussion will examine the literature surrounding timing of several emergency surgical conditions. As emergency clinicians and patient advocates, we are motivated to achieve the best patient outcomes using evidence-based practices. Emergency presentations of hemoperitoneum, gastrointestinal foreign body obstruction (GIFBO), pyometra, and gastric dilatation with volvulus (GDV) may have historically been an automatic middle of the night surgery. Veterinary practice is molded by many factors including literature/evidence, the way we have always done things, hospital culture, personal experience, availability of personnel to take patients to surgery, liability concerns, financial considerations, and certainly, clinical team wellness. Whether you cut your own emergency surgeries, work with in-house surgeon colleagues, or refer to another facility, the recommendations you make to an owner about the timing of surgery is an important conversation. These recommendations may influence euthanasia decisions, add stress to the healthcare delivery system, contribute to the moral distress of many players involved, and affect the public perception of our services. There is also concern in the surgical literature around increased complication rates in the out of hours timeframe that need to be balanced with the outcomes benefit of early immediate surgical intervention.

Canine Acute Spontaneous Hemoperitoneum

The literature around the ideal timeframe to surgically explore a spontaneous (non-traumatic, non-rodenticide coagulopathy) hemoperitoneum is generally lacking and personal observations have noted variability in the timeline for surgery depending on the institution and the perceived ability to stabilize the patient medically until daytime hours. Causes for canine spontaneous hemoperitoneum may be neoplastic and involve any of the spleen, liver, kidney(s), adrenal gland and others. While neoplasia is a frequent cause of spontaneous hemoperitoneum, there are other surgical causes such as splenic torsion and GDV as well as

non-surgical conditions including anaphylactic reactions. Widespread use of point of care ultrasound has provided emergency clinicians and surgeons with more information than ever before, but there may be limitations to diagnostic accuracy of the origin of the hemorrhaging mass and the presence of metastatic lesions. Owners may be provided a daunting prognosis and asked to make expensive surgical decisions or consider euthanasia. Pausing overnight to provide blood or other volume resuscitation and stabilization may set up the patient for a more stable anaesthetic event. Additionally, this approach would take the pressure off the owner making a serious decision while allowing for more investigation/imaging the following day. This approach may also add to the expense of the case management by delaying surgical intervention while adding further advanced diagnostic imaging.

Gastric Dilatation with Volvulus (GDV)

Long considered an immediate surgical emergency (with appropriate decompression and cardiovascular stabilization), recent literature investigating a delay in surgical treatment of GDV has raised speculation around delaying surgery in some circles. In many others (including the author's), GDV remains an emergency surgical condition.

Ultimately, emergency restoration of normal positioning of the stomach to restore tissue perfusion and remove the cause of obstructive shock in canines with GDV remains the best recommendation. Aggressive and robust cardiovascular resuscitation prior to placing the patient under anaesthesia is considered crucial. The described studies may provide some comfort in stabilization and placement of an indwelling nasogastric tube prior to any transportation to an emergency surgical facility, but caution should be exercised when considering routine delay in surgical treatment of canine GDV.

Pyometra

A changing paradigm for the timeline in administering surgical treatment for a pyometra has gained traction with the recent publication of several studies. Historically, pyometra patients have been appropriately assessed as having a septic focus with consistent bloodwork and clinical conditions that fit the label of severe infection and/or sepsis. Urgent stabilization with early administration of antimicrobials and fluid resuscitation was followed by emergency surgery. Recognizing families with dogs (or cats) developing pyometra may not have the means to pursue costly emergency surgery, recent studies have evaluated patients referred to a community-based practice and a delay in time to surgical intervention in some cases up to 1 week. The survival rates in the delayed group in this retrospective analysis were not statistically different from those with more emergent surgical intervention.

Gastrointestinal Foreign Body Obstruction (GIFBO)

Timing of surgical intervention for foreign body obstruction in the GI tract of canine patients has been evaluated in numerous recent studies. A retrospective study (Maxwell, et al. Vet Surg. 2021) used the timing cut off of 6 hours after presentation to delineate immediate versus delayed surgery and found similar outcomes in survival to discharge (96% in each group) and cost of hospitalization, however time to discharge was significantly faster in the immediate surgery group. An interesting finding was the natural fecal passage of an obstructive foreign object in 61 medically managed dogs that were not included in the statistical

evaluation between immediate and delayed. Overall rates of GI necrosis and perforation were higher in the delayed group necessitating more complicated surgical procedures (enterectomy) and possibly contributing to the longer duration of hospitalization in this delayed group. Several other studies (Hayes. JSAP 2009) have documented increased complications rates with worse outcomes with longer duration of clinical signs associated with GIFBOs.

As a clinical standard, the author recommens aggressive stabilization of any patient that is deemed surgical prior to the induction of anesthesia and moving to the operating theatre. Anesthetizing a patient prior to volume resuscitation, optimizing of cardiovascular, electrolyte and perfusion status is not uncommon when the team is faced with urgency to get into surgery or to get the surgical clinician in and out of the hospital efficiently. Regardless of the timing, proper patient stabilization is critical.

Emergency surgery recommendations exist in a complex landscape depending on the surgical condition, the availability of the surgical team, the focus on best patient care balanced with the need to protect our surgical colleagues from burnout, and the perception of pet owners. Evidence to suggest delayed surgery in hemoperitoneum and pyometra cases may have similar positive clinical outcomes as immediate surgery. However, appropriate resuscitation and stabilization are imperative to these outcomes. These delays may protect pet owners form the moral distress of life and death decisions in the middle of the night as well as protect the surgical team while still protecting the patient outcomes. GDV and GIFB cases would appear to have somewhat mixed evidence but, on the whole, more immediate surgery would be indicated once adequate resuscitation has been delivered. Ultimately, discussion of evidence and pre-planning with the surgical team are likely to achieve common approaches to surgical emergencies and their timing of surgical intervention.

References:

Hagman R. Pyometra in Small Animals 2.0. Vet Clin North Am Small Anim Pract. 2022 May;52(3):631-657.

Hayes G. Gastrointestinal foreign bodies in dogs and cats: a retrospective study of 208 cases. J Small Anim Pract. 2009 Nov;50(11):576-83.

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.

Lhuillery E, Velay L, Libermann S, et al. Outcomes of dogs undergoing surgery for gastric dilatation volvulus after rapid versus prolonged medical stabilization. Vet Surg. 2022 Jul;51(5):843-852.

Lynch AM, O'Toole TE, Hamilton J. Transfusion practices for treatment of dogs undergoing splenectomy for splenic masses: 542 cases (2001-2012). J Am Vet Med Assoc. 2015 Sep 15;247(6):636-42.

Maxwell EA, Dugat DR, Waltenburg M, et al. Outcomes of dogs undergoing immediate or delayed surgical treatment for gastrointestinal foreign body obstruction: A retrospective study by the Society of Veterinary Soft Tissue Surgery. Vet Surg. 2021 Jan;50(1):177-185.

McCobb E, Dowling-Guyer S, Pailler S, Intarapanich NP, Rozanski EA. Surgery in a veterinary outpatient community medicine setting has a good outcome for dogs with pyometra. J Am Vet Med Assoc. 2022 May 21;260(S2):S36-S41.

Pailler S, Slater MR, Lesnikowski SM, et al. Findings and prognostic indicators of outcomes for bitches with pyometra treated surgically in a nonspecialized setting. J Am Vet Med Assoc. 2022 May 21;260(S2):S49-S56.

Rosselli D. Updated Information on Gastric Dilatation and Volvulus and Gastropexy in Dogs. Vet Clin North Am Small Anim Pract. 2022 Mar;52(2):317-337.

Song KK, Goldsmid SE, Lee J, Simpson DJ. Retrospective analysis of 736 cases of canine gastric dilatation volvulus. Aust Vet J. 2020 Jun;98(6):232-238.

White RS, Sartor AJ, Bergman PJ. Evaluation of a staged technique of immediate decompressive and delayed surgical treatment for gastric dilatation-volvulus in dogs. J Am Vet Med Assoc. 2021 Jan 1;258(1):72-79.

Wright LV, Renwick M, Soh RWY, et al. Outcomes and Blood Product Use in 89 Surgically Managed and 79 Medically Managed Cases of Acute Spontaneous Hemoperitoneum in the Dog. Front Vet Sci. 2021 Oct 8;8:736329.

Advanced Stream, Thursday 30 May 2024

CARDIOVASCULAR-RENAL AXIS DISORDERS

Jethro Forbes [1]

[1] Cornell University College of Veterinary Medicine, Ithaca, United States

Learning objectives:

- Describe the complex physiologic interaction of the heart and kidneys in health and disease

- Provide a framework of classification of cardiorenal syndrome from the human literature as well as the more recent veterinary literature.

- Recognize diagnostic combinations that may better help veterinarians understand what is happening in both the heart and kidneys in disease.

- Provide individual clinical recommendations in specific disease scenarios when the heart and kidneys may seem to need opposing therapeutic interventions.

Proceeding:

Introduction

Organ systems in a patient rarely act entirely independently of one another either in health or disease. Systemic illness may lead to dysfunction of one system, just as the failure of a single system can have direct effects on others. In the case of the heart-kidney interactions, veterinarians face common challenges balancing the needs of both systems, which can be overwhelming. Understanding these complex interactions may provide a higher level of comfort in treating the heart and kidneys and lead to more successful outcomes. This lecture will present a background on the human classification system for Cardio-Renal Syndrome (CRS), the veterinary consensus guidelines for Cardiovascular-Renal Disorders (CvRD) and delve into some clinical considerations for management of competing disease states in the emergency setting. In 2015, an international consensus group comprised of veterinary nephrologists and cardiologists published guidelines for veterinary medicine to better reflect what is happening in our patient populations (Pouchelon, et al 2015). From this veterinary consensus statement, “cardiovascular-renal disorders (CvRD) are defined as disease, toxin or drug-induced structural and/or functional damage to the kidney and/or cardiovascular system, leading to disruption of the normal interactions between these systems, to the ongoing detriment of one or both.”

$CvRD_H$ (Unstable Disease)

This classification reflects rapid or acute worsening of cardiac function leading to an acute kidney injury (AKI). As defined by the IRIS Acute Kidney Injury Staging system, even a modest increase of creatinine (>0.3mg/dL) may reflect injury in the absence of overt azotemia. Other biomarkers such as symmetric dimethylarginine (SDMA), neutrophil gelatinase-associate lipocalin (NGAL), urinary cystatin B, and serum inosine may lead to earlier recognition of kidney injury in these acute cardiac cases. More than likely, a panel of multiple markers will be most helpful to track the earlier injuries to the kidneys in the face of $CvRD_H$. Failure of cardiac output from a primary cardiac dysfunction may lead to acute or chronic hypoperfusion of the kidneys. Cardiogenic shock, acute decompensated left sided congestive heart failure, neuroendocrine (RAAS) and sympathetic nervous system activation (renal vasoconstriction), can all result in decreased renal blood flow and a hypoperfused state. Congestive nephropathy secondary to parenchymal edema in the encapsulated kidneys creates a condition whereby perfusion of the kidneys is obstructed despite potentially adequate renal blood flow as the inflow is obstructed. Furosemide may not be delivered to the renal vasculature for transport across the proximal renal tubular cells leading to one mechanism of loop diuretic resistance in this state.

$CvRD_H$ (Stable Disease)

Chronic cardiac disease and dysfunction contribute to a decreased effective circulating volume, persistent sympathetic activation and stimulation of the renin-angiotensin-aldosterone system (RAAS). The RAAS activation is meant to be protective, however chronic activation leads to oxidative stress, inflammation, and generation of reactive oxygen species.

ACE-Inhibitors are used to antagonize the RAAS mediated conservation of sodium and water during physiologic states in which the excess volume is counterproductive. They also contribute to antagonism of the RAAS induced pathologic increase in systemic blood pressure and excess glomerular pressures (efferent arteriolar vasoconstriction mediated) causing hyperfiltration, toxic proteinuria and progression of chronic kidney disease. Angiotensin Receptor Blockers (ARBs) are more targeted therapeutics with specificity for angiotensin-II, subtype-1 (AT1) receptor and prescribed therapeutically for the same indications as ACE-Inhibitors. Preservation of the AT-II, subtype-2 (AT2) pathway may provide an escape pathway for patients during times of stress where the traditional blockade of RAAS is detrimental.

The take home message regarding ACE-I and ARBs is that unwell patients with diminished ability to maintain adequate volume intake or those with excess fluid losses (gastrointestinal, urinary, other) should have these therapeutics suspended. Individual titration is essential to maintain kidney function while treating chronic cardiac disease.

$CvRD_K$ (Unstable Disease)

An acute kidney injury (AKI) creates several physiologic changes that are detrimental to many bodily systems including the heart. Alterations in the intravascular volume status from either depletion or iatrogenic over-resuscitation affect cardiovascular stability. Neurohormonal axis activation along with widespread inflammation and abnormal coagulation pathway activation may also be accompanied by bacteremia from an infectious cause of AKI. The electrolyte derangements seen from impaired kidney

excretory function, particularly hyperkalemia, have a potential for inducing cardiac arrhythmias and dysfunction. Many cardiac supporting medications (ACE-I, diuretics) may require a pause for concern worsening of kidney disease which may sacrifice the cardiac benefits of those medications.

$CvRD_K$ (Stable Disease)

Similar to the AKI induced effects on cardiac status, chronic kidney disease (CKD) has the potential for chronic inflammation, electrolyte derangements (hyperkalemia, other), and intravascular volume fluctuation. Chronic presence of known as well as unmeasured uremic toxins can negatively affect cardiomyocytes as can the presence of low-level chronic anemia often associated with CKD. Anemia may also induce a high output state to compensate for decreased oxygen carrying capacity. Systemic hypertension secondary to CKD leads to increases afterload and left ventricular hypertrophy.

$CvRD_O$

This classification encompasses a secondary disease state causing detrimental effects on both cardiac and kidney function. Systemic illnesses such as sepsis and other inflammatory diseases (pancreatitis, neoplasia, hyperadrenocorticism) are thought to be candidate disease states that can affect both systems. There is also the potential for cardiac disease and kidney dysfunction to exist independently and concurrently under this classification. This is the least well-defined category in the definition scheme.

References:

Buliga-Finis ON, Ouatu A, Badescu MC, et al. Beyond the Cardiorenal Syndrome: Pathophysiological Approaches and Biomarkers for Renal and Cardiac Crosstalk. Diagnostics. 2022; 12(4):773.

Kim GH. Renal effects of prostaglandins and cyclooxygenase-2 inhibitors. Electrolyte Blood Press. 2008 Jun;6(1):35-41. Langston C, Gordon D. Effects of IV fluids in dogs and cats with kidney failure. Front Vet Sci. 2021 Apr 20;8:659960.

Orvalho JS, Cowgill LD. Cardiorenal Syndrome: Diagnosis and Management. Vet Clin North Am Small Anim Pract. 2017 Sep;47(5):1083-1102.

Pouchelon JL, Atkins CE, Bussadori C, et al. Cardiovascular-renal axis disorders in the domestic dog and cat: a veterinary consensus statement. J Small Anim Pract. 2015 Sep;56(9):537-52. doi: 10.1111/jsap.12387.

Raina R, Nair N, Chakraborty R, Nemer L, Dasgupta R, Varian K. An Update on the Pathophysiology and Treatment of Cardiorenal Syndrome. Cardiol Res. 2020 Apr;11(2):76-88.

Ronco C, Haapio M, House AA, et al. Cardiorenal syndrome. J Am Coll Cardiol. 2008 Nov 4;52(19):1527-39.

Ronco C, McCullough P, Anker SD, et al; Acute Dialysis Quality Initiative (ADQI) consensus group. Cardio-renal syndromes: report from the consensus conference of the acute dialysis quality initiative. Eur Heart J. 2010 Mar;31(6):703-11.

ELECTROLYTES : WHY NOT ON URINE?

Maxime Cambournac [1]

[1] Centre hospitalier vétérinaire Fregis, Emergency and Critical Care, Gentilly, France

Learning objectives:

- Understanding the Regulation of Electrolytes by the Renal System

-Knowledge of Measurement Methods in Urinary Electrolyte Testing

-Indications and Interpretation of Urinary Electrolytes Testing

-Recognition of Conditions Affecting Urinary Sodium Levels

-Application of Urine Biochemistry in Clinical Diagnosis

Proceeding:

Among the different objectives that the renal system normally accomplishes, the kidneys tightly regulate the body's electrolytes, volume, and acid-base status. Although the time the renal system needs to determine a change in plasma is long, at the level of urine every small variation in excretion of a strong ion will be reflected by a great variation in its concentration. As such, these changes can be tracked by urine biochemistry.

Indications of urinary electrolytes testing

Oliguria

Under normal conditions, the kidneys respond to a decrease in effective circulating volume by avidly reabsorbing sodium with the goal to increase volume status. Regardless of the cause of the low-perfusion state, if the oliguria is due to some sort of hypovolemic shock state, the urinary sodium should be low because the nephron is making every effort to retain it, which theoretically should manifest by a low sodium content in the urine (<10 mEq/L).[2]Although in selected circumstances, a low urine sodium may suggest the kidneys are sensing low perfusion (prerenal cause); this assessment often is confounded, in particular in ICU settings (e.g., resuscitation, diuretic exposure, heart failure with low output state). In the goal of differentiating between prerenal states (or low effective circulation volumes) and established AKI, the fractional excretion of sodium has been considered more reliable than urine sodium alone in differentiating these entities because it directly measures sodium handling.[4] Theoretically, a fractional excretion of sodium (calculated by urinary sodium time plasma creatinine, divided by the product of plasma

sodium and urinary creatinine) of less than 1% indicates the kidneys are retaining sodium and can be found in early AKI (prerenal causes), whereas a fractional excretion of sodium more than 2% may be found with more established AKI when tubular damage disrupts the kidneys' capacity to retain sodium.[4] While it is considered more reliable, there are prerenal conditions where the fractional excretion of sodium may be increased.

Hyponatremia

In patient presented with hyponatremia, urine sodium is essential when elucidating the cause. A low urine sodium (<10 mEq/L) may suggest a reduced effective circulating volume is the cause (except in primary polydipsia, in which the urine sodium is low despite normal volume status), whereas a high urine sodium (>40 mEq/L) suggests syndrome of inappropriate antidiuretic hormone (ADH) secretion or with renal losses (e.g., mineralocorticoid deficiency, salt wasting, diuretics). The recent administration of a loop diuretic, any situation where a substantial amount of bicarbonate is being excreted, hyperglycemia, or mannitol therapy will interfere with sodium excretion or reabsorption.[5]

Hypokalemia

The kidneys normal response to hypokalemia is to decrease potassium excretion and to increase its reabsorption form the tubular lumen. Less than 2mmol/L (in humans) of random urinary potassium confirm that the renal compensatory mechanisms are working, and that the loss of potassium have occurred in some other non-renal way. Conversely, if the urinary potassium is high in hypokalemia, this either represents a "true" potassium wasting or a elevated urinary potassium in the presence of severely concentrated urine. If the urine production is less than 0,5ml/kg/h, it is likely the urine has a near-maximal osmolality, and the urinary potassium is still being conserved appropriately. Under such circumstances, the clinician should probably ask for a 24-hour urine specimen analysis or potassium-to-creatinine ratio.[2] Given that creatinine theoretically is excreted at a near constant state, the urine potassium to creatinine ratio corrects for variations in urine volume, unlike a spot urinary potassium. Another parameter, the transtubular potassium allows for interpretation of potassium excretion in the context of inadequate sodium delivery or decrease renal blood flow. In the evaluation of hypokalemic states, a TTKG of more than 7 represented increased renal losses (e.g., hyperaldosteronism, diuretics), whereas a TTKG of less than 3 implied extrarenal losses (e.g., GI losses). However, in human medicine, some evidence has suggested some uncertainty in the use of TTKG in a clinical setting, probably secondary to many confounding factors.

Conclusion

Urine biochemistry can play an important adjuvant diagnostic role in the evaluation of a number of clinical problems. Thus, clinicians should recognize urine biochemical tests require careful integration in ICU settings into the broader clinical context before directing or informing about diagnosis and therapeutic management.

References:

Measurement of urinary concentration: a critical appraisal of methodologies, Chadha, V., Garg, U. & Alon, U., Pediatr Nephrol 16, 374–382 (2001).

Urinary Electrolytes, Solutes, and Osmolality., Waldrop JE, Vet. Clin. North Am. Small Anim. Pract. 2008;38:503–12.

Clinical Approach to Advanced Renal Function Testing in Dogs and Cats, Pressler BM. Clin. Lab. Med. 2015;35:487–502.

Fractional excretion of electrolytes in volume-responsive and intrinsic acute kidney injury in dogs: Diagnostic and prognostic implications. Troia R, Gruarin M, Grisetti C, Serafini F, Magna L, Monari E, et al., J Vet Intern Med.2018;32:1372–82.

Real-time urinary electrolyte monitoring after furosemide administration in surgical ICU patients with normal renal function, Zazzeron L, Ottolina D, Scotti E, Ferrari M, Bruzzone P, Sibilla S, et al., Ann. Intensive Care. Springer Paris; 2016;:1–10.

KIDNEY REPLACEMENT MODALITIES: WHAT ARE THE OPTIONS?

Jethro Forbes [1]

[1] Cornell University College of Veterinary Medicine, Ithaca, United States

Learning objectives:

- Describe the two primary modalities of diffusion and convection in kidney replacement therapies.

- Identify pros and cons of diffusive, convective and hybrid kidney replacement therapies as they apply to veterinary patients.

- Choose the safest prescription in veterinary kidney replacement therapies utilizing the choices amongst modalities available and the platforms currently in use.

Proceeding:

Kidney Replacement Modalities: What are the options?

Kidney replacement therapies (KRT) are utilized in veterinary practice to provide clearance of uremic solutes, normalize electrolyte derangements, and restore systemic homeostasis in acute kidney injury and end stage chronic kidney disease states.

Navigating convective, diffusive, and hybrid therapies to maximize safe clearance of uremic solutes require understanding the physiology behind traditional continuous and intermittent therapies while respecting the pros and cons of each.

Diffusive therapies used in hemodialysis take advantage of an established concentration gradient between two aqueous solutions separated by a semi-permeable membrane to remove small to middle weight solutes (500-1000Da but up to 10,000 Da) from the blood stream. The gradient is achieved by passing a prescribed dialysate solution over one side of the semi-permeable membrane of the hemodialyzer. Diffusion is the principal clearance method on discontinuous (intermittent) KRT platforms where it is highly efficient at solute removal. In human nephrology, diffusion modalities are employed for intensive and short treatments for cardiovascular stable patient populations undergoing chronic hemodialysis. In smaller veterinary patients with severe azotemia, rapid clearance of uremic solutes has a high risk of causing complications including cardiovascular decompensation and dialysis disequilibrium syndrome. However, the technical differences in administering a discontinuous treatment using inline dialysate generation can impart certain financial benefits over prefabricated dialysate and replacement fluids utilized on continuous

platforms. Duration of treatment has staffing benefits in veterinary KRT as opposed to the continuous modalities.

Convective removal of solutes is achieved through application of sustained pressure on the blood side of a hemofilter to force plasma water across the semi-permeable membrane while dragging solutes into the effluent fluid created. The applied pressure creates conditions amenable for clearing of middle to larger solutes (up to 40,000Da) compared to diffusion. Convection, hemofiltration, can be prescribed as a more gradual process for clearing solutes and is most associated with continuous kidney replacement therapy. Critically ill human patients may be more likely to receive this gentle approach to clearance of solutes to reduce the risk of overly efficient solute removal. This involves 24 hours/day treatment sessions which become labor intensive in veterinary programs.

Hybrid therapies utilizing both diffusion and convection, hemodiafiltration, can be performed on some KRT platforms to take advantage of both modalities. Hemodiafiltration can be a purely continuous therapy or can be shaped into a prolonged intermittent modality to combine the benefits of efficiency of hemodialysis with the safety of hemofiltration. This is primarily achievable using a continuous platform as most discontinuous platforms are not capable of this flexibility.

References:

Cowgill LD, Francey T. Hemodialysis and extracorporeal blood purification. In: DiBartola SP, ed. Fluid, Electrolyte, and Acid-Base Disorders in Small Animal Practice. 4th ed. Saint Louis, MO: W.B. Saunders; 2012:680-713. Dufayet C & Cowgill LD. Reevaluation of prescription strategies for intermittent and prolonged renal replacement therapies. Advances in Small Animal Care. Nov 2021(2):117-129.

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.

Kopecny L, Palm CA, Segev G, Cowgill LD. Ultrafiltration during intermittent hemodialysis in dogs with acute kidney injury. J Vet Intern Med. 2023 May-Jun;37(3):1021-1029.

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

DIAGNOSIS AND MANAGEMENT OF UROABDOMEN IN DOGS AND CATS

Julien Guillaumin [1]

[1] Colorado State University, Clinical Sciences, Fort Collins, United States

Learning objectives:

- Recall the diagnosis of uroabdomen using creatinine and potassium in abdominal fluid
- Recall the causes of uroabdomen in dogs
- Understand the differences in uroabdomen causes in cats versus dogs
- Understand the resuscitation steps to stabilize a uroabdomen
- Recall definitive options for uroabdomen treatment

Proceeding:

Introduction: Uroabdomen, also known as uroperitoneum or urinary peritonitis, is defined by the leakage of urine into peritoneal or retroperitoneal cavity. Up to 16% of dogs with traumatic pelvic fractures had urinary tract lesions, including urinary bladder ruptures (7%), urethral ruptures (5%), and avulsed ureters (5%) in 1 study. Other causes of uroabdomen in dogs and cats include spontaneous rupture secondary to urethral obstruction or bladder neoplasia, as well as some iatrogenic causes such as manual bladder expression, cystocentesis or urethral catherization. In cats, blunt abdominal trauma was the most common cause of uroabdomen of cases, when more than 40% was related to urinary catheterization, bladder expression and urethral obstruction.

Clinical signs: The most specific signs may be related to the cause of the uroabdomen (e.g. trauma or urinary obstruction). Otherwise, uroabdomen clinical signs are non-specific and systemic, such as anorexia, lethargy, vomiting, and diarrhea. Some patients may have lower urinary tract signs, such as hematuria, dysuria, stranguria, pollakiuria, lack of observed urinations or not productive attempts to urinate. In case of an acute traumatic event, the patient may have no clinical signs. In case of a chronic process, polydipsia, dehydration, hypovolemia and abdominal pain are commonly reported clinical signs. Definitive diagnosis is made with laboratory evaluation and imaging studies (1).

Diagnostics: Abdominal effusion, detected by physical examination, ultrasound or radiographs, can be sampled by abdominocentesis. Urine within the peritoneal cavity can appear as a transudate, modified transudate, or exudate, with variation caused by hemorrhage or inflammatory cells. Urosepsis can be diagnosed in case of bacterial growth (1). Most diagnosis are established based on a paired

abdominal/venous sample, focusing on creatinine and/or potassium. Most references quote a fluid:serum creatinine or potassium ratio over 2:1, which has a specificity of 100% and a sensitivity of 86% (2). In acute situations, paired abdominal:venous potassium has a better sensitivity and specificity than creatinine, as urinary potassium leaking into the abdomen would have a much higher value than the blood. Specificity and sensitivity of a paired abdominal:venous of 1.4:1 was 100% and 100%, respectively, in one study (2). However, over time, because the potassium is such a small molecule, potassium will be reabsorbed and equilibrate with blood. The most common bloodwork abnormalities associated with uroabdomen are azotemia, metabolic acidosis, and electrolyte derangements including mild hyponatremia, hyperphosphatemia, and hyperkalemia (1). The magnitude and timing of those changes will be detailed (3). In the most recent retrospective data of canine uroabdomen, the location of the urinary tract rupture mimics the rest of the literature, with the urinary bladder (56%), followed by the urethra (26%), kidney (5%) and ureter (2%). Almost 10% of the cases had an undetermined location for the rupture (4). Bladder rupture is more common with a full bladder, as the probability of bladder injury correlates directly with the degree of bladder distension at the time of trauma (1). Ten cases of ureteral rupture following blunt trauma, with avulsion either from kidney or bladder, have been reported. Regarding a urethral cause of uroabdomen, it would occur if the rupture is proximal enough (i.e. close to trigone) in cases of bladder avulsion or secondary to urethral catherization. Otherwise, a distal rupture of the urethral causes urine leakage in perineal tissues and severe tissue injury.

Although the bladder is the major site of rupture, it is important to identify the exact site of the urinary tract rupture for surgical planning (5). Abdominal ultrasound will show variable amounts of abdominal and retroperitoneal fluid. Renal hematomas can be seen with renal trauma. A “bubble study” can be done in order to identify a bladder tear. In some cases, a contrast study may be required to localize the site of urinary rupture. If higher urinary track injury is suspected (i.e. kidney or ureter), an excretory urography can be performed, using caution in patients with dehydration or underlying renal insufficiency. If a lower urinary track (i.e. bladder or urethra) is suspected, a urethro/cystogram can be done (1).

Treatment and prognosis: Initial stabilization includes trauma assessment and stabilization if applicable, with fluid resuscitation and pain control being two important pillars of therapy. Severe potassium disturbances can be treated with intravenous fluid, calcium therapy, judicious use of dextrose and or insulin, bicarbonates, and the re-establishment of an intact urinary tract. Urinary diversion to remove the source of nitrogenous wastes and chemical peritonitis is also very important. That is usually done by placing a urinary catheter in order to keep bladder empty and promote bladder sealing. A peritoneal dialysis or abdominal drainage catheter is also often placed in order to remove the urine from the abdomen and decrease the chemical peritonitis. Surgery is often necessary to repair the source of urine leakage. Ultrasound, contrast studies, or advanced imaging should be performed to localize the urinary tract disruption before surgery as an exploratory laparotomy is not a means to assess the function or patency of the entire urogenital tract. Ureteral tears can be treated with indwelling ureteral stents. Subcutaneous ureteral bypass is another alternative when ureteral stent is not an option. The overall prognosis of dogs and cats with uroabdomen will depends on all aspects of each individual case. Concomitant traumatic injuries, finances and ability to appropriate manage medical and surgical patients can play a role in the overall prognosis. Overall mortality rate in a 2018 study on 43 dogs with uroabdomen was 21% (4).

References:

Stafford JR, Bartges JW. A clinical review of pathophysiology, diagnosis, and treatment of uroabdomen in the dog and cat. J Vet Emerg Crit Care San Antonio Tex 2001. 2013 Apr;23(2):216–29.

Schmiedt C, Tobias KM, Otto CM. Evaluation of Abdominal Fluid: Peripheral Blood Creatinine and Potassium Ratios for Diagnosis of Uroperitoneum in Dogs. J Vet Emerg Crit Care. 2001 Dec;11(4):275–80.

BURROWS C. Metabolic changes due to experimentally induced rupture of the canine urinary bladder. Am J Vet Res. 1974;35:1083–8.

Grimes JA, Fletcher JM, Schmiedt CW. Outcomes in dogs with uroabdomen: 43 cases (2006-2015). J Am Vet Med Assoc. 2018 Jan 1;252(1):92–7.

Tanko MS, Awasum CA, Hassan AZ, Usman B, Jahun BM. Traumatic urinary bladder injuries in small animals. J Vet Med Anim Health. 2015;7(1):27–32.

MANAGING THROMBOSIS: APPLYING THE CURATIVE GUIDELINES

Robert Goggs [1]

[1] Cornell University College of Veterinary Medicine, Clinical Sciences, Ithaca, United States

Learning objectives:

- Understand how and why thrombosis occurs.

- Determine when antithrombotic therapies are recommended.

- Select appropriate therapeutic options for individual patients.

- Understand how to monitor antithrombotics and how to manage complications.

- Recognize the indications for therapeutic thrombolysis.

Proceeding:

Introduction

Thrombosis is the partial or complete obstruction of a blood vessel by a blood clot secondary to local thrombus formation or thromboembolism. Although thrombosis can be clinically inapparent most affected patients will have local clinical signs including pain and swelling and can develop potentially fatal organ dysfunction. The severity of clinical signs reflects the site affected, the degree of vascular occlusion, and the compensatory reserves of affected organs. Early recognition maximizes therapeutic options, minimizes thrombus propagation, and improves the likelihood that fibrinolysis and recanalization will occur.

How and why does thrombosis occur?

Thrombosis involves interactions between platelets, leukocytes, endothelium and plasma coagulation proteases and occurs following exaggerated or misdirected hemostasis and when fibrinolysis is inadequate. Virchow's triad of endothelial damage and dysfunction, blood flow abnormalities, and hypercoagulability remains a useful means to classify disease mechanisms and identify at-risk patients. Multiple abnormalities are often present in patients with clinical thrombosis. Healthy endothelium promotes blood flow through maintenance of an anticoagulant surface and release of vasodilatory signals. The endothelium localizes normal hemostatic processes and encourages restoration of flow by stimulating fibrinolysis. Endothelial injury contributes to the prothrombotic nature of trauma, sepsis, diabetes mellitus, and ischemia-reperfusion. Blood flow abnormalities such as turbulence due to cardiac valve degeneration or indwelling vascular devices alter the shear stress adjacent to the endothelium, promoting interactions between endothelial cells, platelets, and procoagulant proteins.

Hypercoagulability is a state of altered hemostatic balance that favors thrombus formation through increased coagulation factor activity, inappropriate platelet activation, reductions in endogenous anticoagulant availability and a lack of fibrinolysis.

When is antithrombotic therapy indicated?

Thrombosis complicates numerous disorders in both dogs and cats.[8,9] Prior to initiating antithrombotic therapy, an assessment of an individual's risk for thrombosis should be performed, accounting for the underlying condition, the patient's inflammatory state, and the likelihood the underlying condition will resolve. Some diseases are strongly associated with thrombosis, while the risk associated with other conditions is more limited. Hemostatic testing and imaging can aid decision-making, but establishing a definitive diagnosis of thrombosis antemortem is difficult. The CURATIVE guidelines assessed the risk of thrombosis in various diseases and provide recommendations and suggestions regarding which patient populations warrant thromboprophylaxis. Irrespective, if thrombosis is suspected then antithrombotic therapy is warranted, and all patients with known thrombosis should receive antithrombotics.

Antiplatelet agent or anticoagulant?

Venous thromboemboli (VTE) forming under low shear conditions are typically fibrin-rich, while arterial thromboemboli (ATE) forming in high shear environments are platelet-rich. This physiologic distinction is the basis for the use of anticoagulants for venous thrombi, and antiplatelet agents for arterial thrombi. This may be a simplistic view of thrombosis pathophysiology however, and anticoagulants may be valuable adjuncts to antiplatelet agents in arterial thrombosis, and likewise antiplatelet drugs with venous thrombosis.

Anticoagulants

Various parenteral anticoagulants are available including unfractionated heparin (UFH), several distinct low molecular weight heparins (LMWH) and fondaparinux. In addition, several direct oral anticoagulants (DOACs) have been evaluated in dogs and cats. When deciding which drug to prescribe clinicians should consider route and frequency of administration, ease of use, cost, availability, and ease of monitoring. In many cases the LMWHs may be preferable to UFH because of documented efficacy, better safety profiles and more reliable bioavailability. However, the required frequency of administration of the LMWH products may be limiting. Oral anticoagulation is an alternative option for in-patients and out-patient care. The direct factor Xa inhibitors apixaban and rivaroxaban appear to offer effective anticoagulation in dogs and cats, but therapeutic drug monitoring is prudent to ensure efficacy and minimize bleeding risk.

Antiplatelet agents

In cats, data from the FATCAT study strongly supports the use of clopidogrel rather than aspirin for the prevention of arterial thromboembolism in cats with cardiomyopathy. The study did not investigate dual antiplatelet therapy, so it is unknown if there is any benefit of combining aspirin with clopidogrel. The data on antiplatelet therapies in dogs are less clear. Multiple studies suggest that clopidogrel is effective for prevention of provoked arterial thrombosis in dogs, consistent with ex vivo data suggesting clopidogrel has significant antiplatelet effects. In dogs, aspirin may be effective for prevention of ATE,

but no recommendations can be made for a specific aspirin dose in dogs due to the large variation in dosages and efficacy reported.

Monitoring antithrombotic therapies

Clinicians can maximize drug efficacy and minimize adverse effects through therapeutic drug monitoring (TDM). Clinical efficacy of antithrombotic drugs is often judged by the absence of clinical signs which is not ideal since failure of antithrombotic therapy can lead to life-threatening consequences. TDM is essential for UFH and warfarin because of the risk of hemorrhage and is also of value for LMWH and the direct Xa inhibitors. TDM may be useful to confirm acceptable prodrug metabolism (clopidogrel) and adequate platelet inhibition. Some cats may be clopidogrel "non-responders", and might not experience thromboprophylaxis to the expected degree.

Discontinuing antithrombotics

Temporary interruption of antithrombotics may be necessary to facilitate invasive medical or surgical procedures, while drug withdrawal may be warranted when disease remission diminishes thrombotic risk. Risk stratification (i.e. the strength of the association between the disease process and thrombosis) and drug PK/PD underpin recommendations regarding drug discontinuation. Drug PK/PD differ between antithrombotic agents and influence the potential for, and the speed of, reversal of antithrombotic effects following drug withdrawal. For instance, UFH generally has a short half-life in dogs and discontinuation leads to a rapid return of hemostatic potential. In contrast, some residual antiplatelet effect remains for 5-7 days after cessation of an irreversible antagonist like clopidogrel. When deciding whether to continue or to withdraw antithrombotic therapy, clinicians must attempt to balance the risk of thrombosis against the risk of bleeding and to integrate client and patient factors into the decision.

References:

Goggs R, Blais MC, Brainard BM, 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. J Vet Emerg Crit Care. 2019; 29(1):12-36.

deLaforcade A, Bacek L, Blais MC, et al. Consensus on the Rational Use of Antithrombotics in Veterinary Critical Care (CURATIVE): Domain 1-Defining populations at risk. J Vet Emerg Crit Care. 2019; 29(1):37-48.

Goggs R, Bacek L, Bianco D, et al. Consensus on the Rational Use of Antithrombotics in Veterinary Critical Care (CURATIVE): Domain 2-Defining rational therapeutic usage. J Vet Emerg Crit Care. 2019; 29(1):49-59.

Blais MC, Bianco D, Goggs R, 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.

Sharp CR, deLaforcade AM, Koenigshof AM, et al. Consensus on the Rational Use of Antithrombotics in Veterinary Critical Care (CURATIVE): Domain 4-Refining and monitoring antithrombotic therapies. J Vet Emerg Crit Care. 2019; 29(1):75-87.

Brainard BM, Buriko Y, Good J, et al. Consensus on the Rational Use of Antithrombotics in Veterinary Critical Care (CURATIVE): Domain 5-Discontinuation of anticoagulant therapy in small animals. J Vet Emerg Crit Care. 2019; 29(1):88-97.

Martinod K, Wagner DD. Thrombosis: tangled up in NETs. Blood. 2014; 123(18):2768-2776.

Ralph AG, Brainard BM. Update on disseminated intravascular coagulation: when to consider it, when to expect it, when to treat it. Top Companion Anim Med. 2012; 27(2):65-72.

deLaforcade A, Bacek L, Blais MC, et al. 2022 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 (San Antonio). 2022; 32(3):289-314.

Bauer N, Moritz A. Characterisation of changes in the haemostasis system in dogs with thrombosis. J Small Anim Pract. 2013; 54(3):129-136.

Jeffery U, Staber J, LeVine D. Using the laboratory to predict thrombosis in dogs: An achievable goal? Vet J. 2016; 215:10-20.

Goggs R, Chan DL, Benigni L, et al. Comparison of computed tomography pulmonary angiography and point-of-care tests for pulmonary thromboembolism diagnosis in dogs. J Small Anim Pract. 2014; 55(4):190-197.

Mackman N. New insights into the mechanisms of venous thrombosis. J Clin Invest. 2012; 122(7):2331-2336.

Chiarito M, Cao D, Cannata F, et al. Direct Oral Anticoagulants in Addition to Antiplatelet Therapy for Secondary Prevention After Acute Coronary Syndromes: A Systematic Review and Meta-analysis. JAMA Cardiol. 2018; 3(3):234-241.

Castellucci LA, Cameron C, Le Gal G, et al. Efficacy and safety outcomes of oral anticoagulants and antiplatelet drugs in the secondary prevention of venous thromboembolism: systematic review and network meta-analysis. BMJ. 2013; 347:f5133.

Li RH, Stern JA, Ho V, et al. Platelet Activation and Clopidogrel Effects on ADP-Induced Platelet Activation in Cats with or without the A31P Mutation in MYBPC3. J Vet Intern Med. 2016; 30(5):1619-1629.

VISCOLEASTIC (TEG) - GUIDED TRANSFUSIONS: WHAT IS THE EVIDENCE?

Andrew Linklater [1]

[1] Veterinary Specialists of the Rockies, Castle Rock, CO, United States

Learning objectives:

- Understand concepts of viscoelastic (TEG) coagulation testing .

- Identify a viscoelastic tests consistent with hypocoagulation and consistent with hyperfibrinolysis

- Be able to develop sound reasoning as to when transfusions are indicated based on viscoelastic testing.

Proceeding:

Viscoelastic testing is a catch-all term for a variety of methodologies, which examine the ability of whole blood to form a clot and go through fibrinolysis *in vitro*; common methodologies include thromboelastogram (TEG), rotational thromboelastometry (ROTEM) and a veterinary coagulation monitor (VCM). A curve is generated, from which a variety of values are produced. Both the shape of the curve and the generated numbers can tell a lot about a patient's coagulation status; a tracing from a ROTEM machine is depicted below.

Viscoelastic testing in patients suffering from multi-trauma, prolonged or complicated surgery and systemic critical illness clearly demonstrate a variety of coagulation disorders, likely stemming from connections between the coagulation system, inflammatory/immune system, the endothelium and more.

Pairing coagulation assessment such as TEG, along with specific (transfusion) therapy exists, but there is limited evidence in the veterinary field and conflicting evidence in the human field as to how to apply this information in a clinical setting. Although not directly related, the recent CURATIVE guidelines,[1] which discuss the consensus of the rational use of antithrombotic in veterinary critical care do, if nothing else within this context, clearly identify that much more work needs to be done to identify points of intervention and monitoring patients receiving transfusions, anticoagulants and anti-fibrinolytic therapy.

Certainly it is understood that transfusions of any kind are known to have significant adverse events,[2] carry a significant cost, and several indications for specific interventions are poorly defined. To minimize the cost and adverse reactions, a consensus statement on transfusion reactions (TRACS) was recently published which reviews definitions, clinical signs, prevention, monitoring, diagnosis and treatment of transfusion reactions. [2]

With increasing availability and understanding of viscoelastograms, it seems intuitive to consider that administration of transfusions should be, in part, based on these tests. One of the most important questions to ask ourselves is, "Can proper interpretation of a viscoelastogram guide (or maybe even reduce) the use of transfusions?" The following figure is from reference #3 listed below and originates from Parkland Memorial Hospital's Algorithm for ROTEM-guided transfusions in trauma.

Although a lot of TEG-directed transfusion therapy seems inherently logical, there is much yet to be specifically published in the literature demonstrating improvements of outcome. One study performed in 2017 by Mohamed, et al[4] included 134 patients meeting a Class I trauma system activation where 87 patients were enrolled prior to the use of TEG-guided therapy and 47 patients were enrolled after TEG-guided therapy was instituted. In the first 24 hours of treatment, the patients with TEG-guided resuscitation had fewer transfusions administered (RBC and FFP), a shorter hospitalization, a shorter ICU stay and there was a significant cost savings in select patients.

Several meta-analysis have been been published attempting to outline outcomes: a 2016 Cochrane review[5] identified 15 trials with 1185 participants; 8 trials reported that mortality improved slightly from 3.9% to 7% when TEG or ROTEM-guided transfusions were performed. Many of the trials were on cardiac surgery patients which had low levels of evidence, so may not apply to veterinary medicine. There is a significant heterogenicity in the data which is reported, making it difficult to come to solid conclusions, particularly when specific TEG variables (MCF, LY30, etc.) are examined or when subcategories of patients (e.g. head trauma patients) are analyzed separately.

The recently published iTACTIC trial[6] had the objective to determine if augmenting major hemorrhage protocols with viscoelastic assessment improved outcome compared to conventional coagulation. This study was one of the few MCRCCT studies examining mortality at 24 h and 28 d for 400 patients when a resuscitation protocol with augmented with a coagulogram; there was no difference found in primary and secondary outcomes; however, when examining patients with traumatic brain injury, 64% were alive and free of massive transfusion vs 26% with conventional assessment. The question remains, "What is the impact on veterinary patients?"

The veterinary literature is quite limited, but the body of evidence will continue to grow. Langhorn[7] examined a series of four dogs, most of which had a specific disease resulting in a coagulation disturbance which improved with directed therapy. Other veterinary literature focuses on experimental hemorrhage, transfusion impact on coagulograms and specific disease processes (hemoperitoneum) without examining the impact of transfusion, so much is yet to be learned.

References:

Goggs R, Blais M-C, Brainard BM, et al. American College of Veterinary Emergency and Critical Care (ACVECC Consensus of the Rational Use of Antithrombotics in Veterinary Critical Care (CURATIVE) guidelines: Small Animal. J Vet Emerg Crit Care. 2012;2991): 12-36.

Davidow EB, Blois S, Goy-Thollot I, et al. Association of Veterinary Hematology and Transfusion Medicine (AVHTM) Transfusion Reaction Small Animal Consensus Statement (TRACS). Part 1: Definitions and Clinical Signs. 2021 Mar;31(2)::144-166.

Abdelfattah K, Cripps MW. Thromboelastography and Rotational Thromboelastometry use in Trauma, International Journal of Surgery. 2016;33(B):296-201.

Mohamed M, Mjeske K, Sachwani GR, et al. The impact of early thromboelsotography directed therapy in trauma resuscitation. Scand J Trauma Resusc Emerg Med. 2017 Oct 5;25(1):99.

Wikkelso A, Wetterslev J, Moller AM, Afshari A. Thromboelastography (TEG) or thromboelstometry (ROTEM) to monitor haemostatic treatment versus usual care in adults or children with bleeding. Chochrane Database Syst Rev. 2016 Aug 22;2016(*): CD007871.

Baksaas-Asen K, Gall LS, Stensballe J, et al. Viscoelastic haemostatic assay augmented protocols for major traum haemorrhage (iTACTIC): a randomized, controlled trial. Int Care Med 2021 Jan;47(1):49-59.

Langhorn, et al. Thromboelastography-guided transfusion in dogs with hypocoagulable disorders: a case series. Acta Vet Scan. 2019.

URGENT REVERSAL OF ANTICOAGULATION

Vicente Herrería-Bustillo [1]

[1] Universidad Católica de Valencia, Valencia, Spain

Learning objectives:

- Understand the specific scenarios when urgent reversal of anticoagulation may be required.

- Know the specific drugs and treatments required to reverse the most common used anticoagulant and antiplatelet drugs.

Proceeding:

During this lecture we will explore recent advancements in anticoagulation reversal and haemostatic agents across diverse clinical scenarios. A review of studies highlights the need for standardized approaches in anticoagulation reversal, revealing areas of variability. Challenges in accurately dosing protamine, an essential agent for reversing heparin anticoagulation, are discussed, emphasizing precision dosing's importance to avoid adverse outcomes. Furthermore, etamsylate, has been evaluated for its effect on canine blood coagulation and its antagonistic effect on heparin activity, providing a potential solution to the protamine conundrum. The management of clopidogrel reversal in the context of spontaneous haemorrhage is explored, offering valuable insights into managing this challenging clinical scenario.

Strategies for reversing and removing oral antithrombotic drugs in patients with active or impending bleeding are discussed, addressing challenges in managing bleeding complications in cardiovascular patients. Investigation into the reversal of antiplatelet effects of aspirin and clopidogrel provides insights into optimizing platelet function in thrombotic emergencies. Rapid normalization of prothrombin time in dogs with anticoagulant rodenticide toxicosis following intravenous vitamin K1 administration is demonstrated, highlighting an effective treatment approach. Overall, advancements in anticoagulation reversal and haemostatic agents have improved the management of bleeding complications in various clinical settings, emphasizing the need for further research to standardize protocols and enhance patient outcomes.

References:

Milling TJ, Pollack CV. A review of guidelines on anticoagulation reversal across different clinical scenarios - Is there a general consensus? Am J Emerg Med. 2020 Sep;38(9):1890–903.

Hecht P, Besser M, Falter F. Are We Able to Dose Protamine Accurately Yet? A Review of the Protamine Conundrum. J Extra Corpor Technol. 2020 Mar;52(1):63–70.

Wallisch WJ, Kidd B, Shen L, Hammer R, Siscel J. Coagulopathy and Emergent Reversal of Anticoagulation. Anesthesiol Clin. 2023 Mar;41(1):249–61.

Yee J, Kaide CG. Emergency Reversal of Anticoagulation. West J Emerg Med. 2019 Aug 6;20(5):770–83.

Campbell PG, Yadla S, Sen AN, Jallo J, Jabbour P. Emergency reversal of clopidogrel in the setting of spontaneous intracerebral hemorrhage. World Neurosurg. 2011;76(1–2):100–4; discussion 59-60.

Campbell PG, Yadla S, Sen AN, Jallo J, Jabbour P. Emergency reversal of clopidogrel in the setting of spontaneous intracerebral hemorrhage. World Neurosurg. 2011;76(1–2):100–4; discussion 59-60.

Herrería-Bustillo V, Masiá-Castillo M, Phillips HRP, Gil-Vicente L. Evaluation of the effect of etamsylate on thromboelastographic traces of canine blood with and without the addition of heparin. Vet Q. 2023 Dec;43(1):1–6.

Cobo-Nuñez MY, El Assar M, Cuevas P, Sánchez-Ferrer A, Martínez-González J, Rodríguez-Mañas L, et al. Haemostatic agent etamsylate in vitro and in vivo antagonizes anti-coagulant activity of heparin. Eur J Pharmacol. 2018 May 15;827:167–72.

Mooney ET, Agostini G, Griebsch C, Hickey M. Intravenous vitamin K1 normalises prothrombin time in 1 hour in dogs with anticoagulant rodenticide toxicosis. Aust Vet J. 2020 Jun;98(6):225–31.

Cao D, Amabile N, Chiarito M, Lee VT, Angiolillo DJ, Capodanno D, et al. Reversal and removal of oral antithrombotic drugs in patients with active or perceived imminent bleeding. Eur Heart J. 2023 May 21;44(20):1780–94.

Li C, Hirsh J, Xie C, Johnston MA, Eikelboom JW. Reversal of the anti-platelet effects of aspirin and clopidogrel. J Thromb Haemost JTH. 2012 Apr;10(4):521–8.

CHALLENGING COAGULATION CONUNDRUMS

Robert Goggs [1]

[1] Cornell University College of Veterinary Medicine, Clinical Sciences, Ithaca, United States

Learning objectives:

- Recognize how distinct disorders of hemostasis typically present clinically.

- Use diagnostic tests to screen for and diagnose bleeding disorders.

- Understand how a reference laboratory can aid coagulation disorder diagnosis.

- Use coagulation laboratory diagnostic test data to establish definitive diagnoses.

- Use coagulation laboratory diagnostic test data to guide therapeutic decision-making.

Proceeding:

Introduction

The hemostatic system is generally conceptualized as primary hemostasis, secondary hemostasis, and fibrinolysis. Primary hemostasis refers to the formation of a platelet plug following platelet tethering and activation by VWF and extracellular matrix components exposed by damage to the endothelium. Secondary hemostasis involves assembly of plasma serine protease complexes to generate sufficient thrombin to cleave fibrinogen into fibrin that stabilizes the platelet plug. Fibrinolysis is the process of clot dissolution following repair of the damaged endothelium.[3] These distinctions are less apparent in vivo where thrombus formation occurs in a cell-dependent manner on the surface of activated platelets, and initiation, extension, stabilization and dissolution of the thrombus occurs as a continuum. Dividing the process of clot formation up into components is a didactic convenience but it remains clinically useful. The dysfunctional component of a patient's hemostatic system can often be predicted based on their clinical presentation. Priorities for further investigation and treatment might be discerned from physical examination alone.

Diagnostic evaluation

Efforts should be made to stabilize the major body systems of all bleeding patients prior to undertaking specific diagnostic testing. Collection of critical diagnostic samples during initial stabilization enables early diagnosis and prevents the underlying cause being obscured by therapeutic interventions. Blood samples should be collected by clean venipuncture (i.e. where only one passage of the needle is required to enter the vessel) of a peripheral vessel to enable external pressure to be applied. Alternatively, samples can be collected at the time of intravenous catheter placement. It is acceptable to draw blood

through the catheter for coagulation testing. Initial blood samples should be collected into 3.2% sodium citrate (coagulation testing), gel separator tubes (serum biochemistry) and finally into K^+EDTA (complete blood count) for subsequent analysis. Samples should be carefully assessed for clot fragments, hemolysis and lipemia before analysis.

A full assessment of the patient should be performed once initial stabilization is underway. In addition to a full physical examination, care should be taken to thoroughly examine mucous membranes and skin (esp. axillae and inguinal areas) for petechiae or ecchymoses. A gentle rectal examination should be performed to check for hematochezia or melena. Once the patient is stable a thorough history should be obtained from the client.

If the history and physical examination findings are consistent with a primary hemostatic defect the first rule out is thrombocytopenia. This is best achieved through in-house examination of a fresh blood smear ideally made from an EDTA-anticoagulated sample. The number of platelets visible per x100 field, multiplied by 15 gives a rough estimate of platelets ×10/L. Counts < 50 ×10/L may be associated with spontaneous hemorrhage. The feathered edge must be checked for platelet clumps before thrombocytopenia is diagnosed.

Patients with a primary hemostatic bleeding phenotype with a verifiably normal platelet count may have von Willebrand's disease or a thrombocytopathy (platelet dysfunction). Von Willebrand's disease is best diagnosed by measuring the circulating VWF antigen level via species-specific antibodies. Platelet function disorders may be acquired or congenital. Most acquired platelet function disorders are mild. Various rare inherited disorders of platelet function are described yet can be challenging to definitively diagnose. Specialist advice should be sought if one is suspected. Point-of-care tests of platelet function are available that may aid in the initial screening of suspected thrombocytopathy patients. Definitive diagnosis is typically performed by flow cytometry, platelet aggregometry or by genetic analysis for the underlying causal mutation.

If physical examination findings indicate a secondary hemostatic disorder, this suggests either inadequate clotting factor production, excessive factor consumption or a combination of both processes. Many assays of secondary hemostasis are available. The prothrombin and activated partial thromboplastin times are typically assessed first during the initial screening for secondary hemostatic defects. The prothrombin time (PT) evaluates the extrinsic and common pathways and thus evaluates the concentrations of factors VII, X, V and II. The activated partial thromboplastin time (aPTT) evaluates the intrinsic and common pathways and thus evaluates factors XII, XI, IX, VIII, X, V, II. Factor activity needs to be significantly decreased before these times become prolonged. Point-of-care analyzers can measure the PT and aPTT, but it is generally recommended that abnormalities be confirmed by a reference laboratory before further testing is performed or a definitive diagnosis is made. Individual factor activities can be measured by reference laboratories.

Hyperfibrinolytic disorders are infrequently diagnosed in veterinary medicine and can be primary or secondary. Primary hyperfibrinolysis disorders such as deficiencies of plasminogen activator inhibitor-1 (PAI-1), α_2-antiplasmin or FXIII and Quebec-platelet disorder occur in the absence of a thrombotic state. Secondary hyperfibrinolysis is more common and results from increased fibrinolytic activity following coagulation system activation. Disseminated intravascular coagulation is the most common cause of

secondary hyperfibrinolysis, while trauma associated coagulopathy is also characterized by hypocoagulation and hyperfibrinolysis and is seen in very severely injured patients. Secondary hyperfibrinolysis has been demonstrated in dogs with spontaneous hemoperitoneum, and following envenomation by some snakes.

Treating the coagulopathic patient

Transfusion is the cornerstone of treatment of coagulopathies in veterinary patients. Identification of the nature of the bleeding phenotype is important to tailor component therapy based on patient need. Component therapy to provide only those parts of whole blood that are required to treat the bleeding disorder is preferable to minimize risks including volume overload, polycythemia, or immune-mediated transfusion reactions. Component therapy is also more economical since several patients can then be treated with a single donated unit of fresh whole blood. Platelet-rich plasma and platelet concentrates are not widely available. These products may be produced either through low-speed centrifugation of one or more FWB units or by apheresis. Unfortunately, these products have short half-lives ~5 days and must be kept at room temperature. They are particularly useful for treatment of symptomatic thrombocytopenia or thrombocytopathia. Owing to the difficulty producing and storing these products, attention has focused on producing stable, functional “off-the-shelf” platelet products. Examples include DMSO stabilized platelets and lyophilized platelets. These products are not widely available and there is limited documentation of their safety and efficacy in clinical patients at this time. Specific factor concentrates and pharmaceutical therapies for hemorrhage associated with primary or secondary hemostatic defects are available in human medicine. Currently there is extremely limited information on the use of these products in veterinary medicine. In addition, the majority are prohibitively expensive or are simply not available for use in veterinary patients.

References:

McMichael M. J Vet Emerg Crit Care 2005; 15:1-8.

Hopper K, Bateman S. J Vet Emerg Crit Care 2005; 15:83-91.

Cesarman-Maus G, Hajjar KA. BJH 2005; 129:307-321.

Hoffman M, Monroe DM. Thromb Haemost 2001; 85:958-965.

Smith SA. J Vet Emerg Crit Care 2009; 19:3-10. 6. Smith JW et al. Comp Contin Edu Pract Vet 2005; 27:828

Bauer NB et al. Am J Vet Res 2011; 72:64-72.

Boudreaux MK. J Am Vet Med Assoc 2008; 233:1251-1259. 9. Rozanski E, de Laforcade AM. Clin Tech Small Anim Prac 2004; 19:83-87.

Yaxley PE et al. J Vet Emerg Crit Care 2010; 20:472-478. 11. Tocci LJ. Vet Clin North Am Small Anim Pract 2010; 40:485-494.

Callan MB et al. J Vet Emerg Crit Care 2009; 19:401-415.

Guillaumin J et al. Am J Vet Res 2008; 69:1580-1586.

Guillaumin J et al. J Vet Emerg Crit Care 2010; 20:571-577.

Bode AP et al. Clin Appl Thromb Hemost 2008; 14:38-54.

Lillicrap D. Curr Opin Hematol 2010; 17:393-397.

Key NS, Negrier C. Lancet 2007; 370:439-448. 18. Iorio A et al. Cochrane Database of Systematic Reviews 2010(8).

Franchini M, Lippi G. Semin Thromb Hemost 2010; 36:485-492.

Lin Y et al. Cochrane Database of Systematic Reviews 2011; 2:CD005011.

Kristensen AT, et al. Vet Clin North Am Small Anim Pract 2003; 33:1437-1451.

Nurse & Tech Stream, Thursday 30 May 2024

THE ART OF TRIAGE

Angela Elia [1]

[1] Pieper Veterinary Group, Pieper Veterinary Group , Middletown, CT, United States

Learning objectives:

- Describe the body systems that are a part of every triage exam

- Describe the best techniques to obtain vitals and information during your exams

- Successfully sort patients into a triage system that manages the flow of a busy ER

- Describe special considerations for exotic pet triages

Proceeding:

Triage Definition

Means “To Sort”

In clinical practice means to rapidly assess patients to determine which one needs access to treatment first.

Characteristics of a Great Triage Nurse

Knowledge of basic medical terminology, conditions and how to communicate these to owners

Good grasp on emergency medicine

Critical thinking skills

High level communication skills

Ability to maintain calm in high stress situations

Confident in nursing skills

Role of the Triage Nurse

Arrive to the triage room in a timely manner

Greet owners and identify your role

Visualize all incoming patients – sometimes in passing, or while seeing other cases

Be an advocate for the patient and the client

Maintain client and patient confidentiality and privacy

Provide prescribed treatments to patients and provide repeat assessments as needed

Keep both the doctor and owners informed on case progress

Triage Objectives

Identify which patients need immediate care

Communicate with owners about the status of their pet and their expected wait times to be seen

Organize and drive patient flow through the ER and treatment areas

Triage is not a 'one and done' situation. Assessment and reassessment of vitals and updating owners of situation is important.

Triage Components

Across the Room Assessment

Patient history gathering

Visual patient assessment (hands off)

Primary Survey

Respiratory

Circulatory

Neurological

Urogenital

Triage Decision

Assign the appropriate triage level to each patient

Secondary Survey

Reassessment of vital parameters if significant time has passed

Full physical exam

Prescribed treatments

Types of Triages

Telephone Triage

Gather some information to help the team:

Owner name

Pet name and signalment

Location in relation to the hospital or arrival time

Presenting complaint

Acute or chronic condition/change

Pertinent medical history

Current medications and last dose given

Keep in mind that critical cases should be handled as quickly as possible to avoid delays

The Toxin Triage

Most toxin triages begin over the phone when panicked owners find their pet has ingested something dangerous. It is important to gather information such as the type of pet, the type of toxin, as well as the time and route of exposure. Many practices advise owners to contact a poison control facility while en route to the clinic. A complete list of information needed to create a case with poison control is as follows:

Owner name and Pet name

Pet weight

Exact toxin exposed to

Route of exposure

Amount ingested/exposed to

Time of exposure

Current condition of pet

The In-Person Triage

While introducing yourself and your role, a rapid visual assessment can be done to determine whether the pet is in obvious critical condition. If you determine that the pet is unstable or in questionable condition, your priority changes from getting a complete history to providing access to treatment. Gently communicate the severity of the situation to the client, give an estimate of stabilization costs and get permission for treatment to begin. Do not forget to get the owners consent for CPR before taking the pet to the treatment area.

The Exotic Pet Triage

Exotic animals are at a higher risk than other pets to suffer the physiologic consequences of fear and stress responses. It is important to treat exotic animals with the most minimal and stress-free handling techniques. If there is any complaint or evidence of respiratory effort or distress, provide oxygen

immediately and perform a cage side exam until you can assess the patient with the DVM. There are many parameters that can be assessed through the cage or enclosure of an exotic pet such as breathing rate and effort, nasal and ocular discharge, mentation status and evaluation of fecal matter if present in the cage. Many problems with exotic pets arise from poor husbandry practices. For this reason, it is imperative to gather information from the owner about lighting, heat sources, diet and substrate.

References:

Silverstein D, Hopper K. Small Animal Critical Care Medicine. 2nd Ed. St. Louis: Saunders; 2015.

Battaglia AM, Steele AM. Small Animal Emergency and Critical Care for Veterinary Technicians. 4th ed. St. Louis: Elsevier; 2021.

Norkus, CL. Veterinary Technician's Manual for Small Animal Emergency and Critical Care. 2nd Ed. Hoboken: John Wiley & Sons; 2019

Plunkett SJ. Emergency Procedures for the Small Animal Veterinarian. 2nd ed. St. Louis: Saunders; 2013.

MDB (MINIMUM DATA BASE) AS SIMPLE AS 1-2-3

Nathalie Hack [1]

[1] Vets Now, Emergency Roving team Area D Vets Now, Dunfermline , United Kingdom

Learning objectives:

- In this lecture the minimum date base will be explained in great detail.

- Firstly it will cover what it actually is and how much blood and urine needs to be collected to perform the following tests quickly and adequately: Glucose, Lactate, PCV and TS, BUN and urinalysis.

- At the end of this lecture the listener should have a greater understanding of this subject and be confident to perform these tests in practice.

Proceeding:

MDB stands for Minimum Data Base

Consists of:

Blood glucose (BG)

Lactate (LAC)

Manual Packed Cell Volume (PCV)/ manual haematocrit

Plasma total solids (TS)

Blood urea Nitrogen (BUN)

Urinalysis

What are the benefits?

Only a very small amount of blood is needed

Very quick assessment that can be done in house

Increases patient care

Can give you a lot of information in a very short space of time

Blood Collection

Only a very small amount is needed.

Can be done with fresh blood if the opportunity is there to run the tests immediately.

Blood can be collected in either a heparinised syringe or collected in a lithium heparin

Blood Glucose (BG)

Normal range : 3.3 - 7.2 mmol/L

Results will either be euglycaemic (normal), hyperglycaemic (high) or hypoglycaemic (low)

Lactate (LAC)

Lactate is the best indicator for perfusion

When O2 is inadequately delivered to the cells, lactate builds up as it is an anaerobic by-product of glycolysis. A high lactate concentration in the blood will indicate that an inadequate perfusion and oxygenation of the cells in the body are occurring.

If left untreated, elevated lactate levels could progress to organ dysfunction and death

Normal lactate measurement:

Dog : <2.5 mmol/l

Cat : < 1.5 mmol/l

Mild increase : 3-5 mmol/l

Moderate increase : 5-10 mmol/l

Severe increase >10 mmol/l

Lactate would only increase in a dehydrated patient that had hypovolaemia because of the degree of dehydration and fluid shifts

Two types of lactic acidosis : type A and type B

A one-off lactate reading is not sufficient to make a prognosis

Frequent rechecking is useful

PCV

Packed cell volume or haematocrit

Percentage of red blood cells circulating in the blood

Blood is collected in a capillary tube:

Fresh blood in a heparinised tube (red)

Heparinised blood in a plain tube (blue)l

BUFFY COAT

Gives some idea of the WBC count

PLASMA

Normal : clear yellow fluid

Pink/red : haemolysis

Milky white : hyperlipidaemia

Yellow/orange : icterus (jaundiced)

TS Total solids or total proteins

TS is a measurement of plasma proteins. These proteins include albumin, globulins and fibrinogens.

The normal ranges for TS (as referenced from Merck Veterinary Manual) are :

Dog 60 – 75 grams/litre

Cat 60 – 75 grams/litre

PCV and TS

PCV and TS should always be interpreted together.

BUN

Sensitive colour coded test strips

Measure the amount of Blood Urea Nitrogen

Non-specific test for renal function

Dehydration, anorexia and GI disease can also affect the BUN

Only require 1 drop of blood

Results are ready within 90 s

NORMAL : 0-8.9 mmol/L

ABNORMAL : 9-28.6 mmol/L, further testing is recommended

Urinalysis

Indication of various urinary tract diseases and potentially other systemic diseases such as haemolysis and liver disease

Urine collection:

Voiding

Catheterisation

Cystocentesis : most common approach to obtain a sterile sample

Ideally evaluated within 30 min

Not much is needed

Urine appearance :

Odour

Ammonia

Depends on the concentration of the urine

Urine of a cat is strong smelling due to its composition

Malodorous due to an infection or yeast present

Turbidity

Typical clear

May become turbid because of blood, crystals, pus,...

Colour

Transparent

Amber or yellow coloured

Dehydrated animals will have a darker colour

Specific gravity

Ratio of the volume of liquid compared with the same volume of distilled water

Depends on the number, size and weight of the particles

Is the ability to concentrate urine

SG of urine will depend on what time of the day , esp. cats

Dogs : 1.015-1.045

Cats : 1.035-1.060

Urine reagent (dipstick)

Avoid human ones as the panels for urobilinogen, nitrates and leukocytes are not diagnostic in veterinary medicine

Ketones

Only detects acetoacetate

Blood

Can be an artefact after cystocentesis

PH

Acidic in dogs and cats

Alkaline in horses and ruminants

Protein

Detects albumin in urine

Any positive result might raise suspicion

A positive reaction must be interpreted with the SG, pH and urine sediment

Glucose

If glucosuria is present, blood glucose needs to be determined

Bilirubin

Hyperbilirubinuria is abnormal in cats, but might be normal in dogs

AN INTERACTIVE CASE DISCUSSION: THE ACUTE ADDISONIAN PATIENT

Chloe Fay [1]

[1] New Priory Vets, Brighton, United Kingdom

Learning objectives:

- Define the pathophysiology of hypoadrenocorticism

- Demonstrate an understanding of clinical symptoms and the nursing interventions

- Provide the skills to actively solve problems, transferring theoretical ideas to actual situations

- Develop the ability to react in real-time, imitating the ECC environment

Proceeding:

Pathophysiology

The adrenal glands, located near the kidneys, are part of the endocrine system and produce hormones to regulate homeostasis. These include corticoids and corticosterone (glucocorticoids) regulated by adrenocorticotropic hormone (ACTH), and are responsible for the movement of glucose. These hormones help to increase blood glucose (BG) levels by reducing cell uptake of glucose, aiding gluconeogenesis, and converting fatty acids back to glucose. Adrenal glands also produce aldosterone, a mineralocorticoid, which regulates the function of the kidney and acid/base, by affecting 'minerals'- electrolytes and the excretion of them. In hypoadrenocorticism, either/both cortisol and aldosterone are deficient, and the clinical signs will vary accordingly. The deficiency of these hormones can happen for various reasons including; bacteria/parasitic agents, haemorrhage, neoplasia, iatrogenic or idiopathic causes. Clinical signs include dysregulated sodium, potassium and chloride levels when deficiency in mineralocorticoids is present. Due to the kidneys inappropriately retaining potassium, hyperkalaemia is often present with changes to the electrocardiogram (ECG) and bradycardia, while the inability to reabsorb sodium and chloride causes rapid excretion of these electrolytes, causing hyponatraemia and hypochloraemia. The profound hyponatraemia, causes lethargy, depressed mentation, seizures from cerebral oedema, as well as coma and death. Aldosterone has a direct effect on extracellular fluid; extracellular fluid volume will decrease as the sodium decreases causing dehydration and stimulating polydipsia. In addition, the decrease of corticosteroid levels will concurrently decrease gluconeogenesis, thus creating a mild to moderate hypoglycaemia. Other clinical signs include anorexia, abdominal pain, vomiting, weakness, diarrhoea (often haemorrhagic), respiratory, circulatory and cardiac failure. Acute

presentation may include signs of hypovolaemia; decreased capillary time, weak/ absent pulses, bradycardia, collapse, requiring immediate attention.

Diagnostics and Treatment

A complete blood count, biochemistry including electrolytes, blood gas analysis and ACTH stimulation test are all indicated. Often a mild anaemia will be present, potentially masked by dehydration, as well as hyponatraemia, hyperkalaemia with a sodium/potassium ratio less than 27:1. Diagnostics may also reveal azotaemia, hypocalcaemia, hypoglycaemia and metabolic acidosis. Treatment should focus on correcting these diagnostic results, as well as any hypotension and hypovolaemia that may be present on clinical exam. Intravenous fluid therapy, including shock bolus administration, should be implemented and monitored through the use of blood pressure (BP), heart rate (HR), respiratory rate (RR), and caudal vena cava monitoring. Choice of fluid therapy is widely disputed. Historically, if Addisons was suspected, 0.9% NaCl was considered to be the best choice due to the higher amount of sodium. However, rapid rises in sodium from the administration of 0.9% NaCl can result in neurological effects due to osmotic injury of the neuron, leading to death. 0.9% NaCl is considered an acidotic fluid, which can exacerbate any metabolic acidosis. A balanced isotonic crystalloid, is more beneficial in these patients, as they increase the sodium at a slower rate, avoiding osmotic injury from rapid sodium correction. Dextrose supplementation should be considered in hypoglycaemic patients, and these patients may benefit from placement of a central catheter to enable higher % dextrose administration, as well as avoiding patient discomfort and pain from multiple blood samples throughout the hospital stay. Patients with severe hyperkalaemia, should have treatment to correct the potassium levels. Dextrose administration may be sufficient enough, causing the release of endogenous insulin to drive potassium back into the cells. In more severe cases, regular insulin may be required to do this; this should be followed up with a 2.5-5% dextrose CRI has corrected levels. If a patient has a bradyarrhythmia, calcium gluconate may be beneficial in protecting the cardiac myocytes from the effects of the hyperkalaemia. Once the patient is stabilised cardiovascularly, the focus of treatment should be to provide glucocorticoid replacement. This therapy will continue to assist the electrolyte imbalances. The most appropriate glucocorticoid is dexamethasone, as it is the only corticosteroid that will not react to the ACTH stimulation test.

Nursing Considerations

Further considerations for these patients, in addition to monitoring BP, HR, RR (and effort), ECGs, BG, would be to weigh the patients twice daily, monitor urine output and other dehydration parameters. Any weakness, lethargy, anorexia, vomiting, ataxia may be telling signs that the patient is not responding to the glucocorticoid therapy. It is important that nutrition is not neglected in these cases, as well as gastrointestinal support; feeding tubes or parental nutrition may be considered in anorexic patients. Finally, reducing stress during the hospital stay is paramount, due to the patient's inability to mount an appropriate stress response, this may include fear free handling and calm, quiet wards.

References:

Burkitt Creedon JM. Hypoadrenocorticism. In: Silverstein DC, Hopper K, editors. Small Animal Critical Care Medicine. 2nd ed. St. Louis, MO: Elsevier; 2015:380-383.

Feldman EC, Nelson RW. Canine and Feline Endocrinology and Reproduction, 3rd ed. St. Louis: WB Saunders, 2004:394-439.

Koenig A. Hypoglycemia. In: Silverstein DC, Hopper K, editors. Small Animal Critical Care Medicine. 2nd ed. St. Louis, MO: Elsevier; 2015:352-357.

Lanen Van K, Sande A. Canine hypoadrenocorticism: pathogenesis, diagnosis, and treatment. Top Companion Anim Med, 2014;29(4):88-95.

Pariaut R, Reynolds C. Bradyarrhythmias and conduction disturbances. In: Silverstein DC, Hopper K, editors. Small Animal Critical Care Medicine. 2nd ed. St. Louis, MO: Elsevier; 2015:246-249.

TOXICOLOGICAL EMERGENCIES

Angela Elia [1]

[1] Pieper Veterinary Group, Pieper Veterinary Group , Middletown, CT, United States

Learning objectives:

- List several types of substances that create toxicities in felines and canines

- List the mechanism of action that each toxin uses to create symptoms in patients

- List the available treatments for each toxicity

- Describe the mechanism of action for each antidote used in treatment

Proceeding:

Toxicology Vocabulary

Toxicant: Any solid, liquid or gas, that when introduced to the body, interferes with its homeostasis

Toxin: Poisons that originate from biological sources

Zootoxins: Toxins that originate from animals

Mycotoxins: Toxins that originate from fungus

Bacterial Toxins: Toxins that originate from bacteria

Phycotoxins: Toxins that originate from seaweed and algae

Poisons: Can be classified as organic, inorganic, metallic or biologic. Can be naturally occurring or synthetically manufactured.

Dosage: Amount of toxicant per unit of body weight

Dose: The total amount given or exposed to

LD50: Median Lethal Dose. Usually calculated from one bolus dose, but can also be the result of effects of multi-dose exposure over multiple days. This is the amount of toxicant it takes to be fatal in 50% of a tested population.

Toxicity Classifications:

Acute: Effects of a single dose or multiple doses in a 24-hour period up to one week in some cases

Subacute: Effect seen between 1 week and one month of exposure

Subchronic: Effects seen between 30-90 days of exposure

Chronic: Effects that are produced by prolonged exposure of 3+ months

Factors that Influence Toxicity:

Route of exposure: The route of exposure matters greatly. For example, rubbing a chocolate bar on a dogs fur will not create a chocolate toxicosis, but if the dog licks the chocolate off or eats the whole bar, then we have a route of exposure wherein the chocolate can cause harm.

Concurrent Disease and/or Organ Disfunction: When a toxicant's effects are targeted towards a specific organ group, any previous or concurrent disease or dysfunction can increase the effects of the toxicant.

Breed/Species Sensitivities: Some breeds and species make them more susceptible to toxicosis due to innate sensitivities they have in their genetics. Some examples would be ivermectin sensitivity in collie breeds, felines' inability to process acetaminophen, and avian sensitivity to cookware coatings.

Toxicokinetics:

Toxicokinetics looks at the pathway a toxicant takes through a body from exposure route to elimination. Some key words to remember are:

Absorption: How the body absorbs the toxicant

Distribution: The pathway the toxicant takes trough the body

Storage: How and where the body stores the toxicant

Metabolism: What organ is processing the toxicant and how quickly it does so

Elimination: How the toxicant leaves the body

ABC's of the Poisoned Pet

A: Airway

Any patient that comes in with respiratory impairment or distress should have airway management addressed immediately

Monitor SPO2 and ETCO2 if intubated

B: Breathing

Many toxicants alter breathing ability. This can result in hypoventilation and hypoxemia.

Supplemental oxygen should be provided

C: Circulation

Assess circulation by assessing

Mentation

MM color

CRT

Heart rate and pulse quality

Decontamination

Ocular: Flush the eyes with saline – if owner can do so at home before coming in, this can greatly help to minimize damage. E-collar will be essential to prevent self-trauma to the eye while treating/recovering.

Dermal: When decontaminating, it is important to also protect yourself from dangerous materials by wearing the appropriate PPE.

Bathing: You may need to bathe several times to remove all of the toxicant and to prevent further absorption.

If it is an oil-based toxin, use mild dish soap/detergent to remove from skin/fur

Gastro-Intestinal:

Emesis can be induced easily in canines, while their feline counterparts pose much more of a challenge. Emesis in any species can cause complications such as aspiration pneumonia, hematemesis, and caustic/corrosive injury to the esophagus and oropharynx. Owners should always be made aware of the risks involved, but typically the treatment of the toxicosis outweighs the risks of emesis complications. There are many products that can be used to induce emesis. The most common are listed below:

Ropinirole (Clevor) eye drops

Apomorphine

For felines some commonly used drugs are:

Xylazine

Dexmedetomidine

Hydromorphone

Preventing further Absorption

Activated Charcoal

Activated charcoal is a nonspecific adsorbent that binds to toxins in the GI tract. The typical dose ranges from 1-4 grams/Kg. The dose is given orally or by gastric tube. Doses may need to be repeated for some toxicants that undergo enterohepatic circulation. Activated charcoal often comes with the additive Sorbitol. Sorbitol is a cathartic that speeds up the time it takes to travel through the GI tract. This is important because some toxicants can unbind from AC over time and be reabsorbed before elimination.

Some complications seen with the use of AC are aspiration of the liquid during forced administration, dehydration, and hypernatremia.

Lipid Emulsion Therapy (LET)

Lipids emulsion works very well for fat-soluble toxicants such as ivermectin, baclofen, pyrethrin, Ca+ channel blockers and cholecalciferol. Lipid emulsion has a separate plasma compartment that can store lipophilic agents known as a "lipid sink". The typical dose for LET is a bolus of 1.5 ml/kg over 5-15 minutes followed by a CRI of 0.25 ml/kg/min. LET must be administered with a filter using aseptic techniques.

Supportive Care

The level and type of care a toxicosis patient will vary greatly depending on the conditions previously covered. The most common treatments outside of direct antidote administration are as follows:

Vitals Monitoring: ECG, SPO2, Temperature, Blood Pressure

Fluid Therapy

Cardiovascular support: antiarrhythmic agents

GI support: Protect from ulceration, antiemetics

Famotidine, Omeprazole, Pantoprazole, sucralfate, maropitant

Neurological Support: Neuro assessments, anticonvulsants, muscle relaxers

Sedation

Analgesia

*****Due to proceeding length restrictions, a list of toxicants and their specific antidotes are purposely not included. Instead, I encourage individuals and clinics to purchase any/all of the amazing resources listed below to use as guides for treatment of toxicities.

References:

Hovda L, Brutlag A, et al. Blackwell's Five-Minute Veterinary Consult: Small Animal Toxicology. 2nd ed. Wiley-Blackwell; 2016.

Talcott A, Peterson M. Small Animal Toxicology. 3rd ed. St. Louis. Saunders; 2012.

Silverstein D, Hopper K. Small Animal Critical Care Medicine. 2nd Ed. St. Louis: Saunders; 2015.

Battaglia, Steele. Small Animal Emergency and Critical Care for Veterinary Technicians. 4th ed. St. Louis: Elsevier; 2021.

Norkus, CL. Veterinary Technician's Manual for Small Animal Emergency and Critical Care. 2nd Ed. Hoboken: John Wiley & Sons; 2019.

Plunkett SJ. Emergency Procedures for the Small Animal Veterinarian. 2nd ed. St. Louis: Saunders; 2013.

WHEN SUGAR TURNS SOUR: FELINE DKA

Lindsey Ashburner [1]

[1] University of Edinburgh, University of Edinburgh, Edinburgh, United Kingdom

Learning objectives:

- Understand the pathophysiology of diabetic ketoacidosis (DKA)

- Recognise commonly seen clinical signs

- Diagnosis and treatment of DKA

- Nursing considerations for the feline DKA patient

Proceeding:

Introduction

Diabetic ketoacidosis (DKA) is a potentially fatal condition and is not an uncommon emergency in both newly diagnosed and poorly regulated cats with diabetes mellitus. Diabetes mellitus results either from an insulin deficiency or insulin resistance (impaired action of insulin). This insulin plays a vital role in promoting glucose uptake by the cells, to provide cellular energy. When this process cannot occur effectively what results is that glucose remains in the bloodstream where the body cannot utilise it and the body therefore breaks down lipids instead to provide an alternative energy source. The metabolism of lipids for energy production causes fatty acids to be released into the bloodstream where they are converted into ketones and triglycerides by the liver. DKA occurs when the process of ketone formation and metabolism becomes unregulated. This can be due to a secondary medical condition that places additional stress on the cells and body, increasing the bodies energy need, causing a spike in glucose demand. Some of these conditions include pancreatitis, infection (commonly urinary tract), hepatic lipidosis, kidney disease and neoplasia.

Clinical signs and diagnosis

The most common clinical signs of DKA include depression, anorexia, and vomiting. Weakness and gait abnormalities may also be observed. Owners may also report clinical signs of hyperglycaemia such as polyuria, polydipsia, and weight loss. Physical examination findings can include signs of poor perfusion such as pale mucus membranes, hypotension, and hypothermia. Altered mentation, dehydration, poor body condition, tachypnoea and, in some cases, a sweet smell on the breath (often likened to pear drops) can be smelt secondary to the presence of exhaled ketones. Laboratory confirmation of DKA is based on the presence of hyperglycaemia, glucosuria, ketonemia or ketonuria, and a metabolic acidosis.

Treatment

The treatment goals of DKA are to restore hydration through fluids, restore glucose as the main energy source through insulin therapy, stop ketone production, and correct any electrolyte abnormalities. Intravenous fluid therapy should be initiated prior to any insulin therapy in an aim to correct dehydration and hypoperfusion, as well as any electrolyte abnormalities. There are a few fluid choices available, and the most suitable option should be selected based on the individual patient and their electrolyte/ acid-base status. Electrolyte supplementation is frequently required in our DKA patients as the insulin treatment required to reduce the ketone and glucose concentrations will move potassium (and to a lesser extent phosphate) into cells, reducing circulating levels. Neutral insulin is administered to promote normoglycaemia and eliminate ketone bodies before the patient is then transitioned onto a longer-acting insulin. This neutral insulin can either be administered via intermittent intramuscular injections or a constant rate infusion (CRI). The goal of insulin therapy is to maintain the patient's blood glucose between 8-15mmol/L. If the glucose drops below 8mmol/L, a separate 5% dextrose solution should be administered alongside the insulin therapy. Once the patient's condition stabilised, their dehydration is reverses and they are consistently eating, they can be transitioned to longer-acting insulin.

Nursing care

Following the initial volume replacement and insulin therapy, close monitoring of these patients is needed to recognise alterations in fluid balance, prevent hypoglycaemia, and identify and treat persisting or developing acid-base and electrolyte abnormalities. Sampling lines such as central venous catheters or peripherally inserted central catheters should be considered in these patients due to the need for regular blood glucose assessments (every 1-2 hours) and electrolyte assessments (every 4-8hours). These catheters allow for regular blood draws without the need for repeated venepuncture as well as the administration of fluid therapy and insulin solutions. The patient's nutritional status should also be taken into consideration as they often present with anorexia and vomiting. Appropriate supportive treatments such as anti-nausea medication should be administered and, if the patient has ongoing anorexia despite this, an enteral feeding tube should be placed. This will allow for assisted feeding to meet the patients resting energy requirements.

Conclusion

DKA cats can be very rewarding patients to nurse as they give us the ideal opportunity to use more practical skills such as placing sampling lines, calculating, and administering various CRI's, placing and managing enteral feeding tubes, and interpreting blood gas results.

References:

Feldman EC, Nelson RW., Feline diabetes mellitus, in: Feldman EC, Nelson RW (eds), Canina and Feline endocrinology and reproduction, 3rd ed, Philadelphia: Elsevier Science, 2004, pp 539-579.

Higgs P., Investigating diabetic ketoacidosis, Vet Times, available from https://www.vettimes.co.uk/article/investigating-diabetic-acidosis/ Kerl ME., Diabetic ketoacidosis: treatment recommendations, Compend Contin Educ Pract Vet 2001; 23, pp 330-339.

Merrill L., Small animal internal medicine for veterinary nurses and technicians: Iowa: Wiley-Blackwell, 2012. Rand JS., Pathogenesis of feline diabetes, Vet Clin North America Small Animal Practice 2013: 43, pp 221-231.

LIGHTS, CAMERA, ANAESTHESIA! NAVIGATING COMPLICATIONS

Lisa Angell [1]

[1] Royal Veterinary College, United Kingdom

Learning objectives:

- Explain the five most common anaesthesia related complications seen in small animals.

- Apply knowledge to identify the anaesthesia risks during planning phases, to attempt to reduce the risk before and during anaesthesia where possible.

- Recognise the complication when monitoring the patient under anaesthesia by having an understanding of normal and abnormal parameters.

- Trouble-shoot the most likely cause for the complication, and take a step-by-step approach to implementation of treatment.

Proceeding:

Anaesthesia doesn't come without risk, and this risk increases when the patient presents as emergency or when they are critically unwell. Nurses working in the ECC department are often asked to anaesthetise these patients for a variety of reasons, including diagnostic imaging and for emergency surgery.

As anaesthetists, planning is key, and if we can understand the complication, the cause and treatment options, we can hopefully reduce the impact it can have on the patient.

When planning an anaesthetic, for every patient we should evaluate risk by generating a list of anaesthesia considerations and where possible the solutions to reduce their impact on the patient in the peri-anaesthesia period. The less solutions we can implement, the higher the risk of anaesthesia.

Some complications arise purely from anaesthesia, and should feature on every patients problem list. Anticipating these complications beforehand, should increase your chances of a successful recovery. This is what we are going to cover within this lecture.

Hypotension

Hypotension is the most seen peri-anaesthesia complication observed in veterinary patients. A consensus was made by A & ECVAA boarded anaesthetists in 2015, that the definition of hypotension is a systolic arterial blood pressure (SAP) of <87mmHg and a mean arterial blood pressure (MAP) of <62mmHg. Anaesthetic drugs, like the volatile and induction agents are the main cause of anaesthetic

related drug induced hypotension; these factored with evidence of pre-existing disease can increase the likelihood of a patient becoming hypotensive under anaesthesia. The first step to avoid hypotension is by monitoring blood pressure (BP) and identifying hypotension. We then determine the cause by troubleshooting the BP algorithm. Implementing the right treatment from the early outset will help reduce the likelihood of acute and long-term complications.

Hypothermia

A study from 2012, concluded that more than 80% of dogs and 97% of cats recovered from anaesthesia hypothermic. Anaesthesia, as well as pre-existing systemic disease will reduce the body's ability to thermoregulate and hypothermia will occur unless measures are taken to preserve and support the patient's temperature. There are many reasons why a patient will lose heat under anaesthesia, from the gases and drugs we use to clipping and prepping for surgery. Having open body cavities during surgery which could be in a cold room. Simple things to consider, such as, laying a patient on a cold table or in its kennel during recovery. During your planning phase, identify ways to minimise heat loss and help reduce the consequences of hypothermia which are dramatically underestimated.

Abnormal Heart rate and rhythm

We are likely to see changes in our patient's heart rate under anaesthesia. Patients with pre-existing clinical disease are more predisposed to cardiac arrhythmias and we also induce heart rate and some arrhythmia with certain drug choices that we make during anaesthesia. Critical patients and those with a high anaesthesia risk might also present with or develop ventricular arrhythmias. Evaluating the impact of the arrhythmia on cardiac output will determine if the rhythm can be tolerated or should be treated.

Hypoventilation

Brain and CNS depression caused by general anaesthesia results in a reduced ventilatory drive. This in combination with the drugs we use during anaesthesia for sedation, muscle relaxation or pain management, will exacerbate respiratory depression and increase the likelihood of us seeing hypercapnia and hypoventilation. Reduced lung volumes from a lower tidal volume, a reduction in functional residual capacity and patient positioning can lead to atelectasis, resulting in a reduced surface area for gas exchange. This in time can lead to hypoxaemia. Interestingly, increased inspired oxygen concentrations used routinely used during anaesthesia can speed up this process. Monitor for a reduction in respiratory rate, hypercapnia, and a reduced tidal volume, and initiate careful IPPV to normalise respiratory parameters.

Recovery

CEPSAF highlighted that most patients die in the first 3 hours of recovery from anaesthesia and concluded that 'greater patient care in the post-operative period could reduce fatalities.' The likelihood for this being the period with the highest risk is that the odds increase when a patient returns to the ward because one-on-one continuous monitoring that is implemented when a patient is under anaesthesia reduces to a more sustainable level within the recovery area, therefore changes in cardio-respiratory parameters are slower to be recognised and treated. Increasing observations and monitoring

during this period can reduce mortality rates but we will look at the newer research on mortality rates to compare with the CEPSAF data.

References:

Ruffato M, Novello L, Clark L. What is the definition of intraoperative hypotension in dogs? Results from a survey of diplomates of the ACVAA and ECVAA. Veterinary anaesthesia and analgesia. 2015 Jan 1;42(1):55-64.

Redondo JI, Suesta P, Gil L, Soler G, Serra I, Soler C. Retrospective study of the prevalence of postanaesthetic hypothermia in cats. Veterinary Record. 2012 Feb;170(8):206-.

Brodbelt DC, Blissitt KJ, Hammond RA, Neath PJ, Young LE, Pfeiffer DU, Wood JL. The risk of death: the confidential enquiry into perioperative small animal fatalities. Veterinary anaesthesia and analgesia. 2008 Sep 1;35(5):365-73.

GDV PATIENT CARE: PREPARATION TO RECOVERY WITH A TWIST!

Natasha Summerfield [1]

[1] Zurich University, Zurich University, Zurich, Switzerland

Learning objectives:

- Describe the term GDV and associated pathophysiology

- List the commonly utilized stabilization techniques

- Describe how to create a safe anaesthetic plan and troubleshoot anaesthetic related complications

- Discuss the post operative nursing considerations for the critical patient

Proceeding:

Gastric dilation and volvulus (GDV) or 'gastric torsion' occurs when the stomach becomes distended with gas and rotates. Gastric distension or 'bloat' can occur without gastric rotation. A GDV is a life-threatening surgical emergency and carries a high risk of mortality. While GDVs can occur in any species, dogs are most commonly affected. The underlying cause for GDVs is not commonly understood but is thought to be multifactorial and linked to a number of risk factors. It is thought that the stomach becomes distended and then twists on it's axis leading to impaired gastric blood flow, and gastric outflow which prevents passage of ingesta into the intestines. The stomach often becomes progressively distended with gas and fluid and can lead to impaired ventilation and the patient ultimately developing shock. Venous flow is reduced due to compression of the vena cava which in turn leads to reduced cardiac output.

Initial interventions

The aim should be to normalize cardiovascular parameters with IV fluids, however, this is unlikely to happen until the stomach has been decompressed. GDV patients are often very painful so analgesia in the form of pure opioids (such as methadone) should be given to aid in comfort and offer some mild sedation for gastric decompression and imaging.

A right lateral radiograph is the choice of imaging to confirm a GD or GDV. If a GDV is present, the pylorus moves cranial and dorsal to the gastric fundus and is separated by a soft tissue opacity. It is thought to look like a reverse C, popeye sign or double bubble. Broad spectrum antibiotics can be administered at this stage due to the risk of endotoxemia and sepsis developing.

The patient's shock cannot be resolved until the stomach is decompressed, however decompression should only be attempted after stabilisation has begun. The preferred choice of decompression is to pass a gastric stomach tube. If passing a stomach tube is unsuccessful then gastric trocharisation can be performed.

Surgery

GDV patients can be very challenging to anaesthetise due to a number of complications. Despite aggressive stabilisation methods, they often still have cardiac arrhythmias, hypotension, hypoventilation, acid base and electrolyte abnormalities. One major concern is the risk of regurgitation, so it is important that the patient is anaesthetised, and the airways protected quickly. Suction should be readily available should the patient regurgitate.

Methadone is often the premedication drug of choice, or alternatively fentanyl and diazepam/midazolam can be used as part of a co-induction. Propofol or alfaxalone titrated to effect for induction, and the patient maintained on Isoflourane or Sevoflourane. All basic parameters should be continuously monitored throughout the anaesthetic including, HR and rhythm, RR, T, BP SPO_2 and $etCO_2$.

The veterinary surgeon will make a large mid-line incision so that the abdomen can be explored. The stomach is fully decompressed by passing a stomach tube and lavaging the stomach contents. Once this has been achieved, the stomach is de-rotated back to its normal position. The stomach and spleen are then assessed for viability and any ischaemic tissue is removed. The final step of the surgical procedure is to pexy the pyloric antral region to the right abdominal wall to prevent the GDV happening in the future. Often the surgery will end with abdominal lavage in case there has been any leakage after trocharisation or resection of ischemic areas.

Nursing care

Ideally, the patient should be recovered with oxygen until they are extubated. Active warming should continue until normothermia is achieved. Monitoring should continue including HR and rhythm, RR, temperature and BP. Frameworks such as Kirby's rule of 20 can be a helpful reminder of the important parameters that we could be monitoring and treating in the critical patient. In particular, fluid balance, acid base, monitoring of major body systems (cardiovascular, respiratory and neurological), analgesia, nutrition, appropriate antibiosis and infection control, recumbent patient care and management of indwelling devices are key aspects of the patient's nursing care plan.

Outcomes

The prognosis depends on how quickly the owners detect a problem, present the patient to the clinic and how fast the veterinary team act. Survival rates can be as high as 80 % with the correct management, and patients can make a full recovery. The chance of survival obviously decreases when any of the common complications are encountered, with the most common including disseminated intravascular coagulopathy, SIRS/SEPSIS, wound breakdown, aspiration pneumonia, pain, ileus and bloat.

BEYOND THE BLOOD – NURSING CONSIDERATIONS FOR THE HAEMOABDOMEN

Natasha Summerfield [1] Eleanor Haskey [2]

[1] University Zurich, ICU Animal hospital Zurich, Zurich, Switzerland
[2] Royal Veterinary College, Anaesthesia and Analgesia/ECC, London, United Kingdom

Learning objectives:

- Recognize early clinical signs and symptoms associated with haemoabdomens in cats and dogs.

- Learn effective nursing interventions and strategies for haemoabdomen management.

- Understand the importance of continuous patient monitoring and provide comprehensive nursing care during and after treatment

Proceeding:

Haemoabdomen in cats and dogs refers to the presence of blood within the abdominal cavity, often resulting from trauma, tumours, or bleeding disorders. Early recognition, diagnostic accuracy, and skilled nursing care are pivotal in managing these cases, ensuring the best possible outcomes for our patients.

Initial interventions

For patients with a suspected haemoabdomen, prompt identification and stabilisation is essential. The first step involves a rapid yet thorough assessment, consisting of vital sign monitoring and obtaining a detailed patient history if available. Intravenous catheter placement allows for fluid resuscitation to address shock symptoms. Low-volume/hypotensive fluid therapy can be considered in such cases. Blood pressure should be re-evaluated repeatedly, and permissive hypotension or blood pressure that is just inside the normal range may be acceptable at this stage, as to not disturb any clots which may have been formed.

Analgesia in the form of pure mu opioids is often administered to these patients to manage pain. Patients with haemoabdomen can experience pain due to direct trauma, abdominal distension and peritoneal irritation, among other causes. Adequate pain management is crucial for their comfort and recovery.

Diagnostic tests, such as abdominal ultrasound or radiography, will be performed to identify the source of bleeding. Concurrently, blood samples are collected for a complete blood count (CBC), total solids/protein (TP), a small chemistry panel and coagulation profile along with blood gas analysis when available. This will help determine the level of bleeding, the patient's coagulation state, and any potential co-morbidities.

The patient may be medically or surgically managed. Effective initial treatment involves a coordinated effort of the veterinary team to swiftly address the life-threatening condition and prepare the patient for further diagnostic, therapeutic or surgical interventions.

Surgical management

During surgical management of haemoabdomen patients, an exploratory laparotomy is performed to identify and control the bleeding source, achieve haemostasis, evacuate abdominal blood, assess for additional injuries, and close the incision.

Post-op care starts with the recovery period. Oxygen should be administered during this time until patients are extubated and depending on needs, for a period of time thereafter. This can be administered via flow-by or nasal prongs may be more efficient in larger dogs. Patient temperature must be monitored closely during this time until normothermia has been achieved. The patient frequently remains on electrocardiogram monitoring for a duration of 24 hours to monitor for the occurrence of ventricular premature contractions (VPCs) and, more significantly, the presence of ventricular tachycardia (V-Tach) Blood pressure is often monitored closely for the first 24 hours. The nurse will assess pain, monitor mentation, and wound appearance and note any exudate that may appear from the wound. Infection control is important post-operatively. Patients should be housed in clean kennels and bedding changed regularly, especially if soiled, these patients may remain mostly recumbent for the first 24 hours; therefore recumbent patient care must be part of the patient's nursing care plan.

Medical management

Frequent vital sign monitoring is a key component of medical management. If the patient's condition can be stabilised with fluid or blood component therapy, it may be decided to closely monitor them rather than undergo surgery. Regular monitoring of PCV/TS (TP) is often conducted in both the blood and the abdominal effusion, to check for rising PCV in the abdomen in comparison to the PCV circulating in the blood. Typically, abdominocentesis is carried out under ultrasound guidance, and the volume of fluid present is evaluated concurrently.

Advanced nursing monitoring skills here are essential to detect any changes to the patient’s demeanour early. If there are any concerns, the nurse should not hesitate to perform extra cardiovascular and perfusion parameter monitoring and report any unusual findings to the clinician.

In summary, the management of haemoabdomen in veterinary patients involves a thorough initial assessment and stabilization, with an emphasis on shock reduction and pain management. The decision between surgical intervention and medical management depends on various factors, including the underlying cause of haemoabdomen, the patient's overall health, and the owner's preference, especially considering the quality of life and survival outcomes. The type of underlying pathology significantly influences prognosis and should be a key consideration in deciding on the management strategy.

Outcomes

The outcome will vary depending on how promptly the haemoabdomen is recognized (the owner's ability to recognize signs of distress/shock/pain in their animals and how quickly they present them to the hospital, how quickly the veterinary team react and stabilize the patient). The prognosis will depend

on the cause of the haemoabdomen for example neoplastic origin vs trauma and further complications that may occur, including but not limited to; hospital-acquired infections, transfusion reactions, SIRS/SEPSIS and coagulopathies. A recent study has shown no major difference in outcome between patients that have undergone surgery vs medical management when presenting with spontaneous haemoabdomen.

References:

Wright, L.V. et al. 'Outcomes and Blood Product Use in 89 Surgically Managed and 79 Medically Managed Cases of Acute Spontaneous Hemoperitoneum in the Dog', Frontiers in veterinary science, 2021, 8, p. 736329.

Main Stream, Friday 31 May 2024

TACHY OR TACKY? MAKING FLUID CHOICES

Rita Hanel [1] Anusha Balakrishnan [2]

[1] Veterinary Emergency Group, Veterinary Emergency Group, Raleigh, United States
[2] Veterinary Emergency Group, Raleigh, United States

Learning objectives:

- List clinical findings indicative of dehydration

- List clinical findings consistent with the four main categories of perfusion-based shock

- List and discuss the application of point of care diagnostics in the assessment of volume status

- Outline a fluid plan to address dehydration and hypovolemia

- List and discuss the application of point of care diagnostics in the monitoring of fluid resuscitation

Proceeding:

Hydration versus perfusion; 2 sides of the same coin

As you know fluid therapy occurs in several phases that are dynamic, can be cyclic, and require monitoring. Just like any drug, fluids have huge benefits and are lifesaving, but can also be deleterious. Below are some quick guidelines and equations for reference. We'll dive further into the application of these concepts and the application of various measures of intravascular volume assessment during the lectures. A QR code with more in depth information and a copy of the slides will be provided.

Keep in Mind the 5 R's of Fluid Therapy

Resuscitation

Intravenous fluid therapy is required to replace any intravascular volume deficit, also referred to hypovolemia. Typically, intravascular volume deficits occur secondary to significant dehydration (> 9%), hemorrhage, or vascular leak.

To be most effective, choose a fluid type that mimics the loss. For example, blood products will most effectively replace blood loss.

Redistribution

All patients redistribute fluid between the intra- and extracellular spaces (interstitium and intravenous), and this distribution is markedly impacted by inflammation, trauma, and other factors, such as the health of the glycocalyx.

Reassessment

Once fluids are given, a patient must be frequently assessed, and redistribution should be anticipated and trended. Once resuscitation and redistribution are complete, any remaining fluid deficits can be added to ongoing replacement fluids.

Routine Maintenance

Assumes the patient is euvolemic and does not have ongoing losses in excess of insensible losses (from breathing, perspiring, formed feces, etc..). This is mainly a water requirement with some sodium and potassium requirements.

DOG maintenance fluid dose per 24 hours is 132 x BWt (kg)^{75} 2-6 ml/kg/hr can be used until full calculation is performed

CAT maintenance fluid dose per 24 hours is 80 X BWt (kg)^{75}2-3 ml/kg/hr can be used until full calculation is performed

Replacement

Intravenous fluids prescribed to replace any remaining dehydration deficit as well as ongoing fluid losses, such as diarrhea, gastric aspirations, surgical drains, excessive urine output, etc.…

Losses due to dehydration are estimated using the equation below. Crystalloid fluids administered during initial resuscitation can be subtracted from this amount.

Body weight (lean, in kg) x % dehydration = volume (L) to correct

After resuscitation (see above), the total daily fluid prescription for any patient includes:

The patient's maintenance fluid requirement (Routine Maintenance)

Any deficits due to previous losses (Any deficits remaining after Redistribution of your Resuscitation efforts)

Adjustments for any ongoing losses (Replacement)

For example, Charlie, a 5 kg dog presents for acute hemorrhagic diarrhea. Vital parameters and initial diagnostics are consistent with hypovolemic shock secondary to dehydration. You are waiting on bloodwork, so you resuscitate Charlie with LRS and make an initial plan. You gave one 20 ml/kg bolus of LRS and two subsequent 10mlg/kg boluses of LRS to stabilize him. What is your initial fluid prescription with this in mind?

Dehydration causing hypovolemia is typically > 9%, so the initial deficit is at least 500mls (10% x 5 kg=0.5L)

You gave a total of 200 mls of LRS, so 300 mls remains of the aforementioned deficit.

Routine maintenance for Charlie is 440 mls per day or 18 ml/hour. (132 x 5 ^{75}=441)

There is no heart murmur, so I am choosing to replace the remaining deficit (330 mls) over 12 hours (25 ml/hr). If the bloodwork reveals significant azotemia, I may decrease this to 6 hours.

In summary, my total rate of LRS for the next 12 hours is 43 ml/hr, (18 for maintenance and 25 to finish replacement), after which I will drop to maintenance (18 ml/hr). I will watch for significant ongoing loss, as I may need to account for this, and reassess every 4- 6 hours for this shift.

Methods of monitoring total body water and hydration include signs of extravascular (non-IV) depletion, including:

Daily physical exam, looking for signs of dehydration (turgor, sunken eyes, TACKY mucous membranes)

Body Weight (60% of body weight is water)

Urine specific gravity

If able to concentrate, urine should not be highly concentrated = dehydration

If able to concentrate and urine is iso- or hyposthenuric, consider fluid administration may be in excess of need, especially if anticipated weight gain has taken place.

PCV/TS - daily trends in both

POCUS to assess intravascular volume and signs of overhydration (3rd spacing, B lines on POCUS)

COMPLICATIONS OF INTRAVENOUS FLUID THERAPY: PREVENTION, MONITORING AND INTERVENTION

Andrew Linklater [1]

[1] Veterinary Specialists of the Rockies, Castle Rock, CO, United States

Learning objectives:

- Review of standard intravenous catheter placement techniques

- Learn how to identify the most common complications associated with intravenous catheters

- Learn how to minimize complications and how to treat them when they occur

Proceeding:

Fluids therapy is vitally important to veterinary practice, however the delivery of intravenous fluids and medications come with many complications, which are divided into several categories: method of delivery, volume or type of drug delivery and miscellaneous. Recent veterinary literature has reviewed the complication **frequency** in both cats (21.4%) and dogs (13-24.2%, depending on ward), however these studies included only a small number of patients and at a single institution. Complications included phlebitis, extravasation, patient removal, occlusion and edema formation.[1,2] Two recent meta-analysis in humans demonstrated a frequency of phlebitis in peripheral IV lines nearing 31% with risk factors of longer dwell time, antibiotic infusion, female gender, forearm insertion, infectious disease, Teflon catheter;[3,4] the second study reported a phlebitis rate of 23.8%, extravasation (13.7%, occlusion 8%, leakage 7.3%, pain 6.4% and dislodgement 6%.[5,6]

Method of Delivery. A catheter is essential for IV fluid administration; it must penetrate through skin (ensuring it is not sterile) and is a foreign object, which means the most common complications simply cannot be avoided, even with the best technique. Before placing a catheter, hands should always be washed, and the site should be clipped of hair, be prepared in an aseptic fashion and for long-term lines, the area should also be draped off, and the individual should don sterile gloves, cap and mask. The IV site should be adequately covered with some sort of liquid-resistant tape, bandage or dressing, and changed immediately if it becomes wet or soiled. Placement of a triple antibiotic does not necessarily decrease the risk of IV site infection; if these products are used, they should be obtained from a sterile/dedicated source. The catheter site and all connections must be adequately protected and the patient regularly checked for any evidence of swelling, discharge, pain, redness, tenderness, warmth, discomfort with infusion or a new fever. If an unexplained fever develops in a patient, consider replacing IV catheters. Ideal duration of catheter placement remains controversial. Some institutions regularly change catheters. However, given the limited number of peripheral vein sites available in veterinary patients, regular replacement when adverse effects are not apparent may not be necessary. If one suspects that

sepsis is associated with a catheter, blood cultures (not catheter tip) should be performed, the patient should be started on antibiotics, and the catheter removed. Catheter associated thrombosis results in a firm vein and edema. Certain disease conditions warrant diligent monitoring or even thromboprophylaxis. Medical dissolution of the thrombus is rarely recommended. Catheter emboli (pieces of the catheter itself) are a rare occurrence, typically associated with poor placement or removal techniques.

Fluid complications. Excessive movement, catheter migration, inadvertent subcutaneous placement and excessive injection pressures may result in extra-vasation; when this occurs with large volumes or certain medications (e.g. vincristine, and hypertonic solutions), pain and local tissue necrosis may occur. Fluid intolerance (overload) may occur with several diseases (e.g., cardiac disease renal failure, systemic inflammation) or excessive volume administration. Patients at risk should have their heart rate, respiratory rate and blood pressure closely monitored; serous ocular or nasal discharge chemosis, pulmonary crackles and other signs occur later. Patients with a diuresis (forced or pathophysiological) should have a gradual discontinuation of fluids to avoid dehydration as the medullary concentration gradient may not be sufficient. Changes in tonicity most often results from inappropriate fluid choices. A rapid change in serum Na may result in serious neurologic consequences; serum Na levels should not change more than 0.5 mEq/L/hr, or 8-12 mEq/L/day. Hypokalemia is the most common electrolyte change; potassium supplementation should not exceed 0.5 mEq/kg/hr; however life-threatening hypokalemia may be treated more aggressively. Acidosis and alkalosis may occur as well, particularly when associated with changes in chloride; hyperchloremic patients, where Cl is preferentially resorbed in the kidney (and HCO3 is then excreted), may make acidosis persist. The reverse is true with hypochloremia.

Miscellaneous. Complications can occur with colloid administration; hydroxyethylstarches (HES) has been reported to be associated with coagulopathy at high doses (Vetstarch^{TM} > 50 mL/kg) and acute kidney injury, although this remains controversial. Aggressive crystalloid resuscitation can exacerbate non-compressible hemorrhage, likely from a combination of clotting factor dilution or clot dislodgement. A mean arterial pressure (MAP) of 65 mmHg have improved outcome in patients with non-compressible hemorrhage vs a MAP of 80 mmHg which is why “hypotensive” resuscitation is indicated in hemorrhage.

Summary. Fluid therapy remains a mainstay of many diseases, but patients should be monitored for complications associated with both the type and amount of fluid administered as well as complications associated with the method of delivery (IV catheter). Patients should be regularly assessed for the complications and the fluid prescription regularly re-assessed. Crystalloid administration should be judicious and follow well established guidelines (maintenance of 40-60 mL/kg/day plus dehydration % and ongoing losses).

References:

Bush K, Odunayo A, Hedges K, et al. Peripheral Intravenous catheter complications in hospitalized cats: an observational study. Top Companion Anim Med. 2020 Nov:41:100456. Doi: 10.1016/j.tcam.2020.100456

Simpson SE, Zersen KM. Incidence and type of peripheral intravenous catheter complication documented in hospitalized dogs. J Small Anim Pract. 2023 Mar;64(3):130-135. Doi:10.1111/jsap.13574.

Hagley MJ, Hopper K, Epstein SE. Characteristics of arterial catheter use and related complications in dogs and cats in an intensive care unit. J Vet Emerg Crit Care. 2021 Jul;31(4):469-475. Doi: 10.1111/vec.13084.

Mooshian S, Deitschel S, Haggerty JM, Guenther CL. Incidence of arterial catheter complications: a retrospective study of 35 cats (2010-2014). J Fel Med Surg. 2019 Feb;21(3):173-177. Doi: 10.1177/1098612X18767570

Lv L, Zhang J. The incidence and risk of infusion phlebitis with peripheral intravenous catheters: A meta-analysis. J Vac Access. 2020 May;21(3):342-249. Doi: 10.1177/1129729819877323.

Marsh N, Webster J, Ullman AJ, et al. Peripheral intravenous catheter non-infectious complication in adults: A systematic review and meta-analysis. J Adv Nurs. 2020 Dec;76(12):3346-3362. Doi: 10.1111/jan.14565.

Marsh N, Webster J, Mihala G, Rickard CM. Devices and dressings to secure peripheral venous catheter to prevent complications. Cochrane Database Syst Rev. 2015 Jun 12;2016(6):CD011070. Doi:10.1002/14651858.CD11070.pub2.

DAMAGE CONTROL! MANAGING THE ACUTELY HEMORRHAGING TRAUMA PATIENT

Andrew Linklater [1]

[1] Veterinary Specialists of the Rockies, Castle Rock, CO, United States

Learning objectives:

- Know how to rapidly assess a patient for life-threatening hemorrhage

- Learn the concepts of damage control resuscitation

- Learn the concepts of damage control surgery

- Know of alternative non-surgical techniques for patients with cavitary hemorrhage

Proceeding:

Recognition of a patient with hemorrhagic shock is easily performed within one minute, examining clinical perfusion parameters (heart and respiratory rate, mucous membrane color, capillary refill time, pulse quality and level of consciousness); these occur as a result of baroreceptor input of low intravascular volume and high sympathetic tone. PCV/TS may not be altered acutely and other parameters (blood pressure, urine production, lactate measurement) take longer and should not delay care. Stages of shock is discussed in other resources. Common etiologies of hemorrhagic shock include trauma, ruptured neoplasia and coagulopathies.

Seventy-five percent of human trauma deaths result from exsanguination. Severe blood loss results in the *lethal triad of acidosis, hypothermia and hypotension*, which contributes to mortality. The remaining 25% die from delayed complications including sepsis and multi-organ failure. Subsequently our goal should be to rapidly arrest hemorrhage, which has resulted in the concept of Damage Control. The term Damage Control in medicine was adopted by human surgeons in the 1970s who described of patients with significant hepatic hemorrhage which had improved survival with rapid packing. Over the past 50 years, two arms of Damage Control have evolved and when used together have improved overall outcome.

Damage Control Resuscitation (DCR) involves several aspects. The first is permissive hypotension (PH). PH uses conservative resuscitation strategies (endpoint with MAP >60 or SBP >70, rather traditional endpoints than MAP >80-90 or SBP >100), until the hemorrhage is controlled (in non TBI patients) to minimize blood loss from non-compressible hemorrhage. Although several studies have demonstrated an equivalent to improved outcome, the use PH is not a foregone conclusion; other demonstrated

benefits include less blood loss and fewer transfusions. The second aspect is early use of blood (rather than crystalloids and synthetic colloids). This is an easier technique to employ in human medicine compared to veterinary, where cost and resources must be considered. However, there is solid evidence that resuscitation with blood is superior in both the experimental and clinical (human) setting. The third aspect is to minimize crystalloids during resuscitation; several studies have clearly demonstrated superior outcomes in patients receiving blood compared to crystalloid use. Crystalloids are known to result in dilution of coagulation factors, hypothermia, acidemia, acute lung injury, organ edema and dysfunction and more. There is still much debate on whether component therapy (combination of plasma, platelets and packed red blood cells) is inferior or not to whole blood therapy. The final aspect of DCR is correction of developing complications, particularly the lethal triad: patients should be kept warm by any means possible (although there is still controversy with TBI), correction of acidosis when appropriate (primarily achieved with volume resuscitation, however bicarbonate may be administered) and correction of developing coagulopathies. A recent analysis demonstrated an improvement in mortality from 45% to 27% along with a decrease in units of blood used from 12 units to 4 when DCR is employed in human medicine.

Damage Control Surgery (DCS) should always be used in conjunction with DCR. The goal of DCS is to perform an abbreviated surgery to 1) arrest hemorrhage and 2) minimize contamination (GI, bile, urine); while limiting the negative physiologic impact (lethal triad) of a prolonged surgical/anesthetic intervention and not performing definitive surgical care initially. The team should be well prepared for a challenging anesthesia, the use of multiple hemostatic (stapling, vessel sealing) and contamination-limiting (stapling) techniques, transfusions and more. DCS is only necessary in a small percentage (3-8%) of human trauma victims. It is not always clear who requires DCS in human medicine, although guidelines based on specific physiological parameters have been developed (example, pH of <7.2 or BE of >15 with a temperature <35C); those parameters have not been developed in veterinary medicine. The author is more aggressive about surgery when patients are difficult to resuscitate or patients are resuscitated and shock later returns. After the initial DCS, the patient is then transferred to ICU for stabilization and ongoing DCR. Definitive surgery is performed later (6-72 h later), when the patient is more stable.

The cost of DCS (performing a second definitive surgery) along with the speed and skill of the attending surgeon are very big challenges to be faced in veterinary medicine. When DCS is not possible due to these limitations, techniques such as use of autologous blood transfusion and/or abdominal/hindlimb counter pressure may be useful in patients with hemorrhage (but not with GI or other contamination). These techniques are described in the author's textbook.

References:

Linklater A, Kirby R. Monitoring and Intervention for the Critically Ill Small Animal: The Rule of 20. Wiley Blackwell. 2017

Tran A, Yates J, Lau A. Permissive Hypotension versus conventional resuscitation strategies in adult trauma patients with hemorrhagic shock: a systematic review and meta-analysis of randomized controlled trials. J Trauma Acute Care Surg 2018;84:802-8.

Cole E, Weaver A,. Gall, et al. A decade of DCR: New transfusion practices, new survivors, new directions. Ann Surg 2019. 4. Ntourakis D, Liasis L. Damage control resuscitation in patients with major trauma: prospects and challenges. J Emerg Crit Care Med 2020;4:34.

FELINE ARTERIAL THROMBOEMBOLISM

Julien Guillaumin [1]

[1] Colorado State University, Clinical Sciences, Fort Collins, United States

Learning objectives:

- Understand diagnosis and diagnostic tests for FATE

- Recall the prognosis for bilateral and unilateral FATE

- Understand the treatment options for FATE

Proceeding:

Introduction: Feline aortic thrombo-embolism (FATE) is the most common clinical cause of thromboembolism in veterinary medicine. It is initiated by the sudden migration of a left atrial thrombus into the systemic arteries. It is a devastating syndrome with short term consequences, characterized by acute pain, paralysis, and rhabdomyolysis in the affected limb(s). The major cause (90%) for feline ATE is cardiomyopathy. However, and despite a median age at FATE of 8-12 years old, only 20% of FATE cats have a known cardiomyopathy at the FATE diagnosis. It has been shown that around 25% of hypertrophic cardiomyopathy (HCM) cats will develop FATE.

Diagnosis: Approximately 70-75% of FATE cats are affected in both pelvic limbs. The clinical diagnosis of FATE is easy, and clinical, using the “5Ps rule”: *pallor*, *polar*, *pulselessness*, *paralysis/paresis* and *pain*. Other diagnostic tools include absence of Doppler flow, direct visualization of the thrombus on ultrasound or infrared thermal imaging. It is important to assess signs of cardiac disease and congestive heart failure (CHF), as CHF is present in 50-70% of FATE cats, although not associated with a worse prognosis. Cardiac auscultation abnormalities occur in 2/3 of the cases. Thoroughly assessing the cats for comorbidities is important. Cardiac point-of-care ultrasound (POCUS) can confirm the presence of a cardiogenic origin of FATE. Thoracic POCUS can be performed for rapid assessment of cardiac and thoracic structures. Chest radiographs can be performed upon stabilization to assess presence of CHF, as well as investigate the presence of pulmonary neoplasia, the 2nd most common cause of FATE. Baseline bloodwork is important for baseline renal value.

Prognosis: FATE have been plagued for decades by high euthanasia rates. However, a recent prospective study on cats with bilateral pelvic limb paralysis showed an overall 37.5% discharge rate, and with some cats experiencing >1 year survival. This confirms data from retrospective studies, showing a survival of 27-45% with bilateral FATE. Lower rectal temperature at admission, higher affected limb lactate and longer time from event to treatment are all negative prognostic indicators. A treatment effect has been

challenging to identify: not receiving treatment with aspirin, clopidogrel or both, has been shown to be associated with a poor prognosis. A relatively large retrospective study was able to show that FATE cats receiving early thrombolysis with tissue plasminogen factor (TPA) had an increased chance of recanalization and functional recovery, although that was not translated into a survival benefit. In retrospective studies, survival time after discharge could be up to 350-500 days, with the use of rivaroxaban and clopidogrel having the highest survival time and lowest re-embolization rate.

Treatment: Emergency treatment of FATE cats revolves around analgesia, treatment of the primary disease, nursing care, thromboprophylaxis, and may include thrombolysis and promotion of collateral circulation. Pain control is important for FATE patients, analgesia with a pure μ-agonist is recommended (e.g. methadone, fentanyl). Treatment of cardiomyopathy and CHF should be instituted if appropriate.[6] Anticoagulant is recommended to decrease worsening of the thrombus. The current recommendations in FATE cats are not evidence-based recommendations, but suggest the administration of clopidogrel in combination with LMWH can be considered in cats at risk of FATE. Based on results from recent studies, it is possible that a combination treatment with clopidogrel and rivaroxaban/apixaban may be the preferred long-term treatment option.[5] Thrombolytic agents can be considered for treatment of acute (< 6 hrs) FATE following an assessment of the risk and benefit in individual cats, according to a recent expert consensus.[8] Nursing care is extremely important: physical therapy with passive range of motion and leg warming can be attempted if tolerated by the patient.

If treated, FATE cats usually improve with 24-48 hours regarding ambulation status and/or presence of pulses. If an improvement is not noted within 24-48 hours, long-term prognosis and long-term options such as devices for ambulation assistance or amputation should be discussed.

Complications: Complications are common, especially acute kidney injury (AKI) and reperfusion injuries, regardless of thrombolysis being attempted or not. Regarding AKI, bilateral renal thrombosis is uncommon, so local vasoconstriction of renal arteries should be investigated as a potential cause for AKI. Therefore, drugs promoting the development of collateral circulation may be beneficial. Such candidate drugs include, pentoxifylline or cyproheptadine.

References:

Smith, S. A., Tobias, A. H., Jacob, K. A., Fine, D. M. & Grumbles, P. L. Arterial thromboembolism in cats: acute crisis in 127 cases (1992-2001) and long-term management with low-dose aspirin in 24 cases. J. Vet. Intern. Med. 17, 73–83 (2003).

Guillaumin, J. et al. Bilateral lysis of aortic saddle thrombus with early tissue plasminogen activator (BLASTT): a prospective, randomized, placebo-controlled study in feline acute aortic thromboembolism. J Feline Med Surg 1098612X221135105 (2022) doi:10.1177/1098612X221135105.

Borgeat, K., Wright, J., Garrod, O., Payne, J. R. & Fuentes, V. L. Arterial thromboembolism in 250 cats in general practice: 2004-2012. J. Vet. Intern. Med. 28, 102–108 (2014).

Cambournac, M., Damoiseaux, C., Guillaumin, J., Goy-Thollot, I. & Pouzot-Nevoret, C. Multicentric aortic thromboembolism retrospective study in 158 cats: the MATERS study. in (Ghent, Belgium, 2022).

Lo, S. T., Walker, A. L., Georges, C. J., Li, R. H. & Stern, J. A. Dual therapy with clopidogrel and rivaroxaban in cats with thromboembolic disease. J Feline Med Surg 24, 277–283 (2022).

Luis Fuentes, V. et al. ACVIM consensus statement guidelines for the classification, diagnosis, and management of cardiomyopathies in cats. J Vet Intern Med 34, 1062–1077 (2020).

Sharp, C. R. et al. 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. J Vet Emerg Crit Care (San Antonio) 29, 121–131 (2019).

Sharp, C. R. et al. 2022 Update of the Consensus on the Rational Use of Antithrombotics and Thrombolytics in Veterinary Critical Care (CURATIVE) Domain 6: Defining rational use of thrombolytics. J Vet Emerg Crit Care (San Antonio) 32, 446–470 (2022).

THE FELINE TRAUMA PATIENT

Ivayla Danielova Yozova [1]

[1] School of Veterinary Science - Tāwharau Ora, Massey University, Palmerston North, New Zealand

Learning objectives:

- Current epidemiological information

- Dos and Don'ts on triage and initial stabilization

- Principles of pain management

- Principles of IVF and transfusion requirements

- The value of cat-friendliness

Proceeding:

Trauma is a common cause for presentation of cats to the ER. Trauma is challenging to treat in feline patients and reported mortality rates remain relatively high (8 – 20%) depending on the setting. Types of trauma depend mainly on demographic and spatial factors. In urban areas with high buildings high rise syndrome is seen with thoracic, but also facial and appendicular skeletal injuries involved. Road traffic accidents remain a predominant cause for trauma in outdoor cats, with appendicular skeletal, head and neck injuries as well as thoracic injuries present. Facial trauma associated or not with traumatic brain injury (TBI) is a frequent feature in feline trauma patients. Penetrating trauma from dog bites and lacerations from fences can also be seen. Sometimes, especially for outdoor cats, the cause and time of trauma remain unknown. Young age is reported as a risk factor for trauma in several studies. Other demographic and spatial factors, such as high-density population, fur colour, and sex, yield conflicting results in different studies.

Triage and initial stabilization are essential for the successful management of trauma cats. A triage examination requires a hand-on examination rather than merely observing the cat in the carrier. Admission hypothermia and decreased level of consciousness have been associated with worse prognosis in cats with trauma.

A primary survey focusing on the cardiovascular, respiratory, and neurological system and stabilization of these systems, should be conducted before further diagnostics. Stabilization essentials include supplemental oxygen administration, analgesia (using fast-acting, titratable, and reversable agents), rewarming, low-stress handling and rational IV fluid challenge administration when appropriate. Clinical monitoring is essential and "over-instrumentalization" of trauma cats should be avoided to reduce stress

and discomfort. Minimum database should include PCV/TP, lactate (or venous blood gas) and creatinine, complete point-of-care ultrasound, including a focused cardiac exam to exclude asymptomatic HCM that might exacerbate with interventions. Complete blood count and biochemistry are advised in severe trauma cases and/or older cats. Open wounds and fractures should be covered to prevent further contamination and more extensive management performed after stabilization.

A secondary survey is performed after initial stabilization including orthopaedic and neurological exams coupled with whole-body survey radiographs. Neurologic and orthopaedic exams should be performed based on the level of tolerance of the cat, with focus on suspected injuries and enhanced by imaging. Other emergency procedures such as wound management and external fracture stabilization should be performed only in stable cats. A full-body CT should be considered in the poly-trauma cat, especially if head trauma is present. A full-body CT is likely to have a higher diagnostic yield and be time and radiation-sparing compared to conventional radiographs.

The clinical-examination based severity of illness score - Animal Trauma Triage Score can be used to determine trauma severity, but also as a memory aid to ensure thorough assessment of each body system. Trauma is considered severe in patients with a score of ≥3 to ≥5 and such patients necessitate particular attention. The ATT score additionally allows prognostication and stratification for research. It has been extensively validated in dogs and cats and it performs well in predicting mortality for trauma.

Two recent large scale epidemiological studies from the VetCOT trauma registry demonstrated that only about a third of cats presented for trauma require surgery. Whether the feline patient requires conservative therapy only or is being managed post-operatively general principles for ICU care, such as Kirby's rule of 20, apply. Essentials for successful management of the hospitalized trauma cat are adequate analgesia, early nutrition, rational IV fluid administration to avoid volume overload and low stress handling and cat-friendly environment. Balancing between adequate monitoring and interventions and allowing sufficient hands-off time is challenging, but necessary. Despite that cavitary bleeds are not common in cats (> 90% of 521 cats with severe trauma had a negative abdominal POCUS in a recent study[30]), cats often become anaemic after trauma and may require transfusions. This is presumed to be multifactorial including bleeding around injured limbs or wounds, blunted erythropoietin response and repeated phlebotomies, even though robust clinical data from cats is lacking.

The overall prognosis of cats with trauma is highly depending on the setting, with first opinion settings showing higher mortality rates. A recent study from the VetCOT trauma registry showed that in 9.1% of euthanasia decisions in cats with trauma have a financial constraint component as opposed to 8.1% being due to grave prognosis only. Financial considerations should be factored into offering a "spectrum of care" to owners of feline trauma patients.

References:

McDonald JL, Cleasby IR, Brodbelt DC, Church DB, O'Neill DG. Mortality due to trauma in cats attending veterinary practices in central and south-east England. J Small Anim Pract 2017;58:570-576.

O'Neill DG, Church DB, McGreevy PD, Thomson PC, Brodbelt DC. Longevity and mortality of cats attending primary care veterinary practices in England. J Feline Med Surg 2015;17:125-133.

Fitzgerald WR CN, 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. Journal of Feline Medicine and Surgery 2022.

Lapsley J, Hayes GM, Sumner JP. Performance evaluation and validation of the Animal Trauma Triage score and modified Glasgow Coma Scale in injured cats: A Veterinary Committee on Trauma registry study. J Vet Emerg Crit Care (San Antonio) 2019;29:478-483.

Hernon T, Gurney M, Gibson S. A retrospective study of feline trauma patients admitted to a referral centre. J Small Anim Pract 2018;59:243-247.

Nascimento DC, Costa Neto JM, Solca MS, Estrela-Lima A, Barbosa VF. Clinicoepidemiological profile and risk factors associated with mortality in traumatized cats admitted to a veterinary teaching hospital in Brazil. J Feline Med Surg 2021:1098612X211028027.

Rochlitz I. Study of factors that may predispose domestic cats to road traffic accidents: Part 2. Vet Rec 2003;153:585-588.

Rochlitz I. Study of factors that may predispose domestic cats to road traffic accidents: part 1. Vet Rec 2003;153:549-553.

Hickey MC, Gray R, van Galen G, Ward MP. Distribution of mortality patterns in cats with naturally occurring trauma: A Veterinary Committee on Trauma registry study. Vet J 2021;278:105765.

Zimmermann E, Hittmair KM, Suchodolski JS et al. Serum feline-specific pancreatic lipase immunoreactivity concentrations and abdominal ultrasonographic findings in cats with trauma resulting from high-rise syndrome. Journal of the American Veterinary Medical Association 2013;242:1238-1243.

Whitney W, Mehlhaff C. High-rise syndrome in cats. Journal of the American Veterinary Medical Association 1987;191:1399-1403.

Vnuk D, Pirkić B, Matičić D et al. Feline high-rise syndrome: 119 cases (1998–2001). Journal of feline medicine and surgery 2004;6:305-312.

Bonner SE, Reiter AM, Lewis JR. Orofacial manifestations of high-rise syndrome in cats: a retrospective study of 84 cases. Journal of veterinary dentistry 2012;29:10-18.

Papazoglou L, Galatos A, Patsikas M et al. High-rise syndrome in cats: 207 cases (1988-1998). Univeristy of Thessaly Institutional Repository 2001.

Wilson J, Gruffydd-Jones T, Murray J. Risk factors for road traffic accidents in cats up to age 12 months that were registered between 2010 and 2013 with the UK pet cat cohort (‘Bristol Cats’). Veterinary record 2017;180:195-195.

Rochlitz I. Study of factors that may predispose domestic cats to road traffic accidents: part 1. Veterinary Record 2003;153:549-553.

Tundo I, Southerden P, Perry A, Haydock RM. Location and distribution of craniomaxillofacial fractures in 45 cats presented for the treatment of head trauma. J Feline Med Surg 2019;21:322-328.

Knight R, Meeson RL. Feline head trauma: a CT analysis of skull fractures and their management in 75 cats. Journal of feline medicine and surgery 2018:1098612X18819183.

Cameron S, Weltman JG, Fletcher DJ. The prognostic value of admission point-of-care testing and modified Glasgow Coma Scale score in dogs and cats with traumatic brain injuries (2007-2010): 212 cases. J Vet Emerg Crit Care (San Antonio) 2022;32:75-82.

Pratschke K, Kirby B. High rise syndrome with impalement in three cats. Journal of small animal practice 2002;43:261-264. 21. von Hekkel AF, Halfacree Z. Thoracic dog bite wounds in cats: a retrospective study of 22 cases. BSAVA Library, 2017.

Childs J, Ross L. Urban cats: characteristics and estimation of mortality due to motor vehicles. American journal of veterinary research 1986;47:1643-1648.

Conroy M, O'Neill D, Boag A, Church D, Brodbelt D. Epidemiology of road traffic accidents in cats attending emergency-care practices in the UK. Journal of Small Animal Practice 2019;60:146-152.

Zulauf D, Kaser-Hotz B, Hassig M, Voss K, Montavon PM. Radiographic examination and outcome in consecutive feline trauma patients. Vet Comp Orthop Traumatol 2008;21:36-40.

BAR-AM Y, Pollard RE, Kass PH, Verstraete FJ. The diagnostic yield of conventional radiographs and computed tomography in dogs and cats with maxillofacial trauma. Veterinary Surgery 2008;37:294-299.

Dancer SC, Le Roux C, Fosgate GT, Kirberger RM. Radiography is less sensitive relative to CT for detecting thoracic radiographic changes in dogs affected by blunt trauma secondary to a motor vehicle accident. Veterinary Radiology & Ultrasound 2019;60:648-658.

Lee K, Heng HG, Jeong J, Naughton JF, Rohleder JJ. Feasibility of computed tomography in awake dogs with traumatic pelvic fracture. Veterinary Radiology & Ultrasound 2012;53:412-416.

Rockar RA, Drobatz KS, Shofer FS. Development of a scoring system for the veterinary trauma patient. Journal of Veterinary Emergency and Critical Care 1994;4:77-83.

Ash K, Hayes GM, Goggs R, Sumner JP. Performance evaluation and validation of the animal trauma triage score and modified Glasgow Coma Scale with suggested category adjustment in dogs: A VetCOT registry study. Journal of veterinary emergency and critical care 2018;28:192-200.

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.

Gregory CW, Davros AM, Cockrell DM, Hall KE. Evaluation of outcome associated with feline trauma: A Veterinary Committee on Trauma registry study. J Vet Emerg Crit Care (San Antonio) 2023.

Rudloff E, Kurby R. Feline Circulatory Shock In: Little S, editor. August's Consultations in Feline Internal Medicine Elsevier St Lous, USA 2016:843-858.

Merten N, Weingart C, Kohn B. [Causes, diagnostics and course of disease in 194 cats with anemia]. Berl Munch Tierarztl Wochenschr 2015;128:384-393.

Hobisch-Hagen P, Wiedermann F, Mayr A et al. Blunted erythropoietic response to anemia in multiply traumatized patients. Crit Care Med 2001;29:743-747. 35. Branco BC, Inaba K, Doughty R et al. The increasing burden of phlebotomy in the development of anaemia and need for blood transfusion amongst trauma patients. Injury 2012;43:78-83.

Lynch AM, Respess M, Boll AE et al. Hospital-acquired Anemia in Critically Ill Dogs and Cats: A Multi-Institutional Study. J Vet Intern Med 2016;30:141-146.

WHAT IS NEW FOR THE MANAGEMENT OF FELINE URETHRAL OBSTRUCTION?

Julien Guillaumin [1]

[1] Colorado State University, Clinical Sciences, Fort Collins, United States

Learning objectives:

- Understand how to diagnose feline urethral obstruction
- Recall treatment options for hyperkalemia
- Understand advantages and inconvenients of each hyperkalemia treatment
- Understand that both medical and surgical treatments exist for urethral obtruction
- Analyze recent literature available on feline urethral obstruction

Proceeding:

Introduction: The poster child of feline urethral obstruction (UO) is a young, obese male cat, who lives indoor and eats dry food. Those factors are somewhat intertwined. Many (around 50%) of cats with UO are suffering from idiopathic cystitis and are commonly obstructed by a urethral plug with inflammatory debris and a crystalline matrix. Urolithiasis or urethral spams are not as common (around 20% each). Other causes such as stricture, neoplasia or infection represent less than 5% of combined causes. Clinical signs are lower urinary tract signs such as pollakiuria, stranguria, dysuria and hematuria. Complete lack of urine production and/or systemic signs such as vomiting, lethargy or lateral recumbency should raise concerns for a urethral obstruction in any male cat.

Diagnosis: Feline UO is a triage diagnosis. Visual assessment of the level of consciousness, diagnosis of an acute abdomen with a firm, distended and painful bladder, and cardiovascular assessment are all important steps. The combination of a temperature < 96.6°F and a heart rate < 140 bpm is very predictive (98%) of hyperkalemia. In general, diagnostic tests in a cat with UO should include ECG (check for signs of hyperkalemia such as bradycardia, prolonged P-R interval, diminished/absent P waves, widened qRs complexes, tented T waves and/or a sine wave), blood pressure, blood gas and/or biochemistry profile which includes electrolytes and renal values as well as diagnostic imaging tests. In the author's opinion, a urinalysis and a urine culture are usually not necessary for first offender cats, as less than 10% of cats with UO have a urinary tract infection.

Treatment: Specific treatments for cats with UO are controversial and many options exist. In general, however, treatment can be divided into 4 overall categories ("the 4 pillars"):

Intravenous fluids: A balanced electrolyte solution is recommended for fluid resuscitation, even if the K+ is high. Because of hyperkalemia and dehydration, cats with UO may have a combination of metabolic shock and hypovolemic shock. Therefore, a combination of treating hyperkalemia and severe dehydration is necessary for treating shock. Over-aggressive fluid resuscitation can lead to fluid overload, especially if there is occult cardiac disease. After de-obstruction, cats may have post-obstructive diuresis and may require large amounts of fluids to maintain hydration and compensate fluid losses. Predictors of severe post-obstructive diuresis, such as pH is < 7.2 and/or bicarbonates are < 15 mmol/L, can be found.

Treatment of electrolyte and blood gas disturbances: Hyperkalemia should be treated if there is ECG changes and/or if K+ is above 6-8 mmol/L. Cardio-protection with calcium gluconate allows for acute stabilization, although it does not decrease the serum potassium. Other options include insulin + dextrose, beta-2 agonists, or bicarbonates. Each of the hyperkalemia management strategies have pros and cons. Relieving the UO is important to treat both the hyperkalemia and the metabolic acidosis.

Unblocking the cat: There are multiple ways to both induce sedation and physically remove the UO in cats. Multimodal anesthesia usually allows enough anesthesia and relaxation to unblock the cat but should be adapted to each cat. There are no differences in complication rates or mortality between full anesthesia with intubation and inhalant anesthesia agents compared to sedation without intubation. A coccygeal block is recommended, as it reduces the amount of sedation needed for urinary catheterization.

There are many protocols described for urinary catheterization in blocked cats. The general concept is that hydro-retro-pulsion should be used to relieve the obstruction which allows the placement of an indwelling catheter. The author prefers a regular 22G intravenous catheter (without the stylet!) connected to a T-set and a syringe connected to a mixture of saline solution and sterile lubricant. In general, the catheter is usually left in place until azotemia is resolved, the urine is clear, and the cat is on a reasonable fluid rate, or at least 24 hours, whichever comes first.

In-hospital treatment should include pain medications such as gabapentin or buprenorphine, and may include a muscle relaxant such as acepromazine, prazosin or phenoxybenzamine. Meloxicam may be prescribed, although clinical efficacy is unknow. Decompressive cystocentesis have been advocated by some. Potential benefits may include pain relief, ease of urinary catheterization and need to perform diagnostic testing prior to urinary catheterization. Obvious risks are bladder rupture and aortic laceration. Complication rates are below 5%, but can be devastating.

Intermittent bladder flushing has been advocated by some but does not seem to decrease the recurrence of urethral obstruction. Similarly, the type of urinary catheter used did not seem to influence recurrence of urethral obstruction.

The cat can be discharged soon after removing the catheter, as long as he can urinate on his own. To-go-home medications used varies, but can include gabapentin, buprenorphine, acepromazine or prazosin. The use of prazosin has been shrouded with controversies lately.

Although hospitalization and indwelling catheterization is the recommended treatment, an outpatient protocol has been described.

Treating the cause, which is usually feline idiopathic cystitis, with increased access to water, stress management and other environmental interventions. More information can be found at https://indoorpet.osu.edu/cats.

Prognosis: Short term prognosis is usually good, with discharge rate up to 90%, with the understanding that 51% may have recurrent signs, 36% experience re-obstruction, and 21% may eventually be euthanized.

References:

Cosford, K. L. & Koo, S. T. In-hospital medical management of feline urethral obstruction: A review of recent clinical research. Can. Vet. J. Rev. Veterinaire Can. 61, 595–604 (2020).

Drobatz, K. J. & Cole, S. G. influence of crystalloid type on acid-base and electrolyte status of cats with urethral obstruction. J. Vet. Emerg. Crit. Care (2008).

Ostroski, C. J., Drobatz, K. J. & Reineke, E. L. Retrospective evaluation of and risk factor analysis for presumed fluid overload in cats with urethral obstruction: 11 cases (2002-2012). J. Vet. Emerg. Crit. Care San Antonio Tex 2001 27, 561–568 (2017).

Muller, K. M., Burkitt-Creedon, J. M. & Epstein, S. E. Presentation Variables Associated With the Development of Severe Post-obstructive Diuresis in Male Cats Following Relief of Urethral Obstruction. Front. Vet. Sci. 9, 783874 (2022).

Pratt, C. L., Balakrishnan, A., McGowan, E., Drobatz, K. J. & Reineke, E. L. A prospective randomized, double-blinded clinical study evaluating the efficacy and safety of bupivacaine versus morphine-bupivacaine in caudal epidurals in cats with urethral obstruction. J. Vet. Emerg. Crit. Care San Antonio Tex 2001 30, 170–178 (2020).

Cooper, E. S. Controversies in the management of feline urethral obstruction. J. Vet. Emerg. Crit. Care San Antonio Tex 2001 25, 130–137 (2015).

Young, C. S., Todd, J. M., Rendahl, A. & Lulich, J. P. Radiographic diagnoses in 80 cats before and 73 cats after unobstructing the urethra. J. Small Anim. Pract. 62, 365–372 (2021).

Reineke, E. L., Cooper, E. S., Takacs, J. D., Suran, J. N. & Drobatz, K. J. Multicenter evaluation of decompressive cystocentesis in the treatment of cats with urethral obstruction. J. Am. Vet. Med. Assoc. 258, 483–492 (2021).

Seitz, M. A., Burkitt-Creedon, J. M. & Drobatz, K. J. Evaluation for association between indwelling urethral catheter placement and risk of recurrent urethral obstruction in cats. J. Am. Vet. Med. Assoc. 252, 1509–1520 (2018).

FELINE CIRCULATORY SHOCK

Ivayla Danielova Yozova [1]

[1] School of Veterinary Science - Tāwharau Ora, Massey University, Palmerston North, New Zealand

Learning objectives:

- Understand the pathophysiology of feline circulatory shock.

- Refresh on the available diagnostic tools and their feline specifics.

- Recognise the corner stones of supportive care.

- Treat circulatory shock sensibly and with moderation.

- Update on current veterinary evidence.

Proceeding:

Pathophysiology

Cats very commonly manifest circulatory shock as though in hibernation – with bradycardia, hypotension, and hypothermia. This “feline shock triad” has been well described in textbooks, proceedings and review articles. Components of it have been documented in studies in critically ill cats with diseases such as trauma, sepsis and toxicities and have been associated with prognosis. Furthermore, it lies in the collective memory of all vets that have dealt with a “flat cat” in shock. The processes involved in the clinical manifestation of feline circulatory shock seem to be driven by complex neurohormonal responses, such as central inhibition of the thermoregulatory centre (hypothermia), the adrenergic receptor hyporesponsiveness (vasodilation) and the Bezold-Jerisch reflex (bradycardia) among other phenomena. This evidence is mainly from experimental studies. Considering the complex interactions between the components contributing to the clinical manifestation of feline circulatory shock, it is difficult to believe that the ultimate goal is for them to precipitate death of the sick or injured feline. Rather, it is increasingly believed that, similarly to animals undergoing hibernation (where slowing down metabolism is key to survival), these reflexes might be protective in nature.

Clinical recognition

Clinical recognition can be sometimes straightforward and sometimes challenging especially in felines. Our “diagnostic toolbox” relies on context (history of an event that could lead to circulatory shock), clinical perfusion parameters, non-invasive blood pressure measurements and laboratory variables such as lactate concentration among others. A valuable add-on in the past decade is point-of-care ultrasound.

All the above-mentioned modalities have strengths and weaknesses and there is not one tool capable of single-handedly diagnosing shock. Therefore, they should be used in unison.

Therapeutic approach

Currently there are no evidence-based guidelines for stabilization of circulatory shock in felines. Since shock is not a disease, but rather a sign of severity of disease, supportive care, such as active rewarming, rational IV fluid administration, oxygen therapy, sedation and analgesia (when appropriate) are the pillars of stabilization in addition to finding and treating the underlying cause timely (with tools that do not hinder stabilization). Early vasopressors should also be considered in some scenarios.

Active rewarming

While mild hypothermia is somewhat protective in nature, severe hypothermia can be detrimental. Slow active rewarming (~ 0.5 – 1°C) is recommended to avoid complications such as rewarming shock, afterdrop or rescue collapse. Maintaining mild hypothermia (~ 36°C) has the advantage of reducing metabolic rates and potentially observing whether the patient can increase or maintain its own temperature. These clinical recommendations remain largely empirical.

IV fluid administration

IV fluid administration should be considered very carefully and rationally in cats with circulatory shock. Overall, aggressive IV fluid resuscitation, especially if they are hypothermic, is not recommended for several reasons. Firstly, it is very unlikely to work. Vasodilation is at the centre of feline shock pathophysiology. Secondly, absolute hypovolemia, from haemorrhage or acute severe dehydration, requiring rapid IV fluid “replacement”, is not a common presentation in cats. The few studies with epidemiological data on cats presented to the ER, report commonly underlying diseases with a maldistributive component (such as trauma or sepsis). This lack of fluid responsiveness was reported in two recent clinical studies showing that only 37% and 42% of cats were responders, respectively. Lastly, aggressive fluid resuscitation may lead to volume overload and oedema. This ties in with the concept that cats are in general predisposed to volume overload, because of their smaller blood volume, relatively high incidence of asymptomatic cardiomyopathies, and slower IV fluid elimination rates. Furthermore, anaemia is common in sick cats, which also leads to volume overload. For all these reasons, low volume IV fluid challenges should be implemented with careful monitoring for a physiological response and ceased immediately in the lack of such a response. In cases of moderate to severe hypothermia, some rewarming should be considered first.

Conclusion

Outside of the experimental studies, all the data that we have on cats in circulatory shock are from the ones that have been *admitted* to the ER. Consider an outdoor cat that had suffered road vehicle trauma away from home, develops this pathophysiologically complex circulatory shock, but instead of being brought to the ER and treated aggressively for it, it hides somewhere until it can recover, and returns home a day later only with a “funny leg”. It is perhaps time to acknowledge that feline circulatory shock is very likely protective in nature. Therefore, our efforts should be to support the patient, rather than attempt to reverse all parameters “back to normal”.

References:

Rudloff E, Kirby R. Feline Circulatory Shock In: Little S, ed. August's Consultations in Feline Internal Medicine St Lous, USA Elsevier 2016;843-858.

Oncken AK, Kirby R, Rudloff E. Hypothermia in Critically III Dogs and Cats. COMPENDIUM ON CONTINUING EDUCATION FOR THE PRACTISING VETERINARIAN-NORTH AMERICAN EDITION-2001;23:506-523.

R K. Feline Shock and Resuscitation. WSAVA 2005.

Ottenjann M, Lübke-Becker A, Linzmann H, et al. [Pyothorax in 26 cats: clinical signs, laboratory results and therapy (2000-2007)]. Berl Munch Tierarztl Wochenschr 2008;121:365-373.

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.

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.

Nascimento DC, Costa Neto JM, Solca 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 2021:1098612X211028027.

Fitzgerald WR CN, 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. Journal of Feline Medicine and Surgery 2022.

Ruthrauff CM, Smith J, Glerum L. Primary bacterial septic peritonitis in cats: 13 cases. J Am Anim Hosp Assoc 2009;45:268-276.

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.

Silverstein DC, Wininger FA, Shofer FS, et al. Relationship between Doppler blood pressure and survival or response to treatment in critically ill cats: 83 cases (2003-2004). J Am Vet Med Assoc 2008;232:893-897.

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.

Myers RD, Beleslin DB, Rezvani AH. Hypothermia: role of alpha 1- and alpha 2-noradrenergic receptors in the hypothalamus of the cat. Pharmacol Biochem Behav 1987;26:373-379.

Oberg B, Thoren P. Increased activity in left ventricular receptors during hemorrhage or occlusion of caval veins in the cat. A possible cause of the vaso-vagal reaction. Acta Physiol Scand 1972;85:164-173.

Schadt JC, Ludbrook J. Hemodynamic and neurohumoral responses to acute hypovolemia in conscious mammals. Am J Physiol 1991;260:H305-318.

Alboni P, Alboni M, Bertorelle G. The origin of vasovagal syncope: to protect the heart or to escape predation? Clin Auton Res 2008;18:170-178.

Mark AL. The Bezold-Jarisch reflex revisited: clinical implications of inhibitory reflexes originating in the heart. J Am Coll Cardiol 1983;1:90-102.

Vayssettes-Courchay C, Bouysset F, Laubie M, et al. Central integration of the Bezold-Jarish reflex in the cat. Brain Res 1997;744:272-278.

Kudryashov YA, Tabarov MS, Tkachenko BT. Adrenergic reactivity of visceral veins in hypoxia and hypothermia. Bull Exp Biol Med 2000;130:1063-1065.

Han YS, Tveita T, Prakash YS, et al. Mechanisms underlying hypothermia-induced cardiac contractile dysfunction. Am J Physiol Heart Circ Physiol 2010;298:H890-897.

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.

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.

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.

Simpson KE, McCann TM, Bommer NX, et al. Retrospective analysis of selected predictors of mortality within a veterinary intensive care unit. J Feline Med Surg 2007;9:364-368.

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.

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.

McMurray J, Boysen S, Chalhoub S. Focused assessment with sonography in nontraumatized dogs and cats in the emergency and critical care setting. J Vet Emerg Crit Care (San Antonio) 2016;26:64-73.

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.

Gilday C, Guieu L. Prognostic value of lactate in cats presented in respiratory distress to the emergency room. Front Vet Sci 2022;9:918029.

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.

Groom AC, Rowlands S. The cardiac output and blood volume of the anaesthetized cat. Phys Med Biol 1958;3:138-156.

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.

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.

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.

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.

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.

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.

Advanced Stream, Friday 31 May 2024

HYPERTENSIVE EMERGENCIES

Dave Beeston [1, 2]

[1] Willows Veterinary Referral Centre, Emergency and Critical Care, Birmingham, United Kingdom
[2] Royal Veterinary College, Emergency and Critical Care, Hatfield, United Kingdom

Learning objectives:

- To understand the normal regulation of vascular tone

- To understand the difference between hypertensive emergency and hypertensive urgency

- To recognise the common clinical presentations of hypertensive emergency

- To understand the relevant pharmacology and management strategies of hypertensive emergency

Proceeding:

Physiology and Definitions

Arterial blood pressure is tightly regulated in all mammals through various neuro-humoral, myogenic, metabolic and flow-mediated processes (Magder, 2018). Maintaining arterial blood pressure within a physiologic range of 50 – 150mmHg allows autoregulation of flow to ensure constant delivery of oxygen and nutrients to target tissues (Johnson, 1986).

Systemic hypertension has been associated with many conditions in veterinary species including acute kidney injury (AKI) (Cole et al., 2020), chronic kidney disease (D. G. O'Neill et al., 2013), diabetes mellitus (Herring et al., 2014), immune-mediated haemolytic anaemia (Hall et al., 2022) and pheochromocytoma (Gilson et al., 1994) in dogs, and most commonly chronic kidney disease (Conroy et al., 2019), hyperthyroidism (Williams et al., 2013) and primary hyperaldosteronism (Ash et al., 2005) in cats.

Hypertensive crisis is defined in human medicine as systolic blood pressure (SBP) or diastolic blood pressure (DBP) greater than 180mmHg or 120mmHg, respectively. Hypertensive crisis is then further classified into hypertensive emergency (HE) or hypertensive urgency (HU) based on the presence, or absence, of target organ damage (TOD) (Rodriguez et al., 2010). Risk of TOD in the presence of systemic hypertension has been well defined in veterinary medicine and includes damage to the kidneys, eyes, brain and heart (Acierno et al., 2018). Overt TOD in dogs and cats may most readily be recognised in the presence of ocular (blindness, retinal detachment or haemorrhage, hyphaema) or neurological (seizures, altered mentation) disease (Acierno et al., 2018).

Clinical presentation

Literature on HE in veterinary medicine is scarce (Beeston et al., 2022; Brown et al., 2005; Church et al., 2019; J. O'Neill et al., 2013). In a recent case series, neurological signs were documented in 80% of dogs and cats with confirmed HE, most commonly manifesting in seizures (Beeston et al., 2022). This poses a challenge as the association of neurologic signs and hypertension should prompt the clinician to consider intracranial hypertension and the Cushing reflex (Fodstad *et al.*, 2006). Whilst vasogenic and interstitial oedema are frequently seen with hypertensive encephalopathy, clinical signs in patients with oedema secondary to hypertensive emergency would not be expected to completely resolve with the administration of a hyperosmolar agent alone. Additionally, acute onset blindness was seen frequently with the majority of patients with HE having fundoscopic abnormalities (Beeston et al., 2022).

Underlying pathology

The most reported cause of HE in dogs and cats is AKI (Beeston et al., 2022). Systemic hypertension at admission or during hospitalisation has been documented in 75% of dogs and cats with AKI and has been reported to be severe (>180mmHg) in most cases. Prompt recognition of systemic hypertension and regular monitoring of non-invasive blood pressure should be considered for all patients with AKI to minimise ongoing TOD. Additional differentials include idiopathic hypertension, hyperthyroidism and cutaneous and renal glomerular vasculopathy (Beeston et al., 2022).

Management strategies

Guidelines for the management of HE in veterinary patients do not exist. Treatment goals of HE in people include a reduction in mean arterial pressure by 20-25% in the first hour, followed by a gradual reduction to an SBP/DBP of 160/100mmHg to 160/110mmHg over 2-6 hours and further decrease to normotension over the following days (Rodriguez, 2010; Whelton *et al.*, 2018; Van Den Born *et al.*, 2019).

Amlodipine is commonly used as a first-line agent in veterinary HE (Beeston et al., 2022). Whilst considered for use in hypertensive urgency, oral medications are not recommended in human patients with HE due to the delayed onset of action and difficulty in titration (Van Den Born et al., 2019). A variety of parenteral medications are recommended by the European Society of Cardiology guidelines for management of hypertensive emergency including labetalol, nicardipine, nitroglycerine, nitroprusside and esmolol depending on the underlying aetiology and clinical presentation (Van Den Born et al., 2019). For example, the majority of patients with HE are recommended to have labetalol or nicardipine, however, patients presenting with acute cardiogenic pulmonary oedema are likely to benefit from sodium nitroprusside (Van Den Born et al., 2019). Information regarding the haemodynamic effects of labetalol and nicardipine in veterinary medicine is limited. Labetalol is a combined alpha- and beta-blocker that has been assessed as an anti-hypertensive agent in a population of dogs undergoing surgical procedures and was shown to have a reliable, titratable effect on mean arterial pressure when administered as an infusion (Zublena et al., 2020). Additionally, labetalol has been used to counteract alpha-2-agonist mediated vasoconstriction in healthy dogs undergoing ovariohysterectomy (Sández et al., 2023). Further studies are needed to assess the use of labetalol in hypertensive emergency before formal recommendations can be made. Given the lack of evidence for or against amlodipine in veterinary HE, it would be sensible to consider when parenteral agents aren't available or prior to referral.

Outcome

Unfortunately, hypertensive emergency has been associated with a poor prognosis with an in-hospital mortality of almost 50% (Beeston et al., 2022). However, as with all retrospective studies, caution should be applied when interpreting mortality data as most patients were euthanased and the overall mortality associated with acute kidney injury alone has varied from 34-51% (Legatti et al., 2018; Rimer et al., 2022).

References:

Acierno, M. J., Brown, S., Coleman, A. E., Jepson, R. E., Papich, M., Stepien, R. L., & Syme, H. M. (2018). ACVIM consensus statement: Guidelines for the identification, evaluation, and management of systemic hypertension in dogs and cats. Journal of Veterinary Internal Medicine, 32(6), 1803–1822. https://doi.org/10.1111/JVIM.15331

Ash, R. A., Harvey, A. M., & Tasker, S. (2005). Primary hyperaldosteronism in the cat: A series of 13 cases. Journal of Feline Medicine and Surgery, 7(3), 173–182. https://doi.org/10.1016/j.jfms.2004.08.007

Beeston, D., Jepson, R., & Cortellini, S. (2022). Evaluation of presentation, treatment and outcome in hypertensive emergency in dogs and cats: 15 cases (2003-2019). Journal of Small Animal Practice, 1–8. https://doi.org/10.1111/JSAP.13530

Brown, C. A., Munday, J. S., Mathur, S., & Brown, S. A. (2005). Hypertensive encephalopathy in cats with reduced renal function. Veterinary Pathology, 42(5), 642–649. https://doi.org/10.1354/vp.42-5-642

Church, M. E., Turek, B. J., & Durham, A. C. (2019). Neuropathology of Spontaneous Hypertensive Encephalopathy in Cats. Veterinary Pathology, 56(5), 778–782. https://doi.org/10.1177/0300985819849500

Cole, L. P., Jepson, R., Dawson, C., & Humm, K. (2020). Hypertension, retinopathy, and acute kidney injury in dogs: A prospective study. Journal of Veterinary Internal Medicine, 34(5), 1940. https://doi.org/10.1111/JVIM.15839

Conroy, M., Brodbelt, D. C., O'Neill, D., Chang, Y.-M., & Elliott, J. (2019). Chronic kidney disease in cats attending primary care practice in the UK: a VetCompassTM study. Veterinary Record, 184(17), 526–526. https://doi.org/10.1136/VR.105100

Fodstad, H., Kelly, P. J., & Buchfelder, M. (2006). History of the cushing reflex. Neurosurgery, 59(5), 1132–1137. https://doi.org/10.1227/01.NEU.0000245582.08532.7C

Gilson, S. D., Withrow, S. J., Wheeler, S. L., & Twedt, D. C. (1994). Pheochromocytoma in 50 dogs. Journal of Veterinary Internal Medicine, 8(3), 228–232. https://doi.org/10.1111/J.1939-1676.1994.TB03222.X

Hall, G. B. F., Stoye, D. Q., Thomas, E. K., & Culshaw, G. J. (2022). Retrospective evaluation of systemic hypertension in dogs with nonassociative (primary) immune-mediated hemolytic anemia (December 2016 to April 2019): 26 cases. Journal of Veterinary Emergency and Critical Care (San Antonio, Tex. : 2001). https://doi.org/10.1111/VEC.13128

Herring, I. P., Panciera, D. L., & Werre, S. R. (2014). Longitudinal prevalence of hypertension, proteinuria, and retinopathy in dogs with spontaneous diabetes mellitus. Journal of Veterinary Internal Medicine, 28(2), 488–495. https://doi.org/10.1111/JVIM.12286

Johnson, P. C. (1986). Autoregulation of blood flow. Circulation Research, 59(5), 483–495. https://doi.org/10.1161/01.RES.59.5.483

Legatti, S. A. M., Dib, R. El, Legatti, E., Botan, A. G., Camargo, S. E. A., Agarwal, A., Barretti, P., & Paes, A. C. (2018). Acute kidney injury in cats and dogs: A proportional meta-analysis of case series studies. PLOS ONE, 13(1), e0190772. https://doi.org/10.1371/JOURNAL.PONE.0190772

Magder, S. (2018). The meaning of blood pressure. In Critical Care (Vol. 22, Issue 1). Crit Care. https://doi.org/10.1186/s13054-018-2171-1

O'Neill, D. G., Elliott, J., Church, D. B., Mcgreevy, P. D., Thomson, P. C., & Brodbelt, D. C. (2013). Chronic kidney disease in dogs in UK veterinary practices: prevalence, risk factors, and survival. Journal of Veterinary Internal Medicine, 27(4), 814–821. https://doi.org/10.1111/JVIM.12090

O'Neill, J., Kent, M., Glass, E. N., & Platt, S. R. (2013). Clinicopathologic and MRI characteristics of presumptive hypertensive encephalopathy in two cats and two dogs. Journal of the American Animal Hospital Association, 49(6), 412–420. https://doi.org/10.5326/JAAHA-MS-5942

Rimer, D., Chen, H., Bar-Nathan, M., & Segev, G. (2022). Acute kidney injury in dogs: Etiology, clinical and clinicopathologic findings, prognostic markers, and outcome. Journal of Veterinary Internal Medicine, 36(2), 609–618. https://doi.org/10.1111/jvim.16375

Rodriguez, M. A., Kumar, S. K., & De Caro, M. (2010). Hypertensive crisis. In Cardiology in Review (Vol. 18, Issue 2, pp. 102–107). Cardiol Rev. https://doi.org/10.1097/CRD.0b013e3181c307b7

Sández, I., Martín-Flores, M., Portela, D. A., Márquez-Grados, F., & Monge-García, M. I. (2023). Haemodynamic effects of labetalol in isoflurane-anaesthetized dogs that received dexmedetomidine: A randomized clinical trial. Veterinary Anaesthesia and Analgesia. https://doi.org/10.1016/J.VAA.2023.10.031

Van Den Born, B. J. H., Lip, G. Y. H., Brguljan-Hitij, J., Cremer, A., Segura, J., Morales, E., Mahfoud, F., Amraoui, F., Persu, A., Kahan, T., Agabiti Rosei, E., De Simone, G., Gosse, P., & Williams, B. (2019a). ESC Council on hypertension position document on the management of hypertensive emergencies. European Heart Journal - Cardiovascular Pharmacotherapy, 5(1), 37–46. https://doi.org/10.1093/EHJCVP/PVY032

Van Den Born, B. J. H., Lip, G. Y. H., Brguljan-Hitij, J., Cremer, A., Segura, J., Morales, E., Mahfoud, F., Amraoui, F., Persu, A., Kahan, T., Agabiti Rosei, E., De Simone, G., Gosse, P., & Williams, B. (2019b). ESC Council on hypertension position document on the management of hypertensive emergencies. European Heart Journal - Cardiovascular Pharmacotherapy, 5(1), 37–46. https://doi.org/10.1093/EHJCVP/PVY032

Whelton, P. K., Carey, R. M., Aronow, W. S., Casey, D. E., Collins, K. J., Himmelfarb, C. D., DePalma, S. M., Gidding, S., Jamerson, K. A., Jones, D. W., MacLaughlin, E. J., Muntner, P., Ovbiagele, B., Smith, S. C., Spencer, C. C., Stafford, R. S., Taler, S. J., Thomas, R. J., Williams, K. A., … Wright, J. T. (2018). 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ ASPC/NMA/PCNA guideline for the prevention, detection,

evaluation, and management of high blood pressure in adults: Executive summary: A report of the American college of cardiology/American Heart Association task force on clinical practice guidelines. Hypertension, 71(6), 1269–1324. https://doi.org/10.1161/HYP.0000000000000066/-/DC2

Williams, T. L., Elliott, J., & Syme, H. M. (2013). Renin-Angiotensin-Aldosterone System Activity in Hyperthyroid Cats with and without Concurrent Hypertension. Journal of Veterinary Internal Medicine, 27(3), 522–529. https://doi.org/10.1111/JVIM.12062

Zublena, F., De Gennaro, C., & Corletto, F. (2020). Retrospective evaluation of labetalol as antihypertensive agent in dogs. BMC Veterinary Research, 16(1), 1–8. https://doi.org/10.1186/S12917-020-02475-4/TABLES/2

USING CRITICAL CARE ECHOCARDIOGRAPHY (CCE) TO GUIDE THERAPY IN CRITICALLY ILL PATIENTS

Christopher Kennedy [1]

[1] University of Illinois, Emergency and Critical Care Service, Urbana, United States

Learning objectives:

- understand the difference between formal echocardiography performed by a cardiologist, focused cardiac ultrasound and critical care echocardiography

- explain the mathematics behind stroke volume and cardiac output measurement using Doppler echocardiography

- appreciate where critical care echocardiography may be beneficial in managing a critically ill patient

Proceeding:

In human medicine, critical care echocardiography (CCE) is divided into basic and advanced CCE. In veterinary medicine, we use 'focused cardiac ultrasound' (FCU) as synonymous with basic CCE and 'critical care echocardiography' to refer to advanced CCE. This terminology is helpful, as it reminds critical care practitioners that formal echocardiography remains firmly in the realm of cardiology practitioners and does not mislead clients.

CCE is a new horizon in veterinary critical care. Employing 2-dimensional cardiac ultrasound in FCU and Doppler ultrasound in CCE permits non-invasive, bedside and real-time insight into fluid status (volemia, tolerance and responsiveness) and cardiovascular status. Importantly, no single test can provide all or perfect information – much like lactate is just one piece of the haemodynamic puzzle, FCU and CCE are additive to the standard clinical approach.

FCU provides information on cardiac structure and some information on cardiovascular function; however, much of this information is speculative and, unless it is integrated with the clinical examination, can be wrong. An example is the erroneous prophesying of cardiac output based on inaccurate assumptions of left ventricular systolic function. FCU is perhaps best at identifying 1) pericardial effusion, 2) marked right sided heart disease, and 3) overt fluid intolerance.

CCE can provide haemodynamic data. The velocity-time integral (VTI) of blood flow through the left ventricular outflow tract is one datapoint that can be used to determine stroke volume, cardiac output and response, or lack thereof, to an intervention. When combined with the physical examination and other haemodynamic data (blood pressure), we can gather insight into cardiac function, vascular function and combined cardio-vascular function.

This lecture provides insights into some aspects of CCE and how they may be used in decision making. Comprehensive CCE can be technically challenging, time consuming and requires repeating in full or in part several times in the same unstable patient. These issues limit the effectiveness of CCE in critical patients, which has been demonstrated in human critical care. However, when used logically, CCE can help guide interventions, such as fluid administration or evacuation, inopressor medications and mechanical ventilation.

MULTIDISCIPLINARY APPROACH TO PULMONARY HYPERTENSION

Teresa DeFrancesco [1]

[1] NC State University College of Veterinary Medicine, Department of Clinical Sciences, Raleigh, United States

Learning objectives:

- To understand that a comprehensive clinical assessment is indicated for the diagnosis of pulmonary hypertension.

- To review the utility and clinical findings of imaging (radiographs, echocardiography, CT angiography) and laboratory tests for the diagnosis of pulmonary hypertension.

- To review the new ACVIM clinical classification scheme for pulmonary hypertension in dogs.

- To review the current and promising treatments strategies for the management of pulmonary hypertension.

Proceeding:

General considerations

Pulmonary hypertension (PH) is a complex clinical syndrome, caused by a wide variety of cardiovascular, respiratory, and systemic diseases. A comprehensive approach is recommended to diagnose and treat PH and its underlying conditions. PH is defined as increased pressures in the blood vessels in the lungs. This high pressure in the pulmonary vasculature results from the following (either alone or in combination): increased pulmonary blood flow, increased pulmonary vascular resistance (PVR), and increased pulmonary venous pressure. Increased pulmonary arterial (PA) pressure associated with increased PVR in the absence of increased left atrial pressure defines precapillary PH while increased PA pressures associated with increased pulmonary venous pressures is referred to as postcapillary PH. Postcapillary PH occurs most commonly in dogs with left heart disease that have increased left atrial pressure. In the recent ACVIM consensus on PH, dogs were categorized into 6 clinical groups based on the cause and pathophysiology of the PH. These classifications are designed to improve the diagnostic approach and treatment of a dog with suspected or confirmed PH. The groups are: Group 1 - PH due to PA hypertension; Group 2 - PH due to left heart disease; Group 3 - PH due to chronic lung disease and/or hypoxia; Group 4 - PH due to pulmonary thromboembolic disease; Group 5 - PH due to parasitic diseases (Dirofilaria and Angiostrongylus); and Group 5 - PH due to unclear or multifactorial mechanisms. Regardless of the underlying mechanism of PH, with sustained and severe PH, the right ventricle (RV)

must work harder against increased pulmonary pressures resulting in structural alterations associated with increased afterload and can ultimately lead to right heart failure. The diagnosis of PH may be suspected when a dog presents with a cough, dyspnea, lethargy, syncope, exercise intolerance, or abdominal distension (ascites). Unfortunately, these clinical signs as well as the physical examination findings can be associated with heart failure or primary respiratory disease.

Diagnostic evaluation

The use of echocardiography (2D and doppler) should be an essential and early diagnosis test in the assessment of suspected PH especially if thoracic radiography shows evidence supportive of PH such as dilated or tortuous pulmonary arteries; pulmonary artery bulge; right heart enlargement, and pulmonary infiltrates. Echocardiography can help to determine the cause, and the probability and severity of the PH. Echocardiographic findings of PH include high velocity tricuspid valve regurgitation (> 3 m/s) or pulmonary valve regurgitation (> 2.5 m/s); RV mixed hypertrophy; RV systolic dysfunction (low TAPSE); interventricular septal flattening with underfilled left ventricle; PA enlargement, decreased PV:PA (PV/PA < 0.7) or decreased right PA distensibility (RPAD < 30%); short PA acceleration time (< 52 ms or AT:ET < 0.3) or systolic notching of the RV outflow tract flow profile; right atrial enlargement and distended caudal vena cava. Point of care ultrasound (POCUS) can also identify some 2D findings suggest of PH, notably the flattened interventricular septum and underfilled left ventricle. A recent study showed that a relatively new and easily obtainable left ventricular eccentricity index of > 1.24 – 1.4 optimally discriminated moderate and severe precapillary PH from no and mild PH. The LV eccentricity index, obtained from a right parasternal short axis view just below the mitral valves, is the ratio of the cranio-caudal:latero-lateral left ventricular diameter. It essentially is an assessment of the LV morphology and flattening of the interventricular septum. Other non-cardiac POCUS findings compatible with PH are lung ultrasound abnormalities such as B-lines or shreds, free fluid in the chest and/or abdomen. During the echocardiograph exam, one can look for underlying causes such as parasites (group 5), pulmonary thrombus (group 3), or signs of increased left heart disease (group 2). An enlarged left atrium is a strong indicator of increased pulmonary venous pressures in a dog with high velocity tricuspid regurgitation. Laboratory testing may also yield some diagnostic value in the work up especially in a case of suspected or confirmed pulmonary thromboembolic PH. These tests that serve to assess associated conditions and risk factors for hypercoagulability include but are not limited to complete blood count, serum biochemical profile, parasite testing, urinalysis, urine protein:creatinine ratio, thromboelastography (TEG), and D-dimers. Unfortunately, TEG and d-dimers can be normal in dogs with confirmed pulmonary thromboembolism. If the clinical scenario, radiographs, echocardiogram and routine laboratory tests do not identify the underlying cause, further advanced diagnostics, specifically a CT angiogram and respiratory work up (BAL, bronchoscopy, cytology and cultures), may be pursued. A thoracic CT can provide supportive or definitive evidence for pulmonary vascular diseases, pulmonary parenchymal diseases (neoplasia) and pulmonary thromboembolic diseases. Alternatively, empiric treatment could be pursued at this junction.

Treatment

Treatment of PH can be subdivided into: 1. Treatments to lower PA pressure (PH-specific treatment), 2. Treatment of the underlying diseases and 3. Treatments to decrease the risk of PH progression or complications. Sildenafil, a PDE5 inhibitor, specifically targets and increases nitric oxide in the pulmonary arteries leading to decreased pulmonary vascular resistance. Sildenafil (1-3 mg/kg q8h) is the most common drug used in the management of PH in dogs. Most clinical canine studies show improved clinical signs even though the tricuspid valve regurgitation velocity may not be decreased. Tadalafil (1-2 mg/kg) is another PDE5 inhibitor that has a longer half-life, allowing for q24h dosing. PDE5 inhibitors should be used with caution in dogs with post capillary PH due to left heart disease as it could increase pulmonary flow and lead to pulmonary edema. The author typically cautiously adds a PDE5 inhibitor only in dogs with post-capillary PH if they have ascites. Pimobendan is another drug that is often used in the setting of PH with or without left heart disease. Pimobendan effectively improves RV/LV function and stroke volume but may not necessarily decrease PA pressure. A recent case series in five dogs from Canada with sildenafil-refractory pulmonary hypertension showed that Ambrisentan improved appetite, activity, and respiratory functions when added to sildenafil. Ambrisentan is an endothelin-A selective receptor antagonist. While in veterinary medicine we are limited in PH-specific drugs, there are several other drugs used in the management of PH for humans. The list of currently approved drugs for PH in the US include: Epoprostenol and Treprostinil (prostacyclins delivered via continuous infusion); Iloprost and Treprostinil (prostacyclins delivered by frequent inhalation); Bosentan and Ambrisentan (oral endothelin antagonists); Sildenafil and Tadalafil (oral PDE5 inhibitors); and Riociguat (oral soluble guanylate cyclase). Riociguat, a pulmonary vasodilator used to treat PH secondary to chronic thromboembolic PH in humans, was recently evaluated in experimental PH in dogs showing promising results. Riociguat, inhibited in the increase of PA pressure through PA relaxation via an endothelium-independent increase in cGMP. Beraprost, a prostacyclin analog, is another drug that has been studied in dogs in an experimental settings showing promise. A new drug in the human market, Selexipeg (available in IV and oral formulations) is prostacyclin receptor agonist that has been shown in clinical trials in adult and children with PH to improve clinical signs and progression of PH. Another drug that shows promise is Sotatercept, improving exercise capacity as compared to placebo in recent human clinical trials. Sotatercept is a novel protein that traps activins and growth differentiation factors involved in pulmonary arterial hypertension. Unfortunately, these aforementioned drugs (aside from the PDE5i and ambrisentan) have not been evaluated in veterinary clinical medicine to the author knowledge. And lastly, supplemental oxygen for a dog in respiratory distress is advised in hospital for its PA vasodilatory effects. Long-term oxygen therapy is feasible in dogs and may provide survival benefits in humans with interstitial lung disease and low diffusion capacity for carbon monoxide. Specific treatments of underlying diseases are beyond the scope of this lecture but are important to in the management of PH.

References:

Farber HW, Badesch DB, Benza RL, et al. Use of supplemental oxygen in patients with pulmonary arterial hypertension in REVEAL. J Heart Lung Transplant. 2018 Aug;37(8):948-955.

Goggs R, Chan DL, Benigni L, Hirst C, Kellett-Gregory L, Fuentes VL. Comparison of computed tomography pulmonary angiography and point-of-care tests for pulmonary thromboembolism diagnosis in dogs. J Small Anim Pract. 2014;55:190-7.

Goya S, Yoshida T, Sennba S, Uchide T, Tanaka R. Adjunct ambrisentan therapy had clinical benefits in 5 dogs with sildenafil-refractory pulmonary hypertension. Can Vet J. 2022;63:497-503.

Jaffey JA, Leach SB, Kong LR, Wiggen KE, Bender SB, Reinero CR. Clinical efficacy of tadalafil compared to sildenafil in treatment of moderate to severe canine pulmonary hypertension: a pilot study. J Vet Cardiol. 2019;24:7-19.

Johnson LR, Stern JA. Clinical features and outcome in 25 dogs with respiratory-associated pulmonary hypertension treated with sildenafil. J Vet Intern Med. 2020 Jan;34(1):65-73.

Kameshima S, Nakamura Y, Uehara K, et al. Effects of a Soluble Guanylate Cyclase Stimulator Riociguat on Contractility of Isolated Pulmonary Artery and Hemodynamics of U46619-Induced Pulmonary Hypertension in Dogs. Vet Sci. 2023;10:159. • Khan S, Randhawa S, Patail H, Spira Y, Frishman WH, Aronow WS, Lanier GM. Pharmacological Update and Emerging Treatments of Pulmonary Hypertension. Cardiol Rev. 2024 Jan 31. doi: 10.1097/CRD.0000000000000647. Epub ahead of print. PMID: 38294225.

Lekane M, Burnotte P, Gommeren K, Mc Entee K, Merveille AC. Left ventricular eccentricity index to assess precapillary pulmonary hypertension in dogs. J Vet Cardiol. 2023;51:220-231.

Marschner CB, Kristensen AT, Rozanski EA, et al. Diagnosis of canine pulmonary thromboembolism by computed tomography and mathematical modelling using haemostatic and inflammatory variables. Vet J. 2017;229:6-12.

Min S, Wesselowski SR, Nabity MB, Yankin I. Pulmonary hypertension is associated with hypocoagulability in dogs: a retrospective analysis of 66 cases (2013-2021). Am J Vet Res. 2024;12:1-8.

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.

Roels E, Fastrès A, Merveille AC, et al. The prevalence of pulmonary hypertension assessed using the pulmonary vein-to-right pulmonary artery ratio and its association with survival in West Highland white terriers with canine idiopathic pulmonary fibrosis. BMC Vet Res. 2021;17:171.

Suzuki R, Yuchi Y, Saito T, Teshima T, Matsumoto H, Koyama H. Investigation of Beraprost Sodium on Cardiac Function and Hemodynamics in Canine Models of Chronic Pulmonary Hypertension. Front Vet Sci. 2022 Apr 14;9:876178.

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 Jul;34(4):1379-1388.

Yuchi Y, Suzuki R, Saito T, et al. Echocardiographic characteristics of dogs with pulmonary hypertension secondary to respiratory diseases. J Vet Intern Med. 2023;37:1656-1666.

SEPSIS CONSENSUS DEFINITIONS

Robert Goggs [1]

[1] Cornell University College of Veterinary Medicine, Clinical Sciences, Ithaca, United States

Learning objectives:

- Understand the evolution of sepsis definitions in human and veterinary medicine.

- State how sepsis is presently defined in veterinary medicine.

- Understand the major limitations of the present definitions.

- Recognize the differences between the human and veterinary definitions.

- Describe the process by which new veterinary consensus definitions are being generated.

Proceeding:

Veterinary sepsis definitions

Before 2016, sepsis in veterinary medicine was typically defined per the premise of the 1991/2001 human consensus definitions, i.e. SIRS plus documented or suspected infection. Within the literature, and likely within clinical practice there are variations in what constitutes documentation and suspicion of infection. The standard methods described for documenting infection include culture, cytology, and histopathology. For bacterial sepsis, culture and susceptibility testing is ideal to confirm infection, document the organism(s) involved, and identify antimicrobial drug resistance. However, not all organisms can be grown successfully in the laboratory, and culture might not adequately resolve polymicrobial infections. Molecular techniques including 16S rRNA metagenomics can increase sensitivity and identification of multiple bacterial species but as yet are not readily available to veterinary clinicians. Nonetheless, molecular techniques designed to detect some pathogens are readily available. Point-of-care tests antigen tests, for canine parvovirus for example, often have good specificity, but variable sensitivity. In some studies and in clinical practice, identification of intracellular organisms within samples collected from anatomic locations typically considered to be sterile is diagnostic for infection. For example, the identification of bacteria within phagolysosomes of neutrophils in aseptically collected abdominal fluid samples is diagnostic of septic peritonitis. Less commonly cytologic diagnosis of infection involves identification of intracellular pathogens such as hemotropic pathogens, fungal pathogens, or viral or rickettsial inclusions. However, cytology may have limited sensitivity (i.e., high false negative rates) and visual identification of organisms does not necessarily indicate an infection is active (i.e., identified organisms may be dead). The terms suspected or highly suspected infection are potentially valuable for patient management since they legitimize intensification of diagnostic assessment or

therapeutic intervention where strong clinical suspicion for sepsis exists but where samples cannot readily be obtained or initial testing is pending, negative, or inconclusive. However, from a research perspective, the inclusion of cases with suspected but unconfirmed infection in studies of sepsis might bias results by enrolling animals with less severe disease or by including animals that cannot respond to the treatment under study because they do not have sepsis. As such, variation in the stringency of requirements for documentation or confirmation of infection might lead to, or necessitate, the divergence of sepsis diagnostic criteria for clinical trials compared to clinical practice.

SIRS criteria

Various sets of SIRS criteria have been reported for both dogs and cats with differing sensitivity and specificity. Many of these sets of criteria have been repeatedly used but there is no consensus regarding which set to use. Data from human medicine collected prior to 2016 indicate that the 1991 and 2001 sepsis definitions are highly sensitive, but have poor specificity. A study evaluating the diagnostic accuracy of the 1991 and 2001 definitions found the 1991 sepsis definition to be 95% sensitive and 61% specific, while the 2001 definition was 97% sensitive and 58% specific. Veterinary sepsis definitions derived from these criteria are likely to be similarly affected and legitimate concerns exist regarding construct validity wherein calculations of sensitivity and specificity are predicated on test accuracy that itself may be limited. For instance, in the Hauptman study, the diagnostic criteria for sepsis were evidence of infection and the presence of “systemic illness”, a term that was not further described. Questionable construct validity and lack of consistency within the literature create a tension between the demands of patient management and the requirements of clinical trials that cannot adequately be resolved at present. It has been argued that because the primary aim of the SIRS criteria is to identify animals that are systemically unwell and require prompt attention, the exact cutoff points for these parameters are less important than the implication of the derangement, i.e. that the patient should be thoroughly and urgently assessed. However, a recent study determined that SIRS-positive status was common in small animals presented to the emergency room and primary care, and was only weakly associated with outcome.

Following the publication of Sepsis-3, the human and veterinary definitions are now divergent, further limiting the translation of human sepsis literature to veterinary practice. Moreover, the premise that sepsis is an overexuberant pro-inflammatory response to infection that the underpins use of the SIRS criteria is of questionable validity. Informed by the Sepsis-3 update, the Brazilian Veterinary Emergency and Critical Care Society published consensus definitions for sepsis in 2017, such that there is now geographic variation in the definition of sepsis in veterinary medicine. For all these reasons, it is apparent that a formal redefinition of sepsis in veterinary medicine is warranted to optimally identify sepsis in the clinic and enable future research endeavors.

Ongoing efforts to redefine sepsis

To these ends, we have established a steering committee comprising 12 enthusiastic, engaged sepsis experts and identified a larger group of participants to help accomplish this task. In the first phase, we will define, by consensus, what we consider sepsis “is”; akin to a veterinary dictionary definition. We will independently generate 12 separate definitions before collating and combining these for iterative refinement via an anonymous Delphi survey. Following this, we will perform a systematic review of the

veterinary literature to answer two Population / Exposure / Comparator / Outcome (PECO) format questions. Firstly, we will seek to identify associations between phenotypic factors in animals with infection and negative outcome measures, to identify predictors of sepsis development. Secondly, in animals with sepsis (however defined), we will attempt to find associations with mortality, to identify predictors of sepsis severity or septic shock. A similar approach was successfully used to identify clinical criteria for pediatric sepsis. We will consider how sepsis should be defined from a scientific perspective to enhance the quality, homogeneity, reproducibility, and generalizability of future research and make recommendations about what data should be collected by future veterinary sepsis studies. The third phase of our work will be to evaluate the diagnostic utility and prognostic value of the clinical criteria established during Phase 2 through retrospective medical record review. Subsequently, we intend to launch a sepsis case registry to prospectively gather multicenter data on small animals with sepsis. We envisage this process will be similar to the veterinary committee on trauma (VetCOT) and Reassessment Campaign on Veterinary CPR (RECOVER) registries for trauma and cardiopulmonary resuscitation. Ultimately we aspire to reach a point where the data collected prospectively can be used to refine or replace the consensus definitions we initially establish.

References:

Sharp CR. Systemic Inflammatory Response Syndrome, Sepsis, and Multiple Organ Dysfunction Syndrome. In: Drobatz KJ, Hopper K, Rozanski E, Silverstein DC, eds. Textbook of Small Animal Emergency Medicine, 1st ed. Hoboken, NJ: Wiley Blackwell; 2019:1030-1037. 2

Burkitt JM, Haskins SC, Nelson RW, et al. Relative adrenal insufficiency in dogs with sepsis. J Vet Intern Med. 2007;21:226-231.

Bentley AM, Otto CM, Shofer FS. Comparison of dogs with septic peritonitis: 1988–1993 versus 1999–2003. J Vet Emerg Crit Care. 2007;17:391-398.

DeClue AE, Sharp CR, Harmon M. Plasma inflammatory mediator concentrations at ICU admission in dogs with naturally developing sepsis. J Vet Intern Med. 2012;26:624-630.

Goggs R, Letendre JA. Evaluation of the host cytokine response in dogs with sepsis and noninfectious systemic inflammatory response syndrome. J Vet Emerg Crit Care. 2019;29:593-603.

Summers AM, Vezzi N, Gravelyn T, et al. Clinical features and outcome of septic shock in dogs: 37 Cases (2008-2015). Journal of Veterinary Emergency and Critical Care. 2021;31:360-370.

Stewart EJ. Growing unculturable bacteria. J Bacteriol. 2012;194:4151-4160.

Rutanga JP, Van Puyvelde S, Heroes AS, et al. 16S metagenomics for diagnosis of bloodstream infections: opportunities and pitfalls. Expert Rev Mol Diagn. 2018;18:749-759.

Yang J, Li Z, Liu Y, et al. Tracking bacterial DNA patterns in septic progression using 16s rRNA gene amplicon sequencing analysis. Int J Clin Exp Pathol. 2021;14:753-767.

Walter-Weingärtner J, Bergmann M, Weber K, et al. Comparison of Eight Commercially Available Faecal Point-of-Care Tests for Detection of Canine Parvovirus Antigen. Viruses. 2021;13:2080.

Staatz AJ, Monnet E, Seim HB, 3rd. Open peritoneal drainage versus primary closure for the treatment of septic peritonitis in dogs and cats: 42 cases (1993-1999). Vet Surg. 2002;31:174-180.

Bentley AM, Mayhew PD, Culp WT, et al. Alterations in the hemostatic profiles of dogs with naturally occurring septic peritonitis. J Vet Emerg Crit Care. 2013;23:14-22.

Cortellini S, Pelligand L, Syme H, et al. Neutrophil Gelatinase-Associated Lipocalin in Dogs With Sepsis Undergoing Emergency Laparotomy: A Prospective Case-Control Study. J Vet Intern Med. 2015;29:1595-1602.

Hodgson N, Llewellyn EA, Schaeffer DJ. Utility and Prognostic Significance of Neutrophil-to-Lymphocyte Ratio in Dogs with Septic Peritonitis. Journal of the American Animal Hospital Association. 2018;54:351-359.

Zhao H, Heard SO, Mullen MT, et al. An evaluation of the diagnostic accuracy of the 1991 American College of Chest Physicians/Society of Critical Care Medicine and the 2001 Society of Critical Care Medicine/European Society of Intensive Care Medicine/American College of Chest Physicians/American Thoracic Society/Surgical Infection Society sepsis definition. Crit Care Med. 2012;40:1700-1706.

Spillane AM, Haraschak JL, Gephard SE, et al. Evaluating the clinical utility of the systemic inflammatory response syndrome criteria in dogs and cats presenting to an emergency department. Journal of Veterinary Emergency and Critical Care. 2023;33:315-326.

Castro BGA, Rabelo RC. Sepsis-3: Uma Análise Aplicada à Medicina Veterinária. Journal LAVECC. 2017;9:1-12.

Goldstein B, Giroir B, Randolph A. International pediatric sepsis consensus conference: definitions for sepsis and organ dysfunction in pediatrics. Pediatr Crit Care Med. 2005;6:2-8.

Singer M, Deutschman CS, Seymour CW, et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016;315:801-810.

Shankar-Hari M, Phillips GS, Levy ML, et al. Developing a New Definition and Assessing New Clinical Criteria for Septic Shock: For the Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016;315:775-787.

Menon K, Schlapbach LJ, Akech S, et al. Criteria for Pediatric Sepsis-A Systematic Review and Meta-Analysis by the Pediatric Sepsis Definition Taskforce. Crit Care Med. 2022;50:21-36.

Menon K, Schlapbach LJ, Akech S, et al. Pediatric Sepsis Definition-A Systematic Review Protocol by the Pediatric Sepsis Definition Taskforce. Crit Care Explor. 2020;2:e0123.

Hall KE, Rutten JI, Baird TN, et al. ACVECC-Veterinary Committee on Trauma registry report 2017-2019. J Vet Emerg Crit Care (San Antonio). 2023;33:289-297.

Hoehne SN, Balakrishnan A, Silverstein DC, et al. Reassessment Campaign on Veterinary Resuscitation (RECOVER) Initiative small animal CPR registry report 2016-2021. J Vet Emerg Crit Care (San Antonio). 2023;33:143-155.

IMMUNOTHROMBOSIS: A NOVEL THERAPEUTIC TARGET?

Robert Goggs [1]

[1] Cornell University College of Veterinary Medicine, Clinical Sciences, Ithaca, United States

Learning objectives:

- Describe what NETs are and how they are formed.

- Understand the potential benefits and risks of NETs as host defence mechanisms.

- Understand what the term immunothrombosis means.

- State how immunothrombosis can be recognized and NETs measured.

- Know how immunothrombosis might be targeted with future therapeutics.

Proceeding:

Introduction

Sepsis is a common clinical syndrome in small animals that is associated with substantial morbidity and a high risk of death, with reported mortality rates in dogs of 20-68%. Sepsis is defined as the dysregulated host response to infection that causes organ dysfunction, including coagulation disorders. Sepsis is associated with development of a procoagulant state, that can manifest as disseminated intravascular coagulation, and clinical thrombosis. In small animals, sepsis is recognized clinically by documenting the hallmarks of systemic inflammation (fever, tachycardia, tachypnea, and leukocytosis) and a concurrent infection. Commonly used SIRS criteria are sensitive,[2] but lack specificity for sepsis diagnosis and have limited prognostic utility. Biomarkers such as acute phase proteins, and procalcitonin, have been widely evaluated in sepsis to aid diagnosis and prognostication and enable clinicians to individualize care, but all available biomarkers have limitations. Recent discoveries in leukocyte biology suggest biomarkers of neutrophil extracellular trap (NET) formation might be useful clinically in sepsis, and technological advancements mean that high-throughput screening tools including proteomics and metabolomics can now be applied to veterinary patients. Personalized medicine for dogs and cats with sepsis may yet be within reach.

Immunothrombosis

Neutrophils are the principal effector cells of the innate immune response to bacterial infection. Phagocytosis and degranulation are well-characterized responses to bacterial pathogens, but neutrophils can also release extracellular traps, a process termed NETosis. NETs are extracellular DNA decorated with citrullinated histones and antimicrobial proteins including myeloperoxidase (MPO) and neutrophil elastase (NE).[29] Microbial pathogen-associated molecular patterns (PAMPs) and recognition of damage

associated molecular patterns (DAMPs) can trigger NETosis, which involves intracellular signaling, chromatin decondensation, nuclear envelope disintegration, and controlled extracellular release of DNA and granule contents. Intravascularly, NETs trap *E. coli*, facilitating bacterial clearance, and may also engage in direct microbial killing potentially via the MPO, NE, and cathepsin G on the surface of extruded DNA. Neutrophil activation and NETosis is facilitated by platelets, through binding of platelet P-selectin to neutrophil P-selectin glycoprotein ligand-1.

Current evidence suggests that NETs are an essential component of innate immunity but the role of NETs in host defense is complex. Engelmann and Massberg coined the term 'immunothrombosis' in 2013, defined as an innate immune response where local activation of coagulation facilitates the recognition, containment, and destruction of microbial pathogens. Treatment of *Staphylococcus aureus* skin infections with DNase, an enzyme that digests DNA, led to systemic bacterial dissemination in mice, and early DNAase treatment of septic mice led to a deterioration in the condition. Excessive NETosis, especially during sepsis, can also be detrimental to the host, however. While NETs may be protective when constrained and targeted, dysregulated, widespread or excessive NETosis can lead to thrombosis, microvascular dysfunction, tissue necrosis, multiple organ failure, disseminated intravascular coagulation and death. This thrombosis potentiation has also been described in dogs. In some mouse sepsis models, DNAase treatment attenuates sepsis-induced organ dysfunction and reduces mortality.

Various NET constituents can initiate thrombosis. Through intravascular expression of a polyanionic surface NETs can activate the contact pathway. Extracellular DNA and RNA have high negative charge densities that activate factors XI and XII. Extracellular DNA also binds high molecular weight kininogen, a cofactor for kallikrein activation. Factor colocalization facilitated by extracellular DNA accelerates factor activation. In addition to contact pathway activation, NETs play other prothrombotic roles. NETs are decorated with NE, an enzyme with microbicidal effects, that can also inactivate tissue factor pathway inhibitor (TFPI). This inhibitory effect may tip the hemostatic balance in favor of local thrombin formation. In the context of NETs, the inhibitory effect of NE on TFPI is dependent on the polyanionic surface charge of cfDNA.

Extracellular histones released during NETosis facilitate thrombus formation in both platelet-dependent and independent manners, and impair clot dissolution by inhibiting fibrinolysis. Citrullination of histone H3 by peptidylarginine deminase-4 (PAD-4) leads to chromatin decondensation, a process that precedes NETosis. Systemic treatment of mice with PAD inhibitors improve survival in sepsis models. Extracellular histones released during NETosis can also be scavenged by heparin. This is noteworthy, because treatment with heparin improves survival in mouse sepsis models, while individualized treatment of dogs with unfractionated heparin may improve survival in IMHA.

Fibrinolyis is also affected by NETs. Intercalation of NETs into a fibrin clot produces a network that is more resistant to breakdown. The effect of cfDNA on fibrinolysis is complex and may be concentration dependent. At low concentrations, cfDNA potentiates the activation of plasminogen by tissue plasminogen activator (tPA), accelerating fibrinolysis. At these concentrations cfDNA can also suppress fibrinolysis by enhancing plasminogen activator inhibitor-1 mediated tPA inhibition. At higher concentrations, however, cfDNA decreases the rate of fibrinolysis by acting as a competitive plasmin inhibitor.

References:

King LG. Postoperative complications and prognostic indicators in dogs and cats with septic peritonitis: 23 cases (1989-1992). J Am Vet Med Assoc. 1994;204:407-414.

Hauptman JG, Walshaw R, Olivier NB. Evaluation of the sensitivity and specificity of diagnostic criteria for sepsis in dogs. Vet Surg. 1997;26:393-397.

de Laforcade AM, Freeman LM, Shaw SP, et al. Hemostatic changes in dogs with naturally occurring sepsis. J Vet Intern Med. 2003;17:674-679.

Kenney EM, Rozanski EA, Rush JE, et al. Association between outcome and organ system dysfunction in dogs with sepsis: 114 cases (2003-2007). J Am Vet Med Assoc. 2010;236:83-87.

Singer M, Deutschman CS, Seymour CW, et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016;315:801-810.

de Laforcade AM, Rozanski EA, Freeman LM, et al. Serial evaluation of protein C and antithrombin in dogs with sepsis. J Vet Intern Med. 2008;22:26-30.

Kuzi S, Segev G, Haruvi E, et al. Plasma antithrombin activity as a diagnostic and prognostic indicator in dogs: a retrospective study of 149 dogs. J Vet Intern Med. 2010;24:587-596. Kim SD, Baker P, DeLay J, et al. Thrombomodulin Expression in Tissues From Dogs With Systemic Inflammatory Disease. Vet Pathol. 2016;53:797-802.

Goggs R, Mastrocco A, Brooks MB. Retrospective evaluation of 4 methods for outcome prediction in overt disseminated intravascular coagulation in dogs (2009-2014): 804 cases. J Vet Emerg Crit Care. 2018;28:541-550.

LaRue MJ, Murtaugh RJ. Pulmonary thromboembolism in dogs: 47 cases (1986-1987). J Am Vet Med Assoc. 1990;197:1368-1372.

Palmer KG, King LG, Van Winkle TJ. Clinical manifestations and associated disease syndromes in dogs with cranial vena cava thrombosis: 17 cases (1989-1996). J Am Vet Med Assoc. 1998;213:220-224.

Respess M, O'Toole TE, Taeymans O, et al. Portal vein thrombosis in 33 dogs: 1998-2011. J Vet Intern Med. 2012;26:230-237.

Okano S, Yoshida M, Fukushima U, et al. Usefulness of systemic inflammatory response syndrome criteria as an index for prognosis judgement. Vet Rec. 2002;150:245-246.

Jitpean S, Pettersson A, Hoglund OV, et al. Increased concentrations of Serum amyloid A in dogs with sepsis caused by pyometra. BMC Vet Res. 2014;10:273.

Gebhardt C, Hirschberger J, Rau S, et al. Use of C-reactive protein to predict outcome in dogs with systemic inflammatory response syndrome or sepsis. J Vet Emerg Crit Care (San Antonio). 2009;19:450-458.

Goggs R, Milloway M, Troia R, et al. Plasma procalcitonin concentrations are increased in dogs with sepsis. Vet Rec Open. 2018;5:e000255.

Troia R, Giunti M, Goggs R. Plasma procalcitonin concentrations predict organ dysfunction and outcome in dogs with sepsis. BMC Vet Res. 2018;14:111.

Pierrakos C, Vincent JL. Sepsis biomarkers: a review. Crit Care. 2010;14:R15.

Pierrakos C, Velissaris D, Bisdorff M, et al. Biomarkers of sepsis: time for a reappraisal. Crit Care. 2020;24:287.

Letendre JA, Goggs R. Measurement of plasma cell-free DNA concentrations in dogs with sepsis, trauma, and neoplasia. J Vet Emerg Crit Care (San Antonio). 2017;27:307-314.

Jeffery U, Kimura K, Gray R, et al. Dogs cast NETs too: Canine neutrophil extracellular traps in health and immune-mediated hemolytic anemia. Vet Immunol Immunopathol. 2015;168:262-268.

Hatada T, Wada H, Nobori T, et al. Plasma concentrations and importance of High Mobility Group Box protein in the prognosis of organ failure in patients with disseminated intravascular coagulation. Thromb Haemost. 2005;94:975-979.

Kuleš J, Horvatić A, Guillemin N, et al. The plasma proteome and the acute phase protein response in canine pyometra. J Proteomics. 2020;223:103817. Franco-Martínez L, Horvatić A, Gelemanović A, et al. Changes in the Salivary Proteome Associated With Canine Pyometra. Front Vet Sci. 2020;7:277.

Průcha M, Zazula R, Russwurm S. Sepsis Diagnostics in the Era of "Omics" Technologies. Prague Med Rep. 2018;119:9-29. Eckerle M, Ambroggio L, Puskarich MA, et al. Metabolomics as a Driver in Advancing Precision Medicine in Sepsis. Pharmacotherapy. 2017;37:1023-1032.

Mangioni D, Peri AM, Rossolini GM, et al. Toward Rapid Sepsis Diagnosis and Patient Stratification: What's New From Microbiology and Omics Science. J Infect Dis. 2020;221:1039-1047.

Davenport EE, Burnham KL, Radhakrishnan J, et al. Genomic landscape of the individual host response and outcomes in sepsis: a prospective cohort study. Lancet Respir Med. 2016;4:259-271.

Brinkmann V, Reichard U, Goosmann C, et al. Neutrophil extracellular traps kill bacteria. Science. 2004;303:1532-1535.

Steinberg BE, Grinstein S. Unconventional roles of the NADPH oxidase: signaling, ion homeostasis, and cell death. Sci STKE. 2007;2007:pe11.

Kaplan MJ, Radic M. Neutrophil extracellular traps: double-edged swords of innate immunity. J Immunol. 2012;189:2689-2695.

Tadie JM, Bae HB, Jiang S, et al. HMGB1 promotes neutrophil extracellular trap formation through interactions with Toll-like receptor 4. Am J Physiol Lung Cell Mol Physiol. 2013;304:L342-349.

Maugeri N, Campana L, Gavina M, et al. Activated platelets present high mobility group box 1 to neutrophils, inducing autophagy and promoting the extrusion of neutrophil extracellular traps. J Thromb Haemost. 2014;12:2074-2088.

Mitroulis I, Kambas K, Chrysanthopoulou A, et al. Neutrophil extracellular trap formation is associated with IL-1beta and autophagy-related signaling in gout. PLoS One. 2011;6:e29318.

Schauer C, Janko C, Munoz LE, et al. Aggregated neutrophil extracellular traps limit inflammation by degrading cytokines and chemokines. Nat Med. 2014;20:511-517. 36. Pisetsky DS. The origin and properties of extracellular DNA: from PAMP to DAMP. Clin Immunol. 2012;144:32-40.

Fuchs TA, Abed U, Goosmann C, et al. Novel cell death program leads to neutrophil extracellular traps. J Cell Biol. 2007;176:231-241.

McDonald B, Urrutia R, Yipp BG, et al. Intravascular neutrophil extracellular traps capture bacteria from the bloodstream during sepsis. Cell Host Microbe. 2012;12:324-333.

Egesten A, Breton-Gorius J, Guichard J, et al. The heterogeneity of azurophil granules in neutrophil promyelocytes: immunogold localization of myeloperoxidase, cathepsin G, elastase, proteinase 3, and bactericidal/permeability increasing protein. Blood. 1994;83:2985-2994.

Weinrauch Y, Drujan D, Shapiro SD, et al. Neutrophil elastase targets virulence factors of enterobacteria. Nature. 2002;417:91-94.

Medina E. Murine model of polymicrobial septic peritonitis using cecal ligation and puncture (CLP). Methods Mol Biol. 2010;602:411-415.

Gould TJ, Vu TT, Swystun LL, et al. Neutrophil extracellular traps promote thrombin generation through platelet-dependent and platelet-independent mechanisms. Arterioscler Thromb Vasc Biol. 2014;34:1977-1984.

Clark SR, Ma AC, Tavener SA, et al. Platelet TLR4 activates neutrophil extracellular traps to ensnare bacteria in septic blood. Nat Med. 2007;13:463-469.

Etulain J, Martinod K, Wong SL, et al. P-selectin promotes neutrophil extracellular trap formation in mice. Blood. 2015;126:242-246.

Engelmann B, Massberg S. Thrombosis as an intravascular effector of innate immunity. Nat Rev Immunol. 2013;13:34-45.

Yipp BG, Kubes P. NETosis: how vital is it? Blood. 2013;122:2784-2794.

Mai SH, Khan M, Dwivedi DJ, et al. Delayed but not Early Treatment with DNase Reduces Organ Damage and Improves Outcome in a Murine Model of Sepsis. Shock. 2015;44:166-172.

Fourrier F, Chopin C, Goudemand J, et al. Septic shock, multiple organ failure, and disseminated intravascular coagulation. Compared patterns of antithrombin III, protein C, and protein S deficiencies. Chest. 1992;101:816-823.

Gould TJ, Lysov Z, Liaw PC. Extracellular DNA and histones: double-edged swords in immunothrombosis. J Thromb Haemost. 2015;13 Suppl 1:S82-91.

Jeffery U, LeVine DN. Canine Neutrophil Extracellular Traps Enhance Clot Formation and Delay Lysis. Vet Pathol. 2017;55:116-123.

Czaikoski PG, Mota JM, Nascimento DC, et al. Neutrophil Extracellular Traps Induce Organ Damage during Experimental and Clinical Sepsis. PLoS One. 2016;11:e0148142.

Martinod K, Wagner DD. Thrombosis: tangled up in NETs. Blood. 2014;123:2768-2776.

Liaw PC, Ito T, Iba T, et al. DAMP and DIC: The role of extracellular DNA and DNA-binding proteins in the pathogenesis of DIC. Blood Rev. 2015.

Morrissey JH, Choi SH, Smith SA. Polyphosphate: an ancient molecule that links platelets, coagulation, and inflammation. Blood. 2012;119:5972-5979.

Kannemeier C, Shibamiya A, Nakazawa F, et al. Extracellular RNA constitutes a natural procoagulant cofactor in blood coagulation. Proc Natl Acad Sci U S A. 2007;104:6388-6393.

Swystun LL, Mukherjee S, Liaw PC. Breast cancer chemotherapy induces the release of cell-free DNA, a novel procoagulant stimulus. J Thromb Haemost. 2011;9:2313-2321.

Oehmcke S, Morgelin M, Herwald H. Activation of the human contact system on neutrophil extracellular traps. J Innate Immun. 2009;1:225-230.

Massberg S, Grahl L, von Bruehl ML, et al. Reciprocal coupling of coagulation and innate immunity via neutrophil serine proteases. Nat Med. 2010;16:887-896.

Schulz C, Engelmann B, Massberg S. Crossroads of coagulation and innate immunity: the case of deep vein thrombosis. J Thromb Haemost. 2013;11 Suppl 1:233-241.

Li Y, Liu Z, Liu B, et al. Citrullinated histone H3: a novel target for the treatment of sepsis. Surgery. 2014;156:229-234.

Wildhagen KC, Garcia de Frutos P, Reutelingsperger CP, et al. Nonanticoagulant heparin prevents histone-mediated cytotoxicity in vitro and improves survival in sepsis. Blood. 2014;123:1098-1101.

Helmond SE, Polzin DJ, Armstrong PJ, et al. Treatment of immune-mediated hemolytic anemia with individually adjusted heparin dosing in dogs. J Vet Intern Med. 2010;24:597-605.

Fuchs TA, Brill A, Duerschmied D, et al. Extracellular DNA traps promote thrombosis. Proc Natl Acad Sci U S A. 2010;107:15880-15885.

Komissarov AA, Florova G, Idell S. Effects of extracellular DNA on plasminogen activation and fibrinolysis. J Biol Chem. 2011;286:41949-41962.

Esmon CT. Molecular circuits in thrombosis and inflammation. Thromb Haemost. 2013;109:416-420.

SEPTIC SHOCK- DECISION MAKING FROM THE TRENCHES

Anusha Balakrishnan [1]

[1] Veterinary Emergency Group, Veterinary Emergency Group, White Plains, United States

Learning objectives:

- This will be a case-based session that pulls in real-life cases seen in the emergency setting with a specific focus on clinical decision making in the areas of diagnosis sepsis, source control (specifically with regards to surgical decision-making), antimicrobial choices given real-world limitations as well as vasopressor choices.

- The cases will invite discussion surrounding ideal decisions based on current evidence as well as necessary compromises given common limitations in the ER surrounding owner financial resources, drug shortages and other limitations.

Proceeding:

This session will be a case-based panel discussion reviewing clinical decision making in septic patients. The cases will focus on decision making surrounding initial shock resuscitation, antimicrobial choices, vasopressor therapy and considerations for diagnosing and treating CIRCI (Critical Illness Related Corticosteroid Insufficiency).

Sepsis and Septic Shock in Dogs and Cats

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection.

Septic shock is defined as sepsis with hypotension that is not responsive to fluid resuscitation. This is a subset of sepsis in which underlying circulatory and cellular/metabolic abnormalities are profound enough to substantially increase mortality.

Severe sepsis and septic shock tend to have extremely high mortality rates, estimated to be approximately 30% in people with severe sepsis and up to 50% of those in septic shock. Mortality in dogs is also approximately 50%, with increasing mortality rates for dogs with MODS.

One of the most important aspects of managing severe sepsis and septic shock in veterinary patients is early recognition of sepsis. This can be challenging in some patients, especially cats, where the source of sepsis may be difficult to identify.
Metabolic derangement in sepsis can include hypovolemia, decreased vasomotor tone, decreased arterial oxygen content, myocardial depression, increased metabolic demands, and impairment of systemic oxygen utilization via microcirculatory or mitochondria.

Physical examination signs of sepsis are varied. Septic shock results in a maldistributive type of shock characterized by systemic, inappropriate vasodilation. Vasodilation is caused by massive inflammatory mediator-induced systemic vasodilation and myocardial dysfunction. When evaluating patients presenting to an emergency room, attention must be paid to possible early signs of sepsis. In dogs, early signs of sepsis on physical exam include tachycardia, bounding pulses, rapid capillary refill time, injected mucous membranes (secondary to systemic vasodilation), fever, and decreased blood glucose levels. Tachycardia and an increase in cardiac contractility occur in response to tissue hypoxia. Bounding pulses result from the increase in cardiac output coupled with the systemic vasodilation. Fever is induced by the effects of inflammatory mediators on the thermoregulatory centers in the brain. Cats do not tend to develop injected mucous membranes, bounding pulses, or sometimes even tachycardia. Septic cats will often present with signs of lethargy, pale mucous membranes, tachypnea, weak pulses, hypotension, hypothermia, or icterus. Cats may present with tachycardia or a relative bradycardia of 100-140 beats per minute.

Signs of late sepsis include hypoglycemia, weak and thready pulses, prolonged capillary refill time, pale mucous membranes, cool extremities, stupor, hypothermia, and multiple organ failure.

Initial Stabilization of the Septic Shock Patient

Early Goal Directed Therapy: Septic shock resuscitation in human medicine has changed dramatically in the past 15 years after the publication of a landmark study by Rivers, et al which showed a marked reduction in mortality rate for patients treated early goal directed therapy during the first 6 hours in the emergency department. The resuscitation targets in these patients included a targeted mean arterial blood pressure of >65 mm Hg to ensure vital organ perfusion.

Typically, in a hypotensive patient with elevated lactate levels and hypotension, volume resuscitation can be initiated with the use of isotonic crystalloids. Balanced electrolyte solutions are often the first choice in most veterinary hospitals. A typical starting point for a septic patient would be between 10-30 mL/kg bolus of an isotonic crystalloid.

Goal directed therapy has been explored in a study in septic dogs with pyometra.

Antibiotic administration in sepsis

A retrospective study in adult humans with septic shock showed an 8.5 % increase in mortality for a 6-h delay or a 7.6 % increase in mortality (septic shock) for each hour of delay from the time of diagnosis to antibiotic therapy. A similar finding has not been definitively documented in dogs and cats; however, early, appropriate antimicrobial therapy remains a cornerstone of sepsis management.

Some fundamental points regarding antibiotic choices in these patients include:

Broad spectrum coverage

Escalation vs de-escalation approach

Tissue penetration

Knowledge of some of the common organisms associated with various sources of infection.

Antimicrobial pharmacokinetics

Ultimately, culture and sensitivity samples should be obtained, when possible, prior to antibiotic therapy to better guide de-escalation or other changes in antibiotic therapy. However, it is important to remember that obtaining cultures should not delay administration of antibiotics promptly.

Vasopressor therapy in septic shock

Despite adequate fluid administration, vasopressor agents are often required to correct hypotension, given the inappropriate vasodilation that occurs with sepsis. While vasopressors should be promptly begun in patients in persistent septic shock despite fluid resuscitation; they can also be begun and continued simultaneously with fluid resuscitation, especially in patients with severe hypotension suspected to be from septic shock. No study to date in small animals has demonstrated a statistically significant survival benefit of one vasopressor over another. Therefore, the choice of vasopressor in septic shock is rather empiric.

The guidelines below are based on the human recommendations in the latest Surviving Sepsis guidelines.

Vasopressors should be begun initially to target a mean arterial pressure of 65 mm Hg.

Norepinephrine which is a predominantly alpha-adrenergic agonist should be provided as the first-line vasopressor. Norepinephrine can be administered to dogs and cats at a starting dose of 0.05 mcg/kg/minute. The patient's blood pressure should be assessed every 5-10 minutes, and the norepinephrine is gradually titrated up in increments of 0.05-0.1 mcg/kg minute to maintain a sufficient mean arterial pressure. At doses of over 0.5-0.6 mcg/kg/minute, significant splanchnic vasoconstriction can occur, and the beneficial effect with peripheral vasoconstriction is usually outweighed by potential adverse effects. Generally, a second vasopressor is added at this point.

Vasopressin is appropriate to use with norepinephrine, either to improve perfusion (increase MAP) or to reduce the required dose of norepinephrine. Vasopressin is not recommended for use as a single vasopressor for septic shock. Vasopressin infusion in dogs and cats can be started at the rate of 0.5-1 mU/kg/minute and titrated up in increments of 0.5 mU/kg/minute. Doses of higher that 5-6 mU/kg/minute are recommended to be reserved only for dire situations of septic shock refractory to standard doses of multiple vasopressors.

Epinephrine is recommended in patients in septic shock with refractory hypotension with evidence of cardiac dysfunction. In patients that are not responsive to norepinephrine and vasopressin, either consider the addition of dobutamine to these two vasopressors or consider transitioning instead to epinephrine. The beta-adrenergic effects of epinephrine may afford some positive inotropy in patients that may be experiencing myocardial dysfunction from sepsis.

Dopamine is not recommended as an alternative to norepinephrine in septic shock, except in highly selected patients such as those with inappropriately low heart rates (absolute or relative bradycardia) who are at low risk for tachyarrhythmias. Dopamine is recommended to not be used in low doses in a so-called renal-protective strategy.

Dobutamine, a catecholamine with predominantly beta-1 adrenergic receptor function is useful in patients with evidence of myocardial dysfunction secondary to sepsis and resultant hypotension. A dobutamine infusion at doses ranging from 5-15 mcg/kg/min can be added to any vasopressors in use. Dobutamine should be used with caution in cats as it can cause seizure activity. Lower doses should be used in cats (1-5 mcg/kg/minute).

Steroids in Sepsis

CIRCI is defined as an insufficient cortisol response or inadequate cortisol activity for the existing degree of critical illness. Although serum cortisol concentrations in patients with CIRCI often are increased relative to resting concentrations in healthy individuals, the cortisol response remains insufficient for the markedly increased demands of acute, severe illness.

Critical illness-related corticosteroid insufficiency may be due to hypothalamic-pituitary-adrenal (HPA) axis dysfunction, alterations in cortisol-plasma protein binding, target cell enzymatic changes, changes in glucocorticoid receptor (GR) function, or a combination of these or other factors present during critical illness.

CIRCI has been documented in both dogs and cats in a few veterinary studies.

The definitive diagnosis of CIRCI is typically made through adrenal function testing. However, recent human guidelines no longer recommend routine testing in these patients and instead recommend instituting treatment if CIRCI is suspected.

Therapy for CIRCI should be instituted ONLY in the critically ill patients with septic shock that is persistently hypotensive despite adequate fluid therapy, and if the hypotension is refractory to vasopressor therapy (continued norepinephrine need at doses > 0.25 mcg/kg/min for >4 hours, based on the 2021 Surviving Sepsis Guidelines).

The drug of choice to treat CIRCI is hydrocortisone. Dexamethasone may be used but is less preferred. Hydrocortisone can be started as a constant rate infusion of 2.5-3 mg/kg/day or administered intermittently at a dose of 1 mg/kg IV q8. Generally, steroid therapy is only continued after 24 hours if the patient shows significant improvement in cardiovascular status following initiation of this drug. A recent study evaluating hydrocortisone for treating CIRCI in dogs with septic peritonitis showed no significant difference in survival between the hydrocortisone-treated and non-treated septic shock patients. Further studies are needed to evaluate the use of HC in patients with suspected CIRCI.

References:

Singer, Mervyn, et al. "The third international consensus definitions for sepsis and septic shock (Sepsis-3)." Jama 315.8 (2016): 801-810.

Evans, Laura, et al. "Surviving sepsis campaign: international guidelines for management of sepsis and septic shock 2021." Critical care medicine 49.11 (2021): e1063-e1143.

Summers, April M., et al. "Clinical features and outcome of septic shock in dogs: 37 Cases (2008-2015)." Journal of Veterinary Emergency and Critical Care 31.3 (2021): 360-370

Shipov, Anna, et al. "Aetiology, clinical parameters and outcome in 113 dogs surgically treated for septic peritonitis (2004–2020)." Veterinary Record 192.6 (2023): no-no

Summers, April M., et al. "Retrospective evaluation of the use of hydrocortisone for treatment of suspected critical illness–related corticosteroid insufficiency (CIRCI) in dogs with septic shock (2010–2017): 47 cases." Journal of Veterinary Emergency and Critical Care 31.3 (2021): 371-379.

Nurse & Tech Stream, Friday 31 May 2024

MANAGEMENT OF HOSPITALISED INFECTIOUS FELINE PATIENTS

Nathalie Hack [1]

[1] Vets Now, Dunfermline, United Kingdom

Learning objectives:

- Management of hospitalised infectious Feline patients: goals

- During this lecture an overview of the most common feline infectious diseases seen in practice will be given that might require hospitalisation.

- Nursing management, general nursing, isolation and barrier nursing, fluid therapy, nebulisation, nutrition and medication for these cases will be discussed as well as how to control and prevent these infectious diseases.

Proceeding:

Most common feline infectious diseases that might require hospitalisation:

1. Cat flu (Feline Infectious Upper Respiratory Tract disease or URT)

• Feline Herpes virus (FHV)

• Feline Calici virus (FCV)

2. Chronic rhinosinusitis (CR)

3. Feline infectious Peritonitis (FIP)

1. Cat flu

• Feline Infectious Upper Respiratory Tract disease (URT)

• Associated with several infectious agents which can cause similar signs

• Feline herpes virus (FHV) and feline calicivirus (FCV) are most commonly involved

• Other agents are Chlamydophila Felis, Mycoplasma and Bordetella bronchiseptica

• Acute infectious URT is most severe in kittens and immunosuppressed adults

• Signs:

Anorexia

Fever

Sneezing

Lethargy

Oculonasal discharge

Oral ulceration

Conjunctivitis

Ptyalism

• Feline herpes virus (FHV)

Severe oculonasal signs

Dendritic keratitis

• Feline Calicivirus (FCV)

Lingual and oral ulcers

Ulceration of the nasal planum

2. Chronic Rhinosinusitis (CR)

• Idiopathic

• Can occur after severe FHV infection

• Chronic history (more than 1 month) of sneezing and bilateral discharge

3. Feline Infectious Peritonitis (FIP)

• Caused by Feline Coronavirus

• Most infected cats show no clinical signs

• A stressor such as rehoming/neutering can be the onset of clinical signs

• Signs:

• Fluctuating anorexia

• Lethargy

• Pyrexia

• Pale/jaundiced mucous membranes

• Failure to grow/weight loss

• Effusive FIP

• Distended abdomen caused by large volume of peritoneal effusion

• White fibrinous deposits on the spleen and liver

• Atypical signs:

• Ventral oedema of the chin

• Scrotal enlargement

• Pericardial infusion

• Non-effusive FIP

• Ocular signs are common

• Followed by abdominal and renal signs

General nursing

• Supportive treatment is essential to ensure a positive outcome

• Spending time with them!

• Administering medication

• Checking the usual parameters: temperature, pulse, heart rate, mucous membranes and capillary refill time

• Cleaning oculonasal discharge with warmed saline and cotton wool

• Wiping the coat with a warm damp cloth once or twice daily

Isolation and barrier nursing

• To prevent the spread of infection to other hospitalised patients

• The isolated cat should have its own equipment and utensils like stethoscope, thermometer, food bowls, litter trays, …

• Steel bowls are useful as they can be autoclaved

• Once the cat is discharged, all the surfaces and bedding should be cleaned with the appropriate disinfectant: diluted hypochlorite/detergent mixture for the viral causes of URT

• Barrier nursing protocols include wearing PPE (Personal Protective Equipment):

• Gloves

• Apron

• Mask/visor

• Shoe protector covers

• Good hand hygiene

Fluid therapy

• To restore fluid and electrolyte/acid-base balance

• Can be given

• Intravenously

• Subcutaneous

• Intra-osseous

• Orally

• Initially a bolus is given, followed by maintenance fluids

Nebulisation

• Saline nebulisation is useful for maintaining airway hydration

• It helps to break down mucus within the upper respiratory tract

• The nebuliser should be near the cat's face for 5-10 min, some patients tolerate this better than others

• At home, steam therapy can be used. The cat is placed within a steamy room (usually the bathroom) with a hot tap or shower running.

Nutrition

• Patients should be offered palatable, blended and warmed food

• do not syringe feed

• If anorexia persists, or the patient is unable to eat due to mouth ulcers (Calicivirus) consider placing either a

• naso-oesophageal (NO)

• oesophagostomy tube (O) for patients that are likely to require feeding for a longer period of time

What is a NO tube?

NO stands for Naso-Oesophageal

Provides short term enteral nutritional support, up to 5 to 10 days

Ideal for patients where sedation or anaesthetic is contra-indicated

Advantages of a NO tube

Generally, well tolerated by the patient

Non-invasive

Does not prevent the animal from eating and drinking

Easy to place with minimal chemical restraint and topical local anaesthetic

Suitable for patients that are considered too ill to receive a sedation or general anaesthesia

These tubes can be managed by owners at home after some training

Disadvantages of a NO tube

Easily dislodged, so a buster collar is a must

Not suitable for patients that are unconscious, vomiting, have a poor gag-reflex or have a megaoesophagus

May be vomited up

Can only be used and left in situ for up to 10 days

The bore of the tube is small, so only certain diets can be used

Complications that might arise are rhinitis, tracheal intubation and epistaxis

NO tube material

• Most of the feeding tubes come into a variety of sizes, lengths and materials

(consisting out of red rubber, PVC (polyvinyl chloride), polyurethane or silicone))

NO tube size

• Size used Cats : 5-6 Fr

• The French Gauge or French scale is used to measure the diameter of feeding tubes. It is most often abbreviated as Fr, but can often be seen abbreviated as Fg, Ga, FR or F. It may also be abbreviated as CH or Ch (for Charrière, its inventor).

• The French size is three times the diameter in millimetres.

• D (mm) = Fr / 3 or Fr = D (mm) x 3

• So a 12 Fr feeding tube has a diameter of 4mm (12/3= 4)

What food to give?

• Consistency of most diets is too dense. Watering it down will result in large amounts to be given

• Special commercial diets available with the right consistency and only 1kcal/ml.

Medication

• Several drugs can be used in the treatment of feline infectious diseases:

• Antivirals (for FHV)

• Broad spectrum antibiotics for secondary bacterial infections

• Appetite stimulants: i.e. mirtazapine

• Analgesia: NSAIDS and/or opioids

• Mucolytic drug for relief of nasal congestion

• Eye drops

• Using GS441, polyprenyl and interferon for treatment of FIP!

Control and prevention.

• Prevention is better than cure!

• Vaccination will help to reduce the risk of a cat acquiring the disease, but will not affect the carrier status of an already infected animal

• New arrivals in shelters should be quarantined separately (unless from the same household) for 14 days

• Good cleaning and disinfection protocols in place

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

THE TECHNICIAN'S GUIDE TO BLOCKED CATS

Angela Elia [1]

[1] Pieper Veterinary Group, Pieper Veterinary Group, Middletown, CT, United States

Learning objectives:

List and describe the main causes of Urethral Obstruction in felines

Identify common presenting problems and lab work abnormalities seen with urethral obstruction

Identify complications of urinary obstructions and their treatments

Describe the proper way to care for indwelling urinary catheters

Proceeding:

Feline Lower Urinary Tract Disease (FLUTD)

Now more commonly being referred to as Feline Idiopathic Cystitis (FIC)

Two-thirds of cases have an unknown origin.

More common in indoor-only cats, the obese, and cats on high Magnesium diets

Can be linked to environmental stress factors such as:

Symptoms of FLUTD/FIC

Excessive genital grooming

Dysuria

Inappropriate urination

Hematuria

Diagnostics and treatment for FLUTD/FIC

Radiographs

Ultrasound

Urinalysis

Blood work

Antibiotics (dependent upon urinalysis results)

Special Urinary Diet

Gabapentin

Buprenorphine

How does FLUTD/FIC lead to a urethral obstruction?

Inflammation of the bladder wall can change the pH of urine. This can make the urine more prone to the formation of crystals, which, in turn can cause more inflammation and further pH changes.

The presence of crystals may be caused by other conditions (diet) and cause inflammation that causes pH changes.

These conditions can lead to urethral obstruction by the creation of mucous plugs filled with inflammatory cells and crystals getting trapped in the urethra.

What causes Urethral Obstructions?

Male cats have a narrow urethral lumen making them prone to blocking.

Urethral plugs are most commonly made of a proteinaceous matrix with struvite crystals embedded in them.

Calcium oxalate urethroliths, strictures and neoplasia can also be causes of urethral obstructions.

Strictures can be secondary to trauma or congenital abnormalities. These patients may need surgical intervention as part of their treatment plan.

The urethral obstruction patient on physical examination:

The bladder may be large or small but will be firm and unable to express with gentle pressure.

Abdominal pain on palpation

Dehydration

Bradycardia

Lethargy

Hypothermia

What abnormalities are seen in bloodwork?

Azotemia

Hyperkalemia

Metabolic acidosis

Treatment of Hyperkalemia in the blocked cat:

The only true cure is to unblock the cat.

Calcium Gluconate 0.5 – 1.0 ml/kg given over 10 minutes. This will protect the heart from effects of hyperkalemia but will only last 10-15 minutes.

You must watch for ECG abnormalities during administration.

Insulin 0.1-0.25 units/kg with a 0.25-0.5 g/kg IV dextrose bolus

IV dextrose should be diluted 1:2 before being given.

Dextrose supplementation at 2.5-5% may be necessary to prevent hypoglycemia.

Sodium Bicarb: 1 meq/kg IV slowly will also shift K+ intracellularly by increasing serum pH.

Anesthesia and analgesia for the blocked cat

Reflects the stability of the patient.

Some procedures may be difficult/unsuccessful, so be prepared for general anesthesia.

Pain management:

Buprenorphine

Methadone

Anesthesia:

Alfaxalone

Ketamine/midazolam

Full/ general anesthesia

How to care for an indwelling urinary catheter

Cleaning the UCATH

Should be Q6 or more if things like D+ are an issue.

Wash your hands immediately before putting on gloves – sterile gloves are gold standard.

Use 0.05% diluted chlorhexidine solution-soaked gauze.

Begin at the prepuce and move down the entire collection kit line.

Monitoring urine output in your UO patient:

Quantify urine every 2-4 hours.

Use graduated cylinder, not the numbers on the urine collection bag which can be inaccurate.

Urine output should never be 0 ml!!

The ideal urine output is 1-2 ml/kg/hour.

Important to not the appearance of urine in medical notes along with amount collected.

Long term considerations of care:

Prazosin – falling out of favor with most clinicians.

Phenoxybenzamine – acts to decrease muscle tone of urethra.

Both are high blood pressure medications that target/ block alpha one receptors in the urethral sphincter and surrounding areas which allows for relaxation of the urethra and better urine flow.

Antibiotics – only needed if concurrent infection present.

Buprenorphine, gabapentin – provide pain relief and relaxation.

Urinary specific diet

Stress reduction

Environmental factors

Extra/clean litter boxes

Feliway diffuser

Encouragement to drink more water with fountains.

References:

Conway DS, Rozanski EA, Wayne AS. Prazosin administration increases the rate of recurrent urethral obstruction in cats: 388 cases. Journal of the American Veterinary Medical Association. 2022 June. Volume 260: Issue S2

Silverstein D, Hopper K. Small Animal Critical Care Medicine. 2nd Ed. St. Louis: Saunders; 2015. Battaglia AM, Steele AM. Small Animal Emergency and Critical Care for Veterinary Technicians. 4th ed. St. Louis: Elsevier; 2021.

SAVING NINE LIVES: FELINE BLOOD TRANSFUSIONS

Lindsey Ashburner [1]

[1] University of Edinburgh, Edinburgh, United Kingdom

Learning objectives:

- Identify the indications for blood transfusion

- Understand feline blood types and cross-matching

- Selection a feline donor and how to carry out a donation

- Safe administration of a blood transfusion and possible reactions

- What are xenotransfusions and when are they indicated?

Introduction

The administration of blood to a feline patient can be a lifesaving procedure but does not come without risk. It is important to consider the necessary pre-transfusion testing and to understand the severe harmful reactions that can be seen. The most common indications for transfusion are severe anaemia, haemorrhage or coagulopathies. The decision to provide a red blood cell (RBC) transfusion should not be guided solely by a low packed cell volume (PCV) Cats with chronic anaemia can tolerate lower PCVs than those with an acute anaemia. Parameters suggestive of reduced oxygen carrying capacity i.e. tachycardia, weak pulses, prolonged capillary refill time, lethargy and weakness are all transfusion triggers.

Blood types and crossmatching

Blood types result from the presence of antigens on the surface of the RBCs. Unlike dogs, cats have naturally occurring antibodies against the blood group they are lacking. Resultantly an immune response can be induced if a different blood type is given, hence why blood typing is especially important in cats. The AB blood grouping system in cats identifies blood groups A, B, and AB. Type A is the most common blood type (67-87% in non-pedigree cats), type B is less common (7.9-30%) and type AB is regarded as rare (1.9-5%). Type B cats have anti-type A haemolysins and haemagglutinins which can cause premature destruction of transfused RBCs and severe, acute, and potentially fatal haemolytic transfusion reaction. If type B blood is transfused to a type A cat, there will be premature destruction of the transfused RBCs, but the reaction will be milder, delayed, and unlikely to be fatal. Type AB cats should ideally receive type AB blood or, if not available, type A blood after cross-matching. Unlike blood typing, cross-matching

mimics a transfusion outside of the body, directly assessing if two individuals' blood cells/plasma are compatible when mixed. Major cross-match combines donor red cells and recipient serum/plasma to detect a major transfusion reaction. Minor cross-match combines the donor plasma/serum with recipients red cells to detect reactions to components within the plasma.

Feline donor selection and blood collection

Careful blood donor selection is crucial to the transfusion as it decreases the risk of injury to the donor and of complications to the recipient. The criteria for donor selection are:

- Aged between 1-8 years
- Bodyweight ≥4kg
- Up to date with vaccinations and deworming
- Full biochemistry, complete blood count, blood smear, FIV/FeLV and PCR for *mycoplasma* spp
- Echocardiography ideally performed to rule out cardiac disease
- Amenable temperament
- Further infectious disease testing may be required dependent on location

The volume of blood collected should not exceed 12ml/kg. There are multiple feline blood collection systems available. An in-clinic prepared collection system with syringes, a three-way stopcock, and a butterfly needle is also suitable. In this situation, 1ml of anticoagulant (ACD or CPDA) per 7ml of blood should be preprepared into syringes prior to collection. The jugular area should be clipped and numbing cream applied before aseptically preparing the area. The blood collection should be carried out in an aseptic manner and the donor cat should then be placed on intravenous fluid therapy to replace twice the volume donated.

Administering a blood product

Transfusion is initiated at a slow rate (0.5-1ml/kg/hr) for the first 30mins, whilst the patient is closely monitored for any signs of a reaction. If there are no concerns, the rate can then be increased to deliver the entire transfusion over an appropriate time (e.g. total transfusion time of 4-6hrs). During the transfusion, temperature, heart rate, pulse quality, respiratory rate/effort, blood pressure, mucus membrane colour, capillary refill time and demeanour are regularly monitored. Any vomiting or diarrhoea should be noted. If any urine is passed or blood samples obtained, the urine/plasma colour should be noted to monitor for haemoglobinuria/haemoglobinaemia respectively. The most common reactions are febrile non-haemolytic reactions, allergic reactions, and circulatory overload. If signs of a transfusion reaction are seen, the transfusion should be stopped immediately and the veterinary surgeon informed.

Xenotransfusions

If feline type-compatible blood is not available and the patient has severe life-threatening anaemia or ongoing haemorrhage, canine blood can be used for the transfusion. Delayed haemolytic reactions have been reported in as early as one day after the xenotransfusions and antibodies are synthesised in the post transfusion period. Xenotransfusions should only be performed in severe emergency conditions and can only be performed once.

References:

Barfield D. and Adamantos S., Feline blood transfusions: a pinker shade of pale, Journal of Feline Medicine and Surgery, 2011, 13(1), pp 11-23

Davidow B., Transfusion medicine in small animals, Vet Clin North Am Small Anim Pract 2013: 43, pp 735-756

Day M. and Kohn B., BSAVA Manual of Canine and Feline Haematology and Transfusion Medicine, 2012, Gloucester: BSAVA

Le Gal A., Thomas E.K., Humm K.R., Xenotransfusion of canine blood to cats: a review of 49 cases and their outcome. J Small Anim Pract 2020; 61, pp 156-162

Merrill L., Small Animal Internal Medicine for Veterinary Nurses and Technicians, 2012, Iowa: Wiley-Blackwell

AND THE BEAT GOES ON

Chloe Fay [1]

[1] New Priory Vets, Brighton, United Kingdom

Learning objectives:

- Recognition of a normal ECG trace.

- Use of the algorithm to identify abnormal traces.

- General overview of cardiac anatomy and correlation to the ECG

- Heart rate calculation from paper ECG trace.

Proceeding:

Electrocardiography records the average electrical potential generated in the heart, mapped in voltage and time. Whilst ECG can characterise rhythms, demonstrate hypertrophy and chamber dilation, it does not offer any information regarding cardiac contractility and may even be normal in patients with advanced cardiovascular disease. The ECG as we know it, a bipolar triaxial lead system, was developed in the early 20th century by Willem Einthoven. These leads are used to create multiple angles to view the electrical impulses moving through the myocardial tissue. As the electrical waveform moves toward a positive electrode, it will generate a positive waveform, and those directed away from the same electrode will create a negative waveform. Electrical impulses are caused by the sodium ions rushing into myocytes and the K+ moving out, also known as depolarisation. Atrial depolarisation is initiated by depolarisation of specialised tissue called the sinoatrial (SA) node, and occurs from right atrium to left. A prolonged P-R interval is usually caused by high vagal tone and relates to the time taken for impulse initiated at the SA node to travel to the ventricles via the atrioventricular (AV) node. Atrial repolarisation is not visible on the ECG due to size of waveform and often because of the presence of the QRS complex. The electrical impulse travels via the AV node to the bundle of His, causing an initial depolarisation of the intra-ventricular septum correlating to the negative Q wave. Electrical impulses travel from the bundle of His into the right and left bundle branches within the ventricular walls. Impulses are conducted from the bundle branches to the Purkinje fibres and into the cardiac myocytes, causing a domino effect to initiate contraction of the ventricles. Depolarisation and contraction of the ventricles observed as the completion of the QRS wave.

The basilar portions are the last to depolarise creating a negative S deflection when reading the ECG in lead II. Ventricular repolarisation is seen in the T wave, which can be negative or positive in lead II. Impulses originating from above the AV node are termed ‘supraventricular’ and typically create tall and

narrow QRS complexes. Impulses initiated from the ventricles or the His-Purkinje system are known as ventricular. These ventricular rhythms are impulses that are slowly transmitted from individual cell to cell, producing a wide and bizarre QRS-T complex.

Recording and Interpreting the ECG

ECG electrodes should be placed on opposite sides of the heart to record the electrical activity between them. Most multi-parameter monitors calculate the heart rate (HR) second to second based on the impulses. It is important to bear this in mind when reading a HR from the multi-parameter monitor as this can sometimes be inaccurate due to double reading heart rates and artefacts such as shivering and respiratory rattle. If using a paper trace ECG, the bold lines indicate 5mm boxes, made up of smaller grids of 1mm boxes. The paper speed, alongside the use of these boxes can accurately calculate electrical amplitude calibration, time, electrical voltage and the HR of the patient. Common paper speeds are 25 mm/sec and 50 mm/sec; 30 of the 5-mm boxes (15cm) equates to 6 seconds at 25 mm/sec and 3 seconds at 50 mm/sec, allowing the user to calculate the HR of the patient.

Analysis of the heart's underlying rhythm should include the following steps:

- What is the rhythm (including the regularity and the relationship among complexes)?
- What is the relationship between the P and QRS complex? Is there a P wave for every QRS complex? Is there a QRS complex for every P wave?
- Where do the cardiac impulses originate (site of origin)?

References:

Durham HE. Cardiac anatomy. In: Durham HE, ed. Cardiology for Veterinary Technicians and Nurses. Hoboken, NJ: Wiley-Blackwell; 2017; 17–20.

Day, T.K. (2005) ECG interpretation in the critically ill dog and cat. Ames, Iowa: Blackwell Publishing. Gavaghan, B. (2008) ‘Small animal ecgs: An introduction’, Australian Veterinary Journal, 86(7), pp. 271–271.

Hemsley S. Electrocardiography. In: Durham HE, ed. Cardiology for Veterinary Technicians and Nurses. Hoboken, NJ: Wiley-Blackwell; 2017; 97–98.

Pace, C. (2016) ‘How to use an ECG machine’, The Veterinary Nurse, 7(1), pp. 42–47. Pace, C. (2018) ‘HOW ECG monitoring contributes to patient care’, The Veterinary Nurse, 9(4), pp. 216–223. doi:10.12968/vetn.2018.9.4.216.

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

MEDICAL MATH FOR THE VETERINARY TECHNICIAN

Angela Elia [1]

[1] Pieper Veterinary Group, Pieper Veterinary Group, Middletown, CT, United States

Learning objectives:

- Complete conversions between metric and imperial systems

- Complete continuous rate infusion (CRI) calculations for commonly used analgesics such as fentanyl and lidocaine

- Complete calculations for dextrose fluid additives

- Complete calculations for potassium chloride additives

Proceeding:

Basic Conversions:

Celsius (°C) to Fahrenheit (°F) conversion:

1.8(°C) + 32 = °F

Fahrenheit to Celsius conversion:

(°F - 32) × 5/9 = °C

Kilograms (Kg) to pounds (lbs) conversion:

Kilograms/2.2 = pounds

Milligrams(Mg) to Micrograms (Mcg) conversion:

Milligrams X 1000 = micrograms

Converting % to mg/ml

1% = 1gram in 100ml

2% = 2 grams/100 mL

2 grams = 2000 mg

2000 mg/100 ml = 20 mg/ml

Constant Rate Infusions (CRI):

The information you need to calculate a CRI is as follows:

Drug Concentration

Patient Weight

Dose Required

Number of hours you want the CRI to last

The rate at which the CRI will run

CRI Example:

You need to calculate a metoclopramide CRI to be started on a patient that weighs 17 kgs. The patient will be in the hospital for another 12 hours. The dose of metoclopramide is 2 mg/kg/day and the requested rate of the CRI to be at 1 ml/hour.

The breakdown of what we need looks like this:

Patient weight: 17 kgs

Dose of Metoclopramide: 2mg/kg/day

Rate of CRI: 1 ml/hour

Total Hours needed: 12 hours

First, let's find out how much metoclopramide we need for our patient:

17 kgs X 2 mg/kg = 35 mg per day (24 hours)

Now let's find out how many mg per hour that equals:

35 mg/24 hours = 1.45 mg per hour

Now let's change that mg per hour into ml per hour of metoclopramide

45 mg/5 mg/ml (concentration of metoclopramide) = 0.29 ml /hour

We will have to find out how much diluent to use now:

1 ml – 0.29 ml (metoclopramide) = 0.71 ml of diluent per hour.

So now we know that for each hour we want our CRI to run, we will need 0.29 ml of Reglan and 0.71 ml of diluent.

Since we need 12 hours of the CRI, we would need:

0.29 ml X 12 = 3.48 ml metoclopramide

71 ml X 12 = 8.52 ml diluent

Fentanyl CRI Example:

You are heading into surgery with a 12 kg dog that requires a fentanyl bolus as part of its pre-med and an intra-op fentanyl CRI. The CRI will run at 5 mcg/Kg/hr. How many ml of fentanyl will you need for a 6-hour CRI? What rate in ml/hr will it be run? Does it need to be diluted? The concentration of fentanyl is 50 mcg/ml.

The CRI will run 5 mcg/kg/hr for 6 hours. How many ml of fentanyl will we need? What is the rate in ml/hour?

12 kg X 5 mcg/kg = 60 mcg/hour

60 mcg/hr X 6 hours = 360 mcg

360 mcg / 50 mcg/ml = 7.2 ml of fentanyl needed

7.2ml / 6hr = 1.2ml/hr

Potassium Chloride Additive:

Your patient requires 30 mmol/L of KCl in Lactated Ringers Solution (LRS) for a patient. How many ml would you add to a bag of 500 ml? The concentration of KCL is 2 mmol/ml

There are two ways people like to complete this equation: Change 30 mmol of KCl into ml first or you can calculate for the meq needed and at the end convert to ml.

Using the first method:

30 mmol of KCl = 15 ml of KCl for each litre of intravenous (IV) fluids

15 ml/1000 ml = 0.015 ml of KCl for each ml of IV fluids

0.015 ml X 500 ml (in the bag) = 7.5 ml KCl needed

In the second method we see:

30 mmol of KCl per Litre (1000 ml)

30 mmol / 1000ml = 0.03 mmol/ ml of IV fluid

0.03 mmol X 500 ml = 15 mmol needed

15 mmol/ 2 mmol/ml = 7.5 ml of KCl needed

Dextrose (50%) Additive Short-Cut

Take the % of Dextrose you want to create

Multiply by 2

This is the number of ml you need to add per 100 ml of fluid

5% dextrose needed = 10 ml of dextrose per 100 ml of fluid

7.5% dextrose needed= 15 mls of 50% dextrose per 100 ml of fluid

Remember to remove the same amount of fluid from the bag before adding dextrose!

References:

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

Norkus, CL. Veterinary Technician's Manual for Small Animal Emergency and Critical Care. 2nd Ed. Hoboken: John Wiley & Sons; 2019

Bill, R. Medical Mathematics and dosage calculations for the veterinary technician, Wiley-Blackwell;2019

SOOTHING THE STORM: SEDATION STRATEGIES FOR ICU PATIENTS

Lisa Angell [1]

[1] Royal Veterinary College, United Kingdom

Learning objectives:

- Increase understanding of the different sedation options for reducing anxiety in the ICU hospitalised patient.

- Summarise the considerations and contraindications for different medications, with a particular focus on seratonin syndrome

- Have a better understanding of how we can include local anaesthetics to our protocols to facilitate procedures and reduce systemic sedation requirements.

Proceeding:

There are a variety of reasons why patients in the ICU might require sedation. We see cases like the BOAS patients in respiratory crisis, where if we can manage anxiety and stress levels, this could be key to a helping create a successful outcome. Ignoring stress and anxiety in these patients can lead to development of respiratory fatigue and unltimately respiratory failure necessitating mechanical ventilation.

We also perform an array of essential medical procedures to our patients within the ICU. Procedures such as securing vascular access, chest tube or urinary catheter placement or facilitating other diagnostic procedures, might require a patient to be sedated to be successfully performed.

Experienced ICU nurses are likely to be familiar with benzodiazepines, opioids and alpha 2 agonists used for sedation, so we'll focus within this piece on some other agents that we might be less familiar with that are becoming more regularly used in veterinary patients.

Trazodone

Trazodone is a medication which is commonly prescribed for depression and anxiety in humans and is becoming more popular than acepromazine for managing stress and anxiety in the veterinary patient. Trazodone belongs to the class of drugs known as serotonin antagonists and reuptake inhibitors (SARIs). Serotonin is a neurotransmitter which plays a crucial role in regulating various physiological processes. Contributing to help regulate mood, appetite, digestion, sleep, and in humans increases an overall sense of well-being which influences mental health and emotional stability. Trazodone increases serotonin levels in the brain by blocking certain serotonin receptors. Sometimes, regardless of how sick our

patients are, they get very stressed in hospital, and we've all seen that BOAS case tip over the edge into crisis after being in hospital for only a short period of time. Although off licence, trazodone can be given orally in tablet or liquid form, but we can also achieve similar results by rectal administration. Trazodone should not be used with any other monoamine oxidase inhibitors or tricyclic antidepressants due to the risk of serotonin syndrome.

Gabapentin

Gabapentin belongs to the class of drugs known as anticonvulsants or antiepileptic drugs. Primarily used to treat neurological conditions, such as seizures and neuropathic pain, it is also used off-label to treat anxiety and seems to work very well to relax our 'spicy' feline friends. Gabapentin is mostly used in combination with other drugs in dogs, so typically not reached for as a sole agent in this species. Sedation can be sufficient for general well-being in hospital as well as facilitating minor procedures, sedation can be enhanced in combination with an opioid for more invasive procedures.

Aggressive critical patients

Aggressive critical patients can be a challenge. A particular challenge is in managing those with pre-existing cardiac disease, where maybe we haven't been able to restrain to secure intravenous access. It may be safer in this patient to avoid alpha 2 agonists and butorphanol may not providing sufficient sedation. Where do we go from here? Alfaxalone is an anaesthetic induction agent that can be administered intramuscularly, and with time to take effect and in combination with an opioid can provide appropriate sedation for some minor procedures. A consideration for the use of alfaxalone is the size of patient. The dose required to achieve sedation can result in a large volume to inject in dogs which may preclude its use in some cases.

Local anaesthetics

Local anaesthetics have been used to provide analgesia in human medicine for many years and their use within veterinary pain management protocols is increasing. Their use as part of a multi-modal analgesia plan for a range of surgical procedures under anaesthesia is well established however, their place in management of patients within the ICU should be further explored and considered. Using a local technique can be simple and effective at helping to facilitate of a range of procedures such as drain or catheter placement. As an example, an intercostal block could allow for thoracocentesis without the need for sedation, increasing patient safety whilst providing adequate analgesia. Effective local anaesthetic techniques are proven to reduce the requirement for anaesthesia and are, when appropriately used, considered safe. Local anaesthetic agents can also be administered into drains placed following procedures to provide ongoing analgesia or administered into specific catheters, such as epidural or wound soaker catheters. These techniques help improve patient comfort and facilitate repeated procedures to wounds which require interventions on a regular basis.

References:

Ramsey, I. K. (Ed.)(2023) BSAVA Small Animal Formulary: Part A, Canine and Feline [11th ed.]. British Small Animal Veterinary Association

Mohammad-Zadeh LF, Moses L, Gwaltney-Brant SM. Serotonin: a review. Journal of veterinary pharmacology and therapeutics. 2008 Jun;31(3):187-99.

Chea, Bunna, and Mario Giorgi. "Trazodone: A review of its pharmacological properties and its off-label use in dogs and cats." American Journal of Animal and Veterinary Sciences12.4 (2017): 188-194.

Ferguson LE, McLean MK, Bates JA, Quimby JM. Mirtazapine toxicity in cats: retrospective study of 84 cases (2006–2011). Journal of feline medicine and surgery. 2016 Nov;18(11):868-74.

Angell, Lisa. "Local Anaesthetic Techniques." The Veterinary Nurse's Practical Guide to Small Animal Anaesthesia (2023): 296-318.

AIRWAY MANAGEMENT IN THE ICU

Eleanor Haskey [1]

[1] Royal Veterinary College, Royal Veterinary College, London, United Kingdom

Learning objectives:

- List the indications and contraindications for an artificial airway.

- Describe difficult airway techniques that can be utilised to achieve successful endotracheal intubation

- Discuss how to effectively manage the intubated (endotracheal or tracheostomy) tube patient to prevent complications arising

- Explain how to safely extubate the patient and monitor for signs of decompensation requiring reintubation

Proceeding:

Artificial airway placement

For any nurse or technician working in ECC, patients requiring an artificial airway are common. These patients fall into three groups: those with an upper airway obstruction (such as a BOAS crisis), patients with a reduced gag who have an unprotected airway or patients which require oxygenation and ventilation support. Nurses and technicians will often be responsible for placing these devices/changing them in addition to their ongoing management and troubleshooting complications.

A major body system assessment needs to be performed to identify whether an artificial airway is required. In the majority of situations endotracheal intubation is the preferred method as it is quick to perform and less invasive than a surgical tracheostomy, however in some cases of upper respiratory obstruction it can be challenging and on occasion not possible.

If the patient is painful on assessment, opioid analgesia can be administered intramuscularly with, or followed by, a low dose of acepromazine or alpha-2 agonist such a medetomidine or dexmedetomidine. If the patient is not painful, butorphanol can be administered intramuscularly for its sedative and anxiolytic effects. Intravenous access should be obtained as soon as possible without causing further stress to the patient.

Pre-oxygenation (without causing more stress) will help maintain oxygenation and increase the window for intubation to be achieved, by increased the concentration of oxygen within the lung's functional residual capacity.

In challenging airway cases the use of airway aids can increase the chances of a successful endotracheal intubation. A laryngoscope will dramatically increase visibility of the larynx and helps to ensure a minimal amount of trauma to the tissues. A selection of endotracheal tubes (ET tube) in a range of sizes should be prepared. A rigid dog urinary catheter, which has been adapted to attach to a breathing system can helpful for difficult intubations and to provide oxygenation. Stylets (such as the rigid urinary catheter or bougie) can be used to feed the ET tube into the trachea. Endotracheal intubation should be confirmed on a capnograph. The tube must be secured in situ and the cuff should be checked and inflated to reduce the risk of aspiration should there be regurgitation, as well as protecting the veterinary team from environmental contamination of any volatile agent.

In cases where endotracheal intubation is unachievable then transtracheal oxygen or an emergency surgical tracheostomy are indicated.

Management

Best practice is for any intubated patient to have capnograph monitoring. This will alert the veterinary team to hypercapnia ($ETCO_2$ >60mmHg), and the need to initiate positive pressure ventilation. Pulse oximetry will aid in decision making about increasing the patient's $Fi0_2$.

Patients with an artificial airway require close supervision around the clock. Additional considerations that need to be factored into the patient care plan include tube management such as tube changes, suction of tracheal or lower airway secretions, humidification of inspired oxygen and regular cuff pressure monitoring. Intubated patients will also need oral care comprising suctioning the pharynx and oral cavity, tongue and mucous membrane care to reduce the bacterial load in the mouth and reduce the risk of pneumonia secondary to the artificial airway. Tracheostomy tube patients will also need kennel adaptations such as supervised access to food and water. Having an emergency kit prepared to reintubate in cases of a respiratory obstruction can save time.

For any patient in an unstable or critical condition, Kirby's rule of 20 can be used as a checklist to aid in a holistic management plan for the patient. This can help to identify current patient concerns in addition to potential concerns that might arise during hospitalisation. Clinics should have standard operating procedures for the placement and management of devices so there is consistency in nursing across the team and to prevent complications arising such as hospital acquired infections.

Resident Stream, Saturday 31 May 2024

THE THEORY BEHIND YOUR EMERGENCY SURGICAL PROCEDURES

Anna Frykfors von Hekkel [1, 2]

[1] Royal Veterinary College, Royal Veterinary College, Small Animal Surgery, South Mimms, United Kingdom
[2] Coastal Veterinary Specialists, Royal Veterinary College, Small Animal Surgery, London, United Kingdom

Learning objectives:

- Understand key principles of commonly performed emergency surgical procedures; temporary tracheostomy, lung lobectomy, liver lobectomy, gastric dilation and volvulus (GDV), splenectomy.

- Be aware of key anatomical features relevant to the procedures.

- Know how to best to approach the target area in a safe manner.

Proceeding:

Temporary Tracheostomy

- The outer diameter of the tracheostomy tube should not exceed 75% of tracheal luminal diameter.
- Unless being used for ventilation, cuffed tubes should be avoided to reduce the risk of tracheal necrosis and stenosis.
- Tracheal stay sutures are labelled “up’ and “down” to aid tube replacement.
- Following tube removal, the site is left to heal by second intention.

Lung Lobectomy

- Unilateral access to a lung lobe is typically achieved via intercostal thoracotomy.
- The caudal and accessory lung lobes are attached to the mediastinal pleura by a pulmonary ligament – this is transected to facilitate lobe mobilisation and hilar access.
- Lung lobectomy can be performed by suture ligation or with a vascular stapling device.
- A leak test should be performed prior to closure.

Liver Lobectomy

- The liver in dogs and cats is deeply fissured, giving rise to distinct lobes.
- Triangular ligaments tether part of the right lateral, right medial and left lateral lobe to the diaphragm. These can be transected to facilitate lobe mobilisation and hilar access.
- The afferent blood supply to the liver consists of the portal vein (80% blood volume, 50% oxygen supply) and hepatic artery.

- Efferent supply is via hepatic veins which insert into the caudal vena cava.
- The exact vascular supply of each liver lobe is variable, but typically consists of one hepatic artery branch, one portal vein branch, one hepatic vein and one hepatic duct per lobe.
- Complete liver lobectomy is most easily performed with use of a surgical stapling device placed across the lobar hilus. Dissection and suture ligation can be performed. Use of self-ligating suture loops has also been reported.

The Pringle Manoeuvre

The epiploic foramen lies medial to the caudate process of the liver and is bound dorsally by the caudal vena cava and ventrally by the hepatic portal vein and the hepatic artery.

- A finger is introduced into the foramen and is directed ventrally.
- Compression is applied between thumb and forefinger allowing vessel compression to temporarily control hepatic haemorrhage (5-20 minutes).

GDV

- As a surgeon typically positioned on the left side of the surgical table, de-rotation is achieved by reaching over the patient into the left craniodorsal abdomen to grasp the pylorus and then pulling the pylorus ventrally and towards oneself.
- After de-rotation the stomach is allowed to re-perfuse for around 10 minutes. Abdominal exploration can be carried out during this time with close inspection of the spleen and short gastric blood vessels and finally the stomach including the dorsal aspect.
- Several gastropexy techniques have been reported; incisional, belt-loop, incorporating, circumcostal. Experimentally, belt-loop and circumcostal have been reported to withstand the greatest force, however the force they need to withstand clinically is not known. Incisional gastropexy is recommended as this is most easily performed and performs equivalently to belt-loop gastropexy in clinical cases.
- Owners should be warned that despite gastropexy, recurrence of GDV is possible.

Splenectomy

There are several techniques for performing splenectomy.

- Ligation of hilar blood vessels: This is most easily performed, but is more time consuming. The hilus of the spleen is identified and vessels are ligated individually or in small bunches close to the splenic hilus.
- Ligation of the major splenic blood vessels: The major splenic blood vessels include the splenic artery and vein, the left gastroepiploic artery and vein and the short gastric arteries and veins. Splenectomy by suture ligation can be faster achieved by placing targeted ligatures on these vessels. The location of the distal aspect of the left limb of the pancreas should be inspected prior to placement of the sutures.
- Bipolar vessel sealing device**:** Bipolar vessel sealing devices can be used in place of suture ligation which can drastically reduce surgical time.

FROM MOLECULES TO MEDICINE: CELLULAR PERSPECTIVES ON VASOPRESSORS AND INOTROPES IN THE ICU

Dave Beeston [1, 2]

[1] Willows Veterinary Referral Centre, Emergency and Critical Care, Birmingham, United Kingdom
[2] Royal Veterinary College, Emergency and Critical Care, Hatfield, United Kingdom

Learning objectives:

- To identify and understand the second messenger systems of the cardiovascular system

- To understand the mechanism of action of common haemodynamic agents

- To apply the cell-based pharmacological knowledge to a clinical case

Proceeding:

An Introduction to Cardiovascular Cellular Physiology

The cells of the cardiovascular system have varied structure and function, but at the core of their physiology is a dependence on numerous second messenger systems and their associated guanine nucleotide-binding (G-protein) linked receptors. The three main G-protein linked receptors involved in catecholamine-mediated cardiovascular cellular signalling are the stimulatory G-protein (Gs), the inhibitory G-protein (Gi) and the phospholipase C-coupled G-protein (Gq).

The Cardiomyocyte and Regulation of Inotropy and Lusitropy

Calcium is integral to all cardiovascular function and is tightly regulated. The amount of calcium that enters the cardiac myocyte via the L-type channels during depolarisation is largely regulated by phosphorylation of the L-type channel. The primary mechanism for this involves cyclic adenosine monophosphate (cAMP), the formation of which is coupled to B_1-adrenoreceptors, a Gs-protein-linked receptor. Calcium concentration is also regulated by the sarcoendoplasmic reticulum calcium ATPase (SERCA) pump which transports intracellular calcium into the sarcoplasmic reticulum. Phosphorylation (and subsequent inhibition) of the SERCA inhibitor, phospholamban, leads to increased SERCA activity and sequestration of calcium which enhances both lusitropy (relaxation) and future inotropy. Finally, calcium efflux from the myocyte is regulated by the sarcolemmal sodium-calcium exchanger (NCX) and associated sodium-potassium ATPase, and the ATP-dependent calcium pump.

Circulating norepinephrine and epinephrine and sympathetic-nerve released norepinephrine bind to the B_1-adrenoreceptor leading to activation of the enzyme adenylyl cyclase which in turn hydrolyses adenosine triphosphate (ATP) to cAMP. cAMP acts as a second messenger to activate protein kinase A

which is capable of phosphorylating different sites within the cell, including the L-type calcium channel and phospholamban. Another mechanism of increasing intracellular cAMP is by preventing its degradation by phosphodiesterase (PDE) III with the administration of the PDE III inhibitor, pimobendan. Increased intracellular calcium leads to calcium-induced calcium-release from the sarcoplasmic reticulum via the ryanodine receptor, further increasing intracellular calcium. Free intracellular calcium binds to troponin C leading to a conformational change in the troponin complex that exposes myosin-binding sites on actin, facilitating the cross-bridge movement and cell contraction. Therefore, increasing norepinephrine or epinephrine concentration, or administration of dobutamine, can lead to increased inotropy as a result of B_1-adrenoreceptor activation and increased intracellular calcium. The Gi-protein inhibits adenylyl cyclase and therefore decreases cAMP and subsequent inotropy. This is mediated by the muscarinic receptor (M2) that binds acetylcholine released from the parasympathetic (vagal) nerves within the heart.

The phospholipase C-coupled G-protein (Gq) receptor provides a second cAMP-independent pathway via norepinephrine (alpha-1 adrenoceptor), angiotensin II (AT1 receptor) and endothelin-1 (ET_A). Activation of phospholipase C (PL-C) leads to breakdown of phosphatidylinositol 4,5-bisphosphate (PIP_2) into diacylglycerol and inositol 1,4,5-triphosphate (IP_3), with IP_3 leading to further calcium release from the sarcoplasmic reticulum.

Ultimately, all inotropic pathways that involve the increase of intracellular calcium will lead to increased oxygen demand due to consumption of ATP, either via SERCA, ATP-dependent calcium pumps, the sodium-potassium ATPase (NKA) or the ATP-dependent relaxation of the actin-myosin filaments.

Additionally, alterations in the binding affinity of troponin C can be affected by endogenous and exogenous factors. Acidosis, as occurs during myocardial hypoxia, has been shown to decrease troponin C affinity for calcium binding. Pimobendan has been shown to increase affinity of troponin C for calcium, independent of its affects as a PDE III inhibitor.[2]

Vascular Smooth Muscle and Regulation of Vascular Tone

Vascular smooth muscle is under the influence of sympathetic adrenergic nerves, circulating hormones (e.g. epinephrine, norepinephrine, angiotensin II), endothelial substances (e.g. nitric oxide, endothelial-derived hyperpolarizing factors [EDHF], endothelin-1 and PGI_2), and vasoactive substances released by surrounding tissue (e.g. adenosine, ATP, CO_2, potassium). For the purposes of this talk, we will focus on the response to circulating hormones and potential therapeutic options.

Vascular smooth muscle tone is determined by the balance of contraction and relaxation. The contractile apparatus, myosin light-chain, is regulated by myosin light-chain kinase and ATP (leading to contraction) and myosin light-chain phosphatase (leading to relaxation). Myosin light-chain kinase is stimulated by calcium-bound calmodulin and inhibited by cAMP. Increasing intracellular calcium will therefore lead to increased MLCK activity via the calcium-calmodulin pathway.

As in the cardiac myocyte, cAMP levels are regulated by the Gs and Gi proteins. The Gs protein is linked to the beta-2-adrenoceptor, and the Gi protein is linked to the alpha-2-adrenoceptor. Unlike the cardiac myocyte, increasing cAMP does not lead to increased release of calcium from the sarcoplasmic reticulum. However, the Gq-mediated IP_3 pathway still leads to release of calcium from the sarcoplasmic

reticulum and accounts for the vasoconstrictive effects of alpha-1 agonists, vasopressin, endothelin-1 and angiotensin II.

An additional pathway involves the regulation of myosin light chain phosphatase (MLCP). MLCP leads to relaxation of the myosin light chain and is stimulated by cGMP and inhibited by Rho-kinase. Guanylyl cyclase is stimulated by nitric oxide which diffuses into the muscle cell, leading to production of cGMP from guanosine triphosphate and subsequent vasodilation. Rho-kinase is coupled to the phospholipase C-coupled G-protein (Gq) receptor which is stimulated by the same mediators as noted above and leads to inhibition of MLCP and shifts towards vasoconstriction.

In summary, vascular smooth muscle stimulation by catecholamines leads to either vasoconstriction or vasodilation based on the ratio of affinity for B_2 receptors versus a_1 and a_2 receptors.

References:

Unger M, Debold EP. Acidosis decreases the Ca2+ sensitivity of thin filaments by preventing the first actomyosin interaction. Am J Physiol Cell Physiol. 2019;317(4):C714-C718. doi:10.1152/AJPCELL.00196.2019/ASSET/IMAGES/LARGE/ZH00091985230004.JPEG

Solaro RJ, Fujino K, Sperelakis N. The positive inotropic effect of pimobendan involves stereospecific increases in the calcium sensitivity of cardiac myofilaments. J Cardiovasc Pharmacol. 1989;14 Suppl 2(2):S7-12. doi:10.1097/00005344-198906142-00003

Klabunde RE. Cardiovascular Physiology Concepts. 3rd ed. Wolters Kluwer Health; 2020. Accessed December 31, 2022. https://www.cvphysiology.com/textbook-3

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

PHYSIOLOGY OF CARDIAC TAMPONADE

Christopher Kennedy [1]

[1] University of Illinois, Emergency and Critical Care Service, Urbana, United States

Learning objectives:

- Learn the different terminologies of pericardial diseases

- Review the physiology of cardiac tamponade

- Discuss haemodynamics in pericardial disease

Proceeding:

Pericardial effusion is by far the most recognized pericardial problem, though it is not a specific disease; it is a manifestation of several diseases. Pericardial tamponade is haemodynamic compromise resulting from compression of the cardiac chambers by fluid in the pericardial space. Importantly, pericardial effusion can be present without tamponade physiology, as is commonly seen in cats with congestive heart failure.

Pericarditis is inflammation of the pericardium. It is not a commonly used term in veterinary literature. Pericarditis can result from several diseases including infections, neoplasias, systemic inflammatory diseases, uraemia, radiation therapy and cardiac trauma. Pericarditis can cause pericardial effusion and tamponade. Constrictive pericardial disease is different to pericardial tamponade: this uncommon condition typically occurs because of chronic pericardial inflammation. Constrictive-effusive pericardial disease is a very uncommon combination of constrictive physiology that can also manifest as tamponade physiology.

The haemodynamic changes that occur in pericardial diseases typically exist along a spectrum – from simple pericardial effusion to pericardial tamponade, or from a constrictive yet elastic pericardium to a constrictive, inelastic pericardium. Echocardiography can be useful to describe these haemodynamic changes; however, intracardiac catheterization allowing chamber pressure measurements is the superior methodology.

Human texts describe echocardiographic magnitudes of pericardial effusions ranging through small (< 0.5 cm), moderate (0.5 – 2 cm) and large (> 2 cm). This is unhelpful, as the determining factors of intrapericardial pressure, thus the haemodynamic consequences, are 1) pericardial volume, 2) rate of volume accumulation, 3) pericardial compliance, and 4) heart chamber compliance. The effective circulating volume also contributes to the haemodynamic status. This creates the interesting patient that is hypovolaemic and has pericardial effusion where either tamponade physiology can manifest at lower

intrapericardial pressures, or the classic tamponade physiology may be masked by the hypovolaemia and missed by the clinician. This highlights a danger of diuretic administration to patients with pericardial effusion.

Cardiac tamponade is a physiology, not a disease itself. Practically, tamponade is a clinical diagnosis: tachycardia (compensation) + signs of poor cardiac output + pericardial effusion. Once a critical threshold of pericardial fluid has accumulated, additional fluid increases the intrapericardial pressure exponentially. This pressure is applied to the heart chambers, impairing diastolic filling. Essentially, the pericardial fluid competes with the chambers for space within the volume enclosed by the pericardium. A three-phase model has been proposed to describe the spectrum of changes that occurs. Firstly, the intrapericardial pressure increases and leads to compromised right chamber filling; overt clinical signs may not be present. Secondly, the pressure rises and begins to negatively impact right and left chamber filling; compensatory signs are seen, i.e., increased sympathetic tone (tachycardia, increased contractility) to maintain cardiac output despite diminished stroke volume. Blood pressure may be normal or even elevated due to vasoconstriction: this does not mean that the patient is “stable”; there remains a serious risk to life. As pressure continues to rise, the third phase of overt haemodynamic collapse and obstructive shock is seen.

Pulses paradoxicus is variably described as a decrease in systolic blood pressure of ≥ 10 mmHg during normal inspiration. This can be detected by palpating pulses. Pulses paradoxicus can also be seen with constrictive pericarditis, obstructive pulmonary diseases, restrictive cardiomyopathy, pulmonary emboli or severe hypovolaemia. In approximately 20% cases of pericardial tamponade, pulses paradoxicus may be absent despite tamponade physiology – hypovolemia is a key reason for this.

A clever question to ask is “what effect is the pericardial effusion having on systemic venous return, cardiac output, arterial blood pressure and intracardiac pressures?”

References:

Bussadori C. Textbook of cardiovascular medicine in dogs and cats. Edra, 2023.

Ware, W and Bonagura, J. Cardiovascular disease in companion animals: dog, cat and horse. CRC press, 2021. 3. Stouffer, G. Cardiovascular hemodynamics for the clinician. Wiley Blackwell, 2017.

Otto, C. Textbook of clinical echocardiography. Elsevier, 2023.

THE PHYSIOLOGY OF HAEMODYNAMIC MONITORING

Mark Kim [1, 2] Mathieu Magnin [2, 3]

[1] SIAMU, VetAgro Sup, Marcy L'Etoile, France
[2] APCSe research unit, VetAgro Sup, Marcy L'Etoile, France
[3] Department of Physiology, Pharmacology and Therapeutics, VetAgro Sup, Marcy L'Etoile, France

Learning objectives:

- Understand the difference and clinical relevance of macrocirculation, microcirculation, and tissue perfusion

- Explain the basic mechanics and physiology of lesser known devices that aim to evaluate the microcirculation and/or tissue perfusion

- Describe the use of these devices in current research and clinical settings

Proceeding:

Background

Macro- vs microcirculation vs tissue perfusion

Managing the circulatory system; the heart plus macro- and microcirculation, is central to managing many of our critically ill patients. Doing so ensures optimal circulation of oxygen-carrying red blood cells to tissues and their cells, potentially limiting organ injury to a reversible state and avoiding irreversible changes. Existing monitoring tools of the circulatory system perform well in many of our patients. Global markers, both accessible (blood pressure, blood lactate), and impractical (cardiac output), can provide meaningful data to identify circulatory status as well as monitor the response to treatment. However, in a subset of patients with varying types of circulatory disturbances, these established monitoring tools will not tell the full story.

Hemodynamic coherence

In some patients, resuscitation is associated with the correction of macrocirculatory parameters (blood pressure, cardiac output) even though tissue hypoperfusion persists. This phenomenon has been called loss of hemodynamic coherence and is likely the main obstacle to successful monitoring of the entire circulatory system. An observational study in human sepsis patients showed non-survivors had persistent microcirculatory disturbances compared to survivors, despite normalization of global markers. Much available data exploring this concept focuses on septic patients, who perhaps seem more susceptible to

microcirculatory disturbances. But a growing body of evidence identifies similar behaviour in haemorrhagic shock patients.

Devices

So what tools are available to complement traditional measures of tissue perfusion and circulatory function? Whilst the entire range of tools and devices rests beyond the scope of this presentation, we will focus on the following devices that are in various stages of development and with which we have had personal experience with:

Infrared thermography

Sublingual videomicroscopy

Tissue-to-arterial PCO_2 gradient

Urethral photoplethysmography

Urinary PO2

Infrared thermography

Most widely known by its simpler form as a marker of global perfusion (extremity temperature), infrared thermography offers an objective approach that retains the non-invasive and ease-of-use advantages. Peripheral (hand and foot or paw) surface temperature is mostly the product of cutaneous blood flow. Central (proximal limb, body) surface temperature is also affected by heat production by metabolic processes, notably in the gut. During shock, circulatory failure can activate compensatory mechanisms and peripheral vasoconstriction increasing the delta between the two regions. Earlier studies in human surgical patients demonstrated an association between the haemodynamic status and central-to-peripheral temperature gradients, as well as higher mortality rates in patients with greater temperature gradients and slower improvement following volume resuscitation.

Sublingual videomicroscopy

A key principle in microcirculatory disturbance is the alterations to capillary oxygen delivery (DO_2). Effective cellular oxygenation requires adequate capillary density to minimize the distance between each cell and the nearest capillary endothelium, adequate red blood cell (RBC) flow, and of course adequate RBC oxygen content. Disease, particularly sepsis, can lead to endothelial swelling, glycocalyx shedding, reduced RBC deformability, capillary fluid leak, and trigger leukocyte and platelet activity resulting in loss of microcirculatory flow and capillary density. Videomicroscopy permits the direct visualization and evaluation of these pathologic changes. This technique is generally used in the sublingual region, mainly for practical reasons. Many limitations and uncertainties currently stand in the way of its practical use: variability of measurements, challenges with video analysis, and uncertainties regarding the relevance of sublingual microcirculation. Recent consensus guidelines were published with the aim of overcoming these limitations. Few clinical studies have focused on the utility of videomicroscopy during resuscitation. A recent study showed sublingual microcirculation guided resuscitation was associated with increased mortality rather than survival, although the trial design was hampered by several limitations.

Tissue-to-arterial PCO_2 gradient

Cellular CO_2 production is rapidly cleared by the circulatory system. Even with increased CO_2 production (eg. during anaerobic metabolism, H^+ increase is buffered by $HCO3^-$ leading to CO_2 production as described by the carbonic acid dissociation equation), normal to increased blood flow will rapidly clear this highly soluble gas. But in states of decreased microcirculatory flow, tissue CO_2 can accumulate and increase, relative to blood PCO_2 levels. Transcutaneous tissue PCO_2 alone has been used as a non-invasive alternative to $PaCO_2$ measurements. When paired with simultaneous $PaCO_2$ measurements, the tissue-to-arterial PCO_2 gradient reflects tissue perfusion. A small trial in septic shock patients showed a PCO_2 gradient threshold of 16 mm Hg discriminated survivors from non-survivors with a sensitivity of 83% and a specificity of 90%. But experimental data from a porcine haemorrhagic shock model showed the tissue-to-arterial PCO_2 gap, a purported marker of regional tissue perfusion, correlated better to cardiac output measurements than blood lactate or shock index measures.

Urethral photoplethysmography

Optical monitoring devices offer non-invasive to minimally invasive tissue perfusion monitoring. Several devices have been developed, notably near-infrared spectroscopy and laser doppler flowmetry. Photoplethysmography devices for pulse oximetry have been developed to calculate the peripheral perfusion index. These devices transmit infrared and near-infrared light into a tissue bed and pulsatile, alternate current (AC), and non-pulsatile direct current (DC) light signals are reflected or transmitted accordingly: by tissue (stationary, thus DC) versus blood flow (pulsatile, thus AC). Together with different absorptive capacities of haemoglobin (high absorption) versus other tissues, captured data produces photoplethysmographic waveforms from which tissue perfusion can be evaluated. Photoplethysmography devices can measure both urethral and gastric perfusion. Whilst earlier gastrointestinal mucosal monitoring devices have shown promise, further studies evaluating urethral plethysmography performance are required.

Urinary PO_2

Identifying acute kidney injury (AKI) during the initiation or extension phases would allow prompt treatment and may improve patient outcomes. To achieve early identification, biomarkers are currently the only viable means under investigation, but there is increasing interest in urinary PO_2 as an early marker of renal hypoxia/ischemia. Pioneer studies demonstrating correlation between blood flow and urinary PO_2 have recently been joined by a validation study that highlighted the limitations of such a device when urine production slows, a common complication in critically ill patients.

Conclusion

The development and validation of monitoring tools to improve evaluation of tissue perfusion can be combined with existing methods to greatly improve our care of critically ill patients with severe hemodynamic disturbances. Further research is required in companion animals before these and other tools can be routinely used at the bedside.

References:

Sakr Y, Dubois MJ, De Backer D, Creteur J, Vincent JL. Persistent microcirculatory alterations are associated with organ failure and death in patients with septic shock. Crit Care Med [Internet]. 2004 Sep;32(9):1825–31. Available from: http://dx.doi.org/10.1097/01.ccm.0000138558.16257.3f

Tachon G, Harrois A, Tanaka S, Kato H, Huet O, Pottecher J, et al. Microcirculatory alterations in traumatic hemorrhagic shock. Crit Care Med [Internet]. 2014 Jun;42(6):1433–41. Available from: http://dx.doi.org/10.1097/CCM.0000000000000223

Schey BM, Williams DY, Bucknall T. Skin temperature and core-peripheral temperature gradient as markers of hemodynamic status in critically ill patients: a review. Heart Lung [Internet]. 2010 Jan;39(1):27–40. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0147956309000946

Guven G, Hilty MP, Ince C. Microcirculation: Physiology, Pathophysiology, and Clinical Application. Blood Purif [Internet]. 2020;49(1-2):143–50. Available from: http://dx.doi.org/10.1159/000503775

Jacquet-Lagrèze M, Magnin M, Allaouchiche B, Abrard S. Is handheld video microscopy really the future of microcirculation monitoring? Crit Care [Internet]. 2023 Sep 12;27(1):352. Available from: https://ccforum.biomedcentral.com/articles/10.1186/s13054-023-04642-z

Ince C, Boerma EC, Cecconi M, De Backer D, Shapiro NI, Duranteau J, et al. Second consensus on the assessment of sublingual microcirculation in critically ill patients: results from a task force of the European Society of Intensive Care Medicine. Intensive Care Med [Internet]. 2018 Mar;44(3):281–99. Available from: http://dx.doi.org/10.1007/s00134-018-5070-7

Bruno RR, Wollborn J, Fengler K, Flick M, Wunder C, Allgäuer S, et al. Direct assessment of microcirculation in shock: a randomized-controlled multicenter study. Intensive Care Med [Internet]. 2023 Jun;49(6):645–55. Available from: https://link.springer.com/10.1007/s00134-023-07098-5

Vallée F, Mateo J, Dubreuil G, Poussant T, Tachon G, Ouanounou I, et al. Cutaneous ear lobe Pco_2 at 37°C to evaluate microperfusion in patients with septic shock. Chest [Internet]. 2010 Nov;138(5):1062–70. Available from: http://dx.doi.org/10.1378/chest.09-2690

Endo Y, Hirokawa T, Miyasho T, Takegawa R, Shinozaki K, Rolston DM, et al. Monitoring the tissue perfusion during hemorrhagic shock and resuscitation: tissue-to-arterial carbon dioxide partial pressure gradient in a pig model. J Transl Med [Internet]. 2021 Nov 14;19(1):390. Available from: http://dx.doi.org/10.1186/s12967-021-03060-5

Coutrot M, Dudoignon E, Joachim J, Gayat E, Vallée F, Dépret F. Perfusion index: Physical principles, physiological meanings and clinical implications in anaesthesia and critical care. Anaesth Crit Care Pain Med [Internet]. 2021 Dec;40(6):100964. Available from: http://dx.doi.org/10.1016/j.accpm.2021.100964

Jacquet-Lagrèze M, Bonnet-Garin JM, Allaouchiche B, Vassal O, Restagno D, Paquet C, et al. A new device for continuous assessment of gut perfusion: proof of concept on a porcine model of septic shock. Crit Care [Internet]. 2014 Jul 16;18(4):R153. Available from: http://dx.doi.org/10.1186/cc13992

Cardinali M, Magnin M, Bonnet-Garin JM, Paquet C, Ayoub JY, Allaouchiche B, et al. A new photoplethysmographic device for continuous assessment of urethral mucosa perfusion: evaluation in a

porcine model. J Clin Monit Comput [Internet]. 2021 May;35(3):585–98. Available from: http://dx.doi.org/10.1007/s10877-020-00515-w

Kainuma M, Kimura N, Shimada Y. Effect of acute changes in renal arterial blood flow on urine oxygen tension in dogs. Crit Care Med [Internet]. 1990 Mar;18(3):309–12. Available from: http://dx.doi.org/10.1097/00003246-199003000-00013 14. Lofgren LR, Hoareau GL, Kuck K, Silverton NA. Noninvasive and Invasive Renal Hypoxia Monitoring in a Porcine Model of Hemorrhagic Shock. J Vis Exp [Internet]. 2022 Oct 28;(188). Available from: http://dx.doi.org/10.3791/64461

Main Stream, Saturday 1 June 2024

EMERGENCY MANAGEMENT OF STATUS EPILEPTICUS AND CLUSTER SEIZURES

Emil Olsen [1, 2]

[1] IVC Evidensia Specialistdjursjukshuset Strömsholm, Neurology, Strömsholm, Sweden
[2] SLU, Department of Clinical Sciences, Neurology, Uppsala, Sweden

Learning objectives:

- Differentiate convulsive status epilepticus from seizure look-a-likes

- Understand the pathophysiology of convulsive status epilepticus

- Understand the changes in excitatory and inhibitory receptors with prolonged convulsive status epilepticus

- Feel comfortable choosing anti-seizure medications for a patient presenting with convulsive status epilepticus

Proceeding:

Status epilepticus is a lethal condition that may result in hypoxia and, if lasting longer than 30 minutes, results in permanent damage to neurons. In one referral animal hospital population, the mortality in dogs being presented with status epilepticus was reported to be 25-39%. This talk will focus on the practical implementation of the ACVIM consensus statement on managing status epilepticus and cluster seizures from general practice to referral hospitals.

Epileptic seizures are *hypersynchronous* and often self-limiting with a duration of 1 to 3 minutes on average. Generalised seizures involve both cerebral hemispheres. The most common form is tonic-clonic seizure but may also be purely tonic, clonic or even myoclonic. Focal seizures are limited to one hemisphere and have focal motor activity, autonomic or even behavioural characteristics. Focal seizures can develop into generalised seizures. Reactive seizures look similar to epileptic seizures but are triggered by a toxin or metabolic disturbance and are often reversible. In contrast, syncope, paroxysmal dyskinesia and idiopathic head tremor do not have epileptic seizure activity. When you obtain the history or observe an episode, attempt to characterise it according to the above definitions. If the patient is not actively seizing, it will help you correctly classify the type of episode.

The phases of a seizure are prodrome, ictus and post-ictus. Prodrome potentially helps caregivers detect upcoming seizures where 60% of owners in one study believed they could predict a seizure by the behavioural changes in hours to days before the seizure. Ictus is where the seizure activity is visible, and the phase after ictus, the postictal phase, is where regular brain activity is restored. Because a seizure is located in the forebrain, the postictal phase is characterised by forebrain signs such as disorientation,

compulsive walking, ataxia and blindness. These postictal signs for half of the dogs last for 1 to 30 mins for 20% of the dogs up to 1 hour and the remainder over 1 hour, most of which are less than 2 hours.

Status epilepticus is pragmatically defined as 1) a prolonged seizure that lasts for more than 5 minutes of continuous seizure activity or 2) two or more seizures without recovery of consciousness between the seizures. For both 1 and 2 the normal mechanisms to terminate a seizure fail. Cluster seizures are two or more self-limiting seizures in 24 hours. Once the initial anti-seizure medications are given without cessation of the seizure and the seizure has lasted for over 30 minutes, it is classified as refractory to medication. This is also the time point where neuronal injury begins. The longer a convulsive seizure persists, the greater the metabolic consequences and demand for energy, resulting in hyperlactatemia and acidosis. After 20-40 minutes, cardiovascular compensatory mechanisms fail, resulting in a decline in blood pressure, further causing decreased cerebral perfusion and oxygenation and a decrease in available glucose. During a prolonged seizure over 20-40 minutes, excitatory glutamate receptors (primarily NMDA) are upregulated. Simultaneously to increased excitation, the inhibitory GABA-A receptors are internalised, resulting in a lack of response to medications targeting the GABA-A receptors (benzodiazepines; diazepam and midazolam and barbiturates).

First-line treatments with intravenous (IV) catheter.

Seizure duration: 5-10 minutes.

Midazolam IV or intranasal (IN) (with mucosal administration device(MAD)):0,2mg/kg, can be repeated twice at a minimum of 2-minute intervals. If the seizures stop but relapse within 10 min, a third dose is followed by a continuous rate infusion (CRI) of 0,2-0,5 mg/kg/hour).

Second-line treatment**.**

If seizures continue 10 – 30minutes:

Add levetiracetam 60mg/kg IV followed by 30mg/kg q8h

If phenobarbital-naive: Add phenobarbital 3 - 5mg/kg IV every 20 mins or when the patient seizures to achieve a maximum dose of 20mg/kg.

If not phenobarbital naive: Obtain phenobarbital serum levels, then decide on dosage.

Add fosphenytoin IV 15mg/kg (dogs only)

Third-line treatment.

If seizures continue >30minutes:

Ketamine 5mg/kg IV followed by a CRI 0.1-0.5mg/kg/hour, increase CRI until termination of seizure activity.

Add dexmedetomidine 0.5ug/kg followed by CRI 0.5-3.0mcg/kg/h increase until termination of seizure activity.

If there is no response, propofol IV: 1-6mg/kg bolus for induction followed by 0.1-0.6mg/kg/min CRI (caution with cats!)

References:

Raina N, Yadav M, Rani R, Ojha B, Bhardwaj BY, Gupta M. Status epilepticus: An overview for neuroscientists. Curr Pharmacol Rep. 2022;8: 36–47.

Charalambous M, Muñana K, Patterson EE, Platt SR, Volk HA. ACVIM Consensus Statement on the management of status epilepticus and cluster seizures in dogs and cats. J Vet Intern Med. 2024;38: 19–40.

Berendt M, Farquhar RG, Mandigers PJJ, Pakozdy A, Bhatti SFM, De Risio L, et al. International veterinary epilepsy task force consensus report on epilepsy definition, classification and terminology in companion animals. BMC Vet Res. 2015;11: 182.

Finnegan SL, Volk HA, Asher L, Daley M, Packer RMA. Investigating the potential for seizure prediction in dogs with idiopathic epilepsy: owner-reported prodromal changes and seizure triggers. Vet Rec. 2020;187: 152.

Kähn C, Meyerhoff N, Meller S, Nessler JN, Volk HA, Charalambous M. The Postictal Phase in Canine Idiopathic Epilepsy: Semiology, Management, and Impact on the Quality of Life from the Owners' Perspective. Animals (Basel). 2023;14. doi:10.3390/ani14010103

Khoueiry M, Alvarez V. Status epilepticus in adults: a clinically oriented review of etiologies, diagnostic challenges, and therapeutic advances. Clinical Epileptology. 2023;36: 288–297.

OSMOTHERAPY IN BRAIN OEDEMA - CURRENT EVIDENCE OF PRACTICE

Heidi Saarenkari [1]

[1] Utrecht University, Utrecht University, Faculty of Veterinary Medicine, Utrecht, Netherlands

Learning objectives:

- Know the pathophysiology and clinical signs of increased intracranial pressure

- Understand the mechanism of action and adverse effects of mannitol and hypertonic saline

- Review the relevant human literature and guidelines

Proceeding:

Cerebral oedema is a pathological focal or diffuse swelling of the brain where fluid accumulates either within the brain cells or in the interstitial space of the brain. Cerebral oedema results from localized inflammation, vascular changes, disrupted cellular metabolism or disturbance of the blood-brain barrier. Brain oedema and increased intracranial pressure can occur secondary to traumatic brain injury, inflammatory or infectious brain disease, status epilepticus, intracranial neoplasia, intracranial haemorrhage, ischemic stroke or hepatic encephalopathy. Cerebral blood flow depends on cerebral perfusion pressure (CPP). CPP is calculated as mean arterial pressure (MAP) minus intracranial pressure (ICP). The Monro-Kellie doctrine states that the skull is a rigid structure occupied by brain parenchyma, cerebrospinal fluid, and blood. As the skull cannot expand, an increase in one of these components must be accompanied by a decrease in one or both of the other components to maintain the same intracranial volume. In human medicine, intracranial pressure monitoring is recommended to guide therapy, however, in veterinary medicine, intracranial pressure monitoring is not generally used and the diagnosis of increased intracranial pressure is based on neurological examination. Clinical signs of increased intracranial pressure include deteriorating neurological status, Cushing's reflex and papilledema. Modified Glasgow Coma Score can be used in assessing the progress of the neurological status of a head trauma patient. Hyperosmolar agents mannitol and hypertonic saline are both used in an attempt to reduce increased intracranial pressure. Mannitol is a sugar alcohol that comes as a 10% or 20% solution. Mannitol lowers increased intracranial pressure via two different mechanisms: Initially (within minutes) there is a plasma expansion that reduces plasma viscosity leading to improved blood flow in the cerebrum. Compensatory vasoconstriction develops in the brain areas with intact autoregulation, which reduces intracranial pressure. Mannitol also reduces cerebral oedema by creating an osmotic gradient between plasma and brain, drawing water from the brain parenchyma into the vasculature. Known adverse effects of mannitol are hypernatraemia, metabolic acidosis, hypovolaemia, hypotension, renal failure, allergic reactions and rebound increase in intracranial pressure. In a similar way to mannitol,

hypertonic saline increases the osmotic gradient between the brain and the blood, pulling water from the brain parenchyma to the intravascular space. It also improves cardiac output and intravascular volume and has beneficial vasoregulatory and immunologic effects. Potential adverse effects include osmotic demyelination syndrome or central pontine myelinolysis, renal insufficiency, haematological abnormalities, fluid overload, electrolyte abnormalities and rebound increased intracranial pressure. An ideal hyperosmotic agent in traumatic brain injury should simultaneously lower ICP and maintain or improve CPP given the brain's autoregulation may be lost in the injured brain. Mannitol is typically considered the gold standard medical treatment for increased intracranial pressure in people. There are several studies published in people comparing the effect of mannitol and hypertonic saline mainly in cases of traumatic brain injury, however, the heterogeneity of the studies and small patient numbers make drawing definitive conclusions difficult. Studies comparing these agents in other conditions causing increased intracranial pressure are sparse. Osmolar loads must be similar to make a valid comparison between hypertonic saline and mannitol groups. Most of the studies that compared similar osmolar loads of hypertonic saline and mannitol in traumatic brain injury patients found that both agents reduced increased intracranial pressure, but hypertonic saline had a more pronounced and prolonged effect compared to mannitol. There is also some evidence of better improvement of cerebral perfusion pressure in patients treated with hypertonic saline. Hypertonic saline may also have a better safety profile, which may be an important factor when selecting a hyperosmolar agent. The Brain Trauma Foundation's Guidelines for the Management of Severe Traumatic Brain Injury do not give any recommendations for the use of hyperosmolar agents in traumatic brain injury given the lack of concrete evidence to suggest one over the other. A recent European-wide study showed that trauma centre preference for hyperosmolar agent was a bigger driver than patient characteristics when selecting which agent to use. Given the lack of concrete evidence on what hyperosmolar agent to use in which patients, a multicentre randomized trial of mannitol and hypertonic saline in severe traumatic brain injury and intracranial hypertension is currently ongoing in the UK to try to address this debate. Unfortunately, to date, there are no veterinary studies comparing the efficacy of mannitol and hypertonic saline in increased intracranial pressure.

References:

Kimelberg HK. Current concepts of brain edema: Review of laboratory investigations. J Neurosurg 1995;83:1051–1059. Shawkat H, Westwood M-M, Mortimer A. Mannitol: a review of its clinical uses. Contin Educ Anaesth Crit Care Pain 2012;12:82–85.

Tyagi R, Donaldson K, Loftus CM et al. Hypertonic saline: a clinical review. Neurosurg Rev 2007;30:277–290.

Carney N, Totten AM, O'Reilly C et al. Guidelines for the Management of Severe Traumatic Brain Injury, Fourth Edition. Neurosurgery 2017;80:6–15.

Cook AM, Jones GM, Hawryluk GWJ et al. Guidelines for the Acute Treatment of Cerebral Edema in Neurocritical Care Patients. Neurocritical Care 2020;32:647–666.

Veen E van, Nieboer D, Kompanje EJO et al. Comparative Effectiveness of Mannitol Versus Hypertonic Saline in Patients With Traumatic Brain Injury: A CENTER-TBI Study. J Neurotrauma 2023;40:1352–1365.
Huang X, Yang L, Ye J et al. Equimolar doses of hypertonic agents (saline or mannitol) in the treatment of intracranial hypertension after severe traumatic brain injury. Medicine 2020;99:e22004.

Battison C, Andrews PJD, Graham C et al. Randomized, controlled trial on the effect of a 20% mannitol solution and a 7.5% saline/6% dextran solution on increased intracranial pressure after brain injury. Crit Care Med 2005;33:196–202.

Francony G, Fauvage B, Falcon D et al. Equimolar doses of mannitol and hypertonic saline in the treatment of increased intracranial pressure. Crit Care Med 2008;36:795–800. S

u Y, Liu Y, Chen Z et al. Comparison of equiosmolar doses of 10% hypertonic saline and 20% mannitol for controlling intracranial hypertention in patients with large hemispheric infarction. Clin Neurol Neurosurg 2021;200:106359.

Cottenceau V, Masson F, Mahamid E et al. Comparison of Effects of Equiosmolar Doses of Mannitol and Hypertonic Saline on Cerebral Blood Flow and Metabolism in Traumatic Brain Injury. J Neurotrauma 2011;28:2003–2012.

Patil H, Gupta R. A Comparative Study of Bolus Dose of Hypertonic Saline, Mannitol, and Mannitol Plus Glycerol Combination in Patients with Severe Traumatic Brain Injury. World Neurosurg 2019;125:e221–e228.

Jagannatha AT, Sriganesh K, Devi BI et al. An equiosmolar study on early intracranial physiology and long term outcome in severe traumatic brain injury comparing mannitol and hypertonic saline. J Clin Neurosci 2016;27:68–73.

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. Intensiv Care Med 2009;35:471–479.

Sakellaridis N, Pavlou E, Karatzas S et al. Comparison of mannitol and hypertonic saline in the treatment of severe brain injuries: Clinical article. J Neurosurg 2011;114:545–548.

Oddo M, Levine JM, Frangos S et al. Effect of mannitol and hypertonic saline on cerebral oxygenation in patients with severe traumatic brain injury and refractory intracranial hypertension. J Neurol, Neurosurg Psychiatry 2009;80:916.

Kumar SA, Devi BI, Reddy M et al. Comparison of equiosmolar dose of hyperosmolar agents in reducing intracranial pressure—a randomized control study in pediatric traumatic brain injury. Child's Nerv Syst 2019;35:999–1005.

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

DISORDERS OF AMBULATION IN THE ER, PART I: OBSERVATION, EXAM NUANCES AND SORTING OUT SYSTEMS

Christopher Mariani [1]

[1] North Carolina State University, Clinical Sciences, Raleigh, United States

Learning objectives:

- Describe the utility and key components of a gait examination, and the main abnormalities that might be observed

- Describe the different types of ataxia

- List the components of the neurological examination most important to assessment of these patients

- Given an animal with a gait abnormality, localize the problem to the appropriate body system and, if applicable, region of the nervous system

Proceeding:

Small animals commonly present to the emergency room for difficulty walking and these difficulties can take a variety of forms. Observation of the animal and the gait examination are paramount in confirming where a problem with ambulation is originating from and in localizing it to the correct region of the nervous system or alternatively, to another body system (e.g., orthopaedic). Identifying the body system and anatomic structures responsible are critical to recommending subsequent diagnostic testing, therapy, and possibly referral to the appropriate specialist. The most critical steps in this evaluation are careful observation of the animal's gait, and the neurologic and orthopaedic examinations.

Gait Examination

Gait examination is arguably the most important part of the evaluation of patients with ambulation difficulties, but it is also the portion of the exam that is most often neglected by veterinary practitioners. Honing one's observational skills are a key early step in the successful management of these patients. The animal should be evaluated while walking towards and away from the examiner and should also be observed from the side. If the animal is unable to stand or bear weight, adequate support of the limbs in question should be provided while assessing the ability of the animal to voluntarily advance its limbs, bear weight, and move in a coordinated manner. Several abnormalities may be detected during the gait examination. These include:

Ataxia: incoordination characterized by a failure to walk or move the limbs in a straight line, crossing of the limbs over the body midline, and possibly stumbling and falling. Ataxia indicates neurologic dysfunction and may be caused by involvement of several areas of the nervous system.

Sensory (proprioceptive) ataxia: Lesions of the peripheral sensory nerves, spinal cord or brainstem commonly cause incoordination. Spinal cord and brainstem lesions are typically accompanied by paresis (see below). Peripheral sensory nerve lesions are very rare in veterinary patients.

Cerebellar ataxia: Cerebellar lesions can cause a profound ataxia characterized by dysmetria (hypermetria and hypometria), and intention tremors. Animals with pure cerebellar lesions maintain good strength without obvious paresis.

Vestibular ataxia: Characteristic incoordination typified by leaning, drifting, stumbling, falling, and occasionally rolling to one side. Frequently accompanied by a head tilt, nystagmus, and possibly positional ventral strabismus.

Paresis: incomplete voluntary movement, or muscular weakness. On the gait exam, this is characterized by scuffing of the nails, dragging of one or more limbs, a short-strided gait, or rapid tiring with activity/exercise. Paresis denotes dysfunction of the nervous (motor) or muscular systems.

Lameness: Inability or reluctance to bear weight on one or more limbs. Lameness often indicates a lesion in the long bones, joints, tendons, or musculature (i.e., orthopaedic disease), although entrapment or compression of a nerve or nerve root can also lead to lameness (known as a “root signature”).

Orthopaedic Examination

Orthopaedic examination should include careful examination of the affected and normal limbs, evaluating the long bones, joints, tendons and musculature. Joints are palpated for evidence of effusion and pain, and flexed and extended for evidence of instability, excessive or restricted motion, and pain. Long bones and tendons are palpated for evidence of swelling or pain. Muscles are palpated to assess tone, atrophy or hypertrophy, and for evidence of pain.

Neurologic Examination

A thorough neurologic examination is an important part of the evaluation of animals with gait dysfunction. However, particular attention should be paid to gait examination (see above), assessment of postural reactions, segmental spinal and brainstem reflexes, and muscular and spinal palpation.

Postural reactions: These include tests such as proprioceptive placing (“conscious proprioception”), hopping, hemistanding, hemiwalking, wheelbarrowing, and the extensor postural thrust. A convincing postural reaction deficit definitively indicates a lesion within the nervous system.

Segmental spinal and brainstem reflexes: Convincing reflex deficits indicate dysfunction of the peripheral or cranial nerves, associated brainstem or spinal cord segments, or neuromuscular junction.

Muscular and spinal palpation: Palpation of the spine for evidence of pain or discomfort is an important part of the examination. Pain may be a sign of a lesion affecting the spine itself or referred from adjacent

nervous system or muscular structures (e.g., nerve roots, meninges). Assessment of muscles for pain or atrophy is also important.

DISORDERS OF AMBULATION IN THE ER, PART II: DIAGNOSTIC AND THERAPEUTIC MANAGEMENT

Christopher Mariani [1]

[1] North Carolina State University, Clinical Sciences, Raleigh, United States

Learning objectives:

- Given a dog or cat with a complaint of difficulty walking, generate a list of differential diagnoses for the patient

- Describe the utility of spinal radiographs for these patients and discuss the radiographic findings expected for intervertebral disk disease, diskospondylitis, spinal trauma and spinal tumours

- Given a dog or cat with a complaint of difficulty walking, generate a diagnostic plan for the patient in the emergency room setting

- Discuss the indications, likely diagnostic testing options and benefits of referral for animals with disorders of ambulation

Proceeding:

This talk will build upon the concepts introduced in the preceding talk. After sorting out a likely body system and (potential) region of the nervous system, emergency clinicians need to develop a list of potential differential diagnoses and usually take at least some preliminary steps in ruling in or ruling out these disease processes. We will focus on practical diagnostic testing that can be performed in a standard ER environment, deciding which cases would ideally benefit from more specialized diagnostics (and possible referral) and finally, therapy within the emergency environment.

Generation of a Differential Diagnosis

When generating a list of differential diagnoses, it is critical to take several aspects of the patient and their presentation into account. These include the signalment, the onset and progression of clinical signs, the presence of pain (on spinal palpation), and any lateralization of the signs noted.

The region of the nervous system affected, as defined by the neurological examination and subsequent neurolocalization, is another important factor in compiling potential etiologies. Although there are some disorders that can occur anywhere along the spine, others are restricted to specific areas (e.g., atlantoaxial subluxation, lumbosacral disease), while others may have a predilection for certain areas (e.g., diskospondylitis).

Diagnostic Testing

Diagnostic tests chosen depend on the results of the gait, orthopedic, and neurologic examinations, index of suspicion of disease, severity of presentation and financial constraints of the owner.

Orthoapedic disease

Radiographic examination of the appendicular skeleton is a useful and relatively low-cost diagnostic test to identify a number of disease processes. In some cases, advanced diagnostic imaging such as radionucleotide imaging (bone scan), computed tomography (CT), or magnetic resonance imaging (MRI) may provide additional information and are more sensitive than survey radiography. Collection of a sample of joint fluid is usually diagnostic for polyarthritis.

Neurological or neuromuscular disease

Survey radiographs of the spine are useful to detect diskospondylitis, intervertebral disk disease, and lytic vertebral tumors. CT and MRI are frequently used to identify spinal cord compression or inflammation secondary to intervertebral disk disease, meningomyelitis, central or peripheral nervous system neoplasia and vertebral tumours. Cerebrospinal fluid (CSF) analysis is used to document inflammation within the CNS. Electrodiagnostic testing (e.g., electromyography [EMG], nerve conduction velocity, repetitive nerve stimulation, spinal evoked potentials) is particularly useful in documenting evidence of peripheral nerve or neuromuscular disease, and occasionally myopathic diseases. Muscle biopsy with or without nerve biopsy is effective in confirming neuropathic or myopathic disease and in further defining potential aetiologies.

Therapeutic Intervention

A discussion of treatment of all the conditions outlined in this talk is outside the scope of these proceedings. However, a few relevant points of therapy can be highlighted. Directly addressing the underlying disease process is the ideal goal of any therapeutic strategy, and may involve surgical correction (e.g., cranial cruciate ligament repair, hemilaminectomy for intervertebral disk removal) or medical therapy (e.g., immunosuppression in animals with polyarthritis). Where correction of the underlying condition is not possible, palliation of the signs associated with the disease (often pain) is warranted.

Opioids and nonsteroidal anti-inflammatory drugs (NSAIDs) are often effective against the acute pain associated with trauma or operative interventions. NSAIDs are frequently used with chronic orthopaedic conditions and although they can provide some relief in animals with neuropathic pain, this drug class is often not the most effective in this scenario. Muscle relaxants (e.g., diazepam or methocarbamol) are useful in animals with intervertebral disk disease or neoplasia causing nerve root irritation, as much of the pain in these conditions is associated with muscle spasm. Gabapentin and pregabalin are some of the most effective drugs for neuropathic pain, and act through inhibition of voltage-gated calcium channels. Finally, the NMDA receptor antagonists ketamine and amantadine can be useful in acute and chronic scenarios respectively for the control of the “wind-up” phenomenon. A multimodal therapeutic strategy using several drugs and other interventions often works best for animals with significant pain as part of their clinical presentation.

DISORDERS OF AMBULATION IN THE ER, PART III: VIDEO CASE EXTRAVAGANZA!

Christopher Mariani [1]

[1] North Carolina State University, Clinical Sciences, Raleigh, United States

Learning objectives:

- Discuss various presentations for animals with ambulation disorders and how these relate to likely underlying etiologies

- Select appropriate diagnostics for different case presentations and describe their utility

- Generate a diagnostic plan for a case presenting with ambulation difficulties

- Generate a therapeutic plan for a case presenting with ambulation difficulties

Proceeding:

In this talk, a series of cases will be presented, including signalments, presenting complaints and examination videos. We will work our way through these cases together, discussing exam abnormalities, likely differential diagnoses, and appropriate diagnostic and therapeutic management, utilizing the concepts presented in the first two sessions. Results from diagnostic testing will also be presented. Audience participation will be a key part of this session, so come prepared to contribute and have some fun!

Clinical Presentations

Most clinical cases will fit into one of the presentations and disease categories listed below.

Acute lameness

Acute lameness is most often associated with orthopaedic disease. Evidence of joint or soft tissue swelling or effusion, long bone fracture, or joint instability on orthopaedic examination increases the index of suspicion or confirms the diagnosis. However, a neurologic aetiology is possible, and is seen most often as a "root signature" secondary to nerve root compression by a herniated intervertebral disk. Neck or back pain, ataxia and paresis suggest nervous system involvement.

<u>Orthopedic disorders</u>

Ligament tear or rupture (e.g. cranial cruciate). Muscle strain. Long bone fracture. Panosteitis. Hypertrophic osteodystrophy.

<u>Neurologic disorders</u>

Intervertebral disk disease. Lumbosacral stenosis

Chronic lameness

Chronic lameness is also most often orthopaedic in nature, and the aetiology is confirmed by similar means. However, neurologic aetiologies can certainly lead to similar gait dysfunction. A chronic lameness (particularly involving a thoracic limb) that is unresponsive to NSAIDs should raise concern for a peripheral nerve sheath tumour.

Orthopedic disorders

Osteoarthritis. Ligament injury. Bone neoplasia. Neurologic disorders.

Peripheral nerve sheath tumor. Intervertebral disc disease. Lumbosacral disease.

Acute ataxia & paresis

Although paresis can occur secondary to myopathic disorders, ataxia signifies involvement of the nervous system. These two gait abnormalities frequently occur together with disorders involving the spinal cord or brainstem.

Spinal cord disorders

Intervertebral disc disease. Meningomyelitis. Neoplasia (vertebrae, meninges, nerve roots, etc.) Vascular myelopathies. Diskospondylitis. Atlantoaxial subluxation. Cervical spondylomyelopathy (Wobbler syndrome). Congenital anomalies. Spinal cord trauma. Brainstem disorders

Meningoencephalitis. Neoplasia. Stroke

Acute ataxia without paresis

It is possible for many of the spinal cord disorders to present with ataxia as the sole or predominant clinical sign. Disorders of other regions of the nervous system may lead to ataxia as the primary gait abnormality, including peripheral vestibular or cerebellar lesions.

Spinal cord disorders

Similar to those listed above

Peripheral vestibular disorders

Otitis media-interna. Idiopathic vestibular syndrome(s) Neoplastic disease involving the vestibular nerve or inner ear. . OtotoxicosisCerebellar disorders

Similar to brainstem disorders (above)

Acute paresis without ataxia

Again, although they usually occur together, it is possible for spinal cord lesions listed above to show a predominant paresis without obvious ataxia; it may also be difficult to appreciate ataxia in animals that are recumbent. However, scenarios where the paresis is truly without accompanying ataxia can occur,

such as in the caudal lumbar spine/lumbosacral region (caudal to the termination of the spinal cord, where disease affects the nerve roots) or in diseases that affect the neuromuscular system (motor unit: peripheral nerve, neuromuscular junction and muscle).

Caudal lumbar or lumbosacral disorders

Lumbosacral disease.

Many of etiologies listed above under spinal cord disorders

Neuromuscular disorders

Acute polyradiculoneuritis. Botulism. Tick paralysis (regional). Snake envenomation (regional)

Stiff, short-strided gait

A stiff, short-strided gait may occur in single or multiple limbs due to either pain or paresis. A number of etiologies can potentially cause this presentation, and careful orthopaedic and neurologic examinations are required to sort these out.

Orthopaedic disorders

Polyarthritis. Panosteitis. Hypertrophic osteodystrophy. Neurologic/neuromuscular disorders

Myasthenia gravis (pelvic limbs or all four limbs) Polymyopathy. . Polyneuropathy. Lumbosacral disease (pelvic limbs only). C6-T2 lesion (IVDD, cervical spondylomyelopathy; thoracic limbs only)

Advanced Stream, Saturday 1 June 2024

ERAS® FOR VETERINARY EMERGENCY AND CRITICAL CARE: TIME FOR ACTION

Angela Briganti [1]

[1] Department of Veterinary Sciences, University of Pisa, Pisa, Italy

Learning objectives:

- The lecture will describe the cornerstones of the ERAS® approach and the methods used to build a specific ERAS® protocol for veterinary emergency laparotomy surgery and the results obtained by its use. The lecture will also focus on the difficulties encountered in the application of the ERAS® protocols in 2 different clinical settings.

Proceeding:

ERAS®

The Enhanced Recovery After Surgery (ERAS®) program is a multimodal perioperative care pathway composed of evidence-based interventions to optimize perioperative management of patients undergoing major surgical procedures. ERAS® has been developed over the past 20 years in human medicine and its main objective is to improve patient outcomes after surgery.

ERAS® objective are to maximize pain control, optimize fluid balance and minimize perioperative metabolic stress thereby accelerating recovery after surgery and decreasing convalescence time. Application of ERAS® protocols in surgery patients has demonstrated a reduction in hospitalization length and postoperative complications rate in different kind of surgeries. ERAS® protocols have also been used in emergency patients and have resulted in a faster postoperative return to normal function and lower risk of overall complications.

Emergency abdominal surgery is a common procedure in both veterinary and human medicine. In human medicine, emergency surgery accounts for 11-15% of all total surgeries, however, it is responsible for almost 50% of all postoperative deaths and 30% of all surgical complications.

How to insert an ERAS® protocol in the clinical activity? The first key point of the ERAS® is the multidisciplinary approach and the construction of specific pathways for specific procedures or patients. Having a pathway with defined measurable components facilitates improvement and allows comparison of processes and outcomes between different practitioners and centers. An important component for improving care is mortality review which is a standard part of any audit and quality improvement programs.

The systematic evaluation of the specific risks of some patients or procedures is fundamental to build a personalized ERAS® protocol. Using a structured approach to review all laparotomy deaths can provide

thematic insights into system, teamwork and communication issues and enable sharing and categorization of harming events, and development of areas of improvement. Then, the approval of the protocol by all the professional figures involved (intensivist, anesthetist, internist, surgeon, critical care nurse, etc.) is crucial to build a protocol that can be easily applied. The use of an ERAS® protocol designed for a different facility can lead to great problems in the feasibility and applicability of the protocol. A key point of the ERAS® protocol is that the pathway needs to be followed entirely, in order to be effective in reducing morbidity and mortality.

The application of the ERAS® protocol involves the establishment of a check list that identifies three different perioperative moments: preoperative, intraoperative and postoperative. Specific end points and actions are set for each phase.

Such an approach has been shown to improve outcomes for very high-risk patients, increasing awareness of the specific risks of critical and emergent patients.

We decided to build a specific ERAS® for laparotomy emergency surgeries. First, we did a retrospective study to identify the perioperative factors associated with death. Then we did a prospective study in which we applied the pathways decided for laparotomic emergency surgeries.

ERAS® significantly improved outcomes in two different hospitals providing evidence of external validity of the use of this approach to reduce mortality and complications after emergency laparotomy in dogs.

References:

Fearon KC, Ljunqvist O, Von Meyenfeldt M, et al. Enhanced recovery after surgery: a consensus review of clinical care for patients undergoing colonic resection. Clin Nutr 2005; 24:466-77

Peden, C. J., Aggarwal, G., Aitken, R. J., Anderson, I. D., Balfour, A., Foss, N. B., Cooper, Z., Dhesi, J. K., French, W. B., Grant, M. C., Hammarqvist, F., Hare, S. P., Havens, J. M., Holena, D. N., Hübner, M., Johnston, C., Kim, J. S., Lees, N. P., Ljungqvist, O., Lobo, D. N., … Scott, M. J. (2023). Enhanced Recovery After Surgery (ERAS®) Society Consensus Guidelines for Emergency Laparotomy Part 3: Organizational Aspects and General Considerations for Management of the Emergency Laparotomy Patient. World journal of surgery, 47(8), 1881–1898. https://doi.org/10.1007/s00268-023-07039-9

Peden, C. J., Aggarwal, G., Aitken, R. J., Anderson, I. D., Bang Foss, N., Cooper, Z., Dhesi, J. K., French, W. B., Grant, M. C., Hammarqvist, F., Hare, S. P., Havens, J. M., Holena, D. N., Hübner, M., Kim, J. S., Lees, N. P., Ljungqvist, O., Lobo, D. N., Mohseni, S., Ordoñez, C. A., … Scott, M. (2021). Guidelines for Perioperative Care for Emergency Laparotomy Enhanced Recovery After Surgery (ERAS) Society Recommendations: Part 1-Preoperative: Diagnosis, Rapid Assessment and Optimization. World journal of surgery, 45(5), 1272–1290. https://doi.org/10.1007/s00268-021-05994-9.

Scott, M. J., Aggarwal, G., Aitken, R. J., Anderson, I. D., Balfour, A., Foss, N. B., Cooper, Z., Dhesi, J. K., French, W. B., Grant, M. C., Hammarqvist, F., Hare, S. P., Havens, J. M., Holena, D. N., Hübner, M., Johnston, C., Kim, J. S., Lees, N. P., Ljungqvist, O., Lobo, D. N., … Peden, C. J. (2023). Consensus Guidelines for Perioperative Care for Emergency Laparotomy Enhanced Recovery After Surgery (ERAS®) Society

Recommendations Part 2-Emergency Laparotomy: Intra- and Postoperative Care. World journal of surgery, 47(8), 1850–1880. https://doi.org/10.1007/s00268-023-07020-6.

PANS : A NEW MEDICAL ENTITY ?

Maxime Cambournac [1]

[1] Centre hospitalier vétérinaire Fregis, Emergency and Critical Care, Gentilly, France

Learning objectives:

- Knowing the clinical entity PANS
- Being able to characterize clinical features
- Being able to identify what differentiates PANS from hepatic encephalopathy
- Listing the different treatments

Proceeding:

Normal detoxification depends on the passage of blood from the intestines into the liver. In the case of a hepatic shunt, blood from the intestines bypasses the liver and flows directly into the systemic circulation. In the past, complete ligation was the treatment of choice of extrahepatic shunts. In recent years, gradual occlusion methods have become popular in an attempt to minimize the risk of perioperative complications. Advances in veterinary medicine now offer new alternatives in the treatment of hepatic shunts, especially with the use of endovascular occlusion in the treatment of intrahepatic shunts. Reported complications after shunt surgery include portal hypertension, hypothermia, thrombosis, and seizures.

Referred by a variety of terms, probably reflecting the variety of clinical presentations, post attenuation neurologic signs (PANS) is a poorly understood and potentially devastating complication after surgical attenuation of congenital portosystemic shunts. With a reported incidence varying from 3 to 8%, this clinical syndrome includes seizures, but also blindness, ataxia, abnormal behavior, tremors and twitching. Initially, PANS were thought to be an evolution or degradation of the initial pre-surgical signs, as it shares some similarity with signs of shunt. As stated by Mullins et al, the refractory nature of PANS in some cases compared with those observed preoperatively suggests an alternative etiology than simply continuation of preoperative seizures associated with hepatic encephalopathy in the early postoperative period[1]. While frequently compared, in cases of PANS, blood glucose, ammonia and electrolytes are normal. Differentiated features of PANS include refractoriness to antiepileptic drugs, and occurrence of seizures when absent before surgical attenuation. Occurrence of new neurologic signs, in the absence of pre-surgical neurological disorders represents around 70% of cases. Among pathophysiological theory, abnormal brain blood flow or preexisting brain disease have been suggested, but not validated yet.

Abnormal intrahepatic vasculature development and electrolyte variation, sometimes within the normal range, are the two mechanisms advocated to date.

As PANS remain a complex medical entity, treatment recommendations are variable, and without a randomized control trial, best options are still unknown. In the largest study, on cats, phenobarbital was the first line of treatment, and the most effective. Among other treatments, the number of patients is too small to draw conclusions at this time. In one case report, a cat has been treated successfully with therapeutic plasma exchange, suggesting homeostasis dysregulation. As prevention is frequently the key, attempting administration of prophylactic antiepileptics remains unsuccessful at this time[2]. While signs might be frequent and require intensive care, survival to discharge has been reported to vary from 25 to 100%. Neurologic outcome at discharge is variable, with half of them presenting persistent sign such as ataxia, blindness, tremors, disorientation, circling or abnormal gaits[3]. In one study in dogs, signs mostly resolved within a 6-month period post-discharge.

References:

Mullins RA, Creheut TS. Postattenuation neurologic signs after surgical correction of congenital portosystemic shunts in cats: A narrative review. Vet Surg 2023; 52: 349–360.

Carrera AE, Morrissey AM, Lipscomb VJ, et al. Long-term outcome and quality of life of dogs that developed neurologic signs after surgical treatment of a congenital portosystemic shunt: 50 cases (2005–2020). J Am Vet Méd Assoc 2022; 260: 326–334.

Economu L, Strickland R, Lipscomb VJ. Surgical treatment and outcome of intrahepatic shunts in 12 cats. J Feline Med Surg 2022; 24: e411–e419.

VETERINARY ARDS AND REFRACTORY HYPOXEMIA- A REVIEW

Anusha Balakrishnan [1]

[1] Veterinary Emergency Group, Veterinary Emergency Group, White Plains, United States

Learning objectives:

The goal of this session is to briefly review the pathophysiology of acute lung injury and ARDS as well as the updated nomenclature in humans with the Berlin definitions. This will be followed by a brief review of current veterinary evidence and efforts to develop updated veterinary definitions. The last section of this talk will focus on refractory hypoxemia and some strategies including novel modes of ventilation to address this.

Proceeding:

Acute respiratory distress syndrome (ARDS) is a devastating clinical condition documented in both humans and veterinary patients that is associated with a per-acute onset of respiratory distress. ARDS is characterized by severe hypoxemia and bilateral diffuse alveolar infiltrates seen on thoracic radiographs that are not caused by left atrial hypertension or hydrostatic pulmonary edema.

Most recently, the Berlin Definition of ARDS was proposed in people and major changes represented with these definitions included the addition of a definitive time frame for onset of clinical signs, removal of the criterion for pulmonary artery wedge pressure, and addition of a minimum level of positive end-expiratory pressure (PEEP) as well as mutually exclusive thresholds of PaO2/FiO2 to stratify patients based on the severity of ARDS (mild, moderate, and severe). The term “Acute Lung Injury” was removed from these definitions and is no longer used in people.

Etiology and Pathogenesis

The etiology of ARDS in small animals is equally multifactorial, and several risk factors, including aspiration pneumonia, sepsis, SIRS, trauma, smoke or aerosolized toxin inhalation, near-drowning and pancreatitis have all been reported in small animals.

ARDS represents a vicious cycle of lung injury that begins with an acute insult (either pulmonary or extrapulmonary) that causes activation of alveolar macrophages. Release of cytokines and chemokines by the alveolar macrophages causes a further inflammatory cascade by activating circulating neutrophils and an ongoing release of inflammatory mediators. This cascade, while intended to address the primary insult or pathogen, also has the effect of causing injury to the alveolar-epithelial barrier. Perhaps most importantly, the alveolar type-II pneumocytes that secrete surfactant and play a vital role in ion-transport mediated clearance of fluid, are damaged, effectively causing denudation of the alveolar

basement membrane. This ultimately leads to flooding of the alveolar spaces with protein-rich fluid, due to the loss of the normal mechanisms limiting lung permeability. This fluid accumulation represents the exudative phase of ARDS and sets the stage for progressive alveolar injury, collapse and derecruitment, and ultimately, a precipitous decline in gas exchange. In most small animals diagnosed with ARDS, this is the stage that represents the highest likelihood of mortality or humane euthanasia, often due to the magnitude of intensive respiratory support that is necessary during this time.

The next stage of the pathogenesis of ARDS in animals that survive the exudative phase represents the proliferative phase. This is the stage where pathogens and damaged host cells are cleared from the alveolar space, damaged alveolar epithelial regrowth and differentiation occurs back into type I and type II alveolar pneumocytes, and then ultimately recovery and normalization of gas exchange. If this stage is prolonged, ongoing inflammation and proliferation of fibroblasts may cause long term fibrosis and scarring in some patients.

Mechanical Ventilation in Patients With ARDS

Mechanical ventilation in patients with ARDS allows for the application of positive pressure breaths and ultimately, can help to relieve respiratory distress, improve gas exchange and oxygenation, reduce hypercapnia and concomitant respiratory acidosis and allow for interventions targeted at resolving underlying disease, while reserving and managing respiratory muscle fatigue.

The prognosis and outcomes for small animals undergoing MV is well described in veterinary literature. Depending on the study, between 30% and 62.5% of dogs undergoing MV for various reasons survive to discharge. Mechanically ventilated cats have been shown to have considerably worse outcomes with between 15% and 42% of patients surviving to discharge, although a study evaluating mechanically ventilated cats with congestive heart failure showed a higher survival of 66%. However, when it comes to ventilation for ARDS, outcomes are considerably worse across both dogs and cats, reflecting that this is a much more challenging and heterogenous clinical entity overall, with no definitive therapies that are currently available. The median duration of MV in small animals with ARDS is between 15-44 hours and is likely unfortunately heavily impacted by high euthanasia rates and pet-owner financial constraints, given that MV and the associated ICU care needed for these patients can typically be quite cost-prohibitive.

An important aspect of managing patients with ARDS on the ventilator is using positive end-expiratory pressure (PEEP). PEEP, when used appropriately, can help splint open and recruit alveolar units at the end of expiration and prevent collapse from atelectasis. This can potentially help to minimize atelectrauma, which is the shear injury resulting from cyclical opening and closing of alveoli. While high levels of PEEP can be deleterious and potentially cause cardiovascular compromise or alveolar overdistension, careful use of PEEP remains a cornerstone of ventilatory management of ARDS.

Refractory Hypoxemia

When is hypoxemia defined as refractory? Unfortunately, no standard definition exists, but it is defined in most human studies as a PaO_2/FiO_2 ratio <150 while on a PEEP of 5cm H_2O or higher. Strategies to manage refractory hypoxemia include:

1) Ventilatory Modes:

- ARDSNet vs Open Lung Approach

ARDSNet Approach: Based on seminal ARMA trial in 2000, this approach advocates:

Low tidal volume (6 mL/kg)

Limit Pplat to <30 cm H_2O

Focuses on three things: 1) Protecting the “baby lung” that is open and avoiding overdistension of the lung that is open at FRC, 2) “Resting” the dependent and edema-filled collapsed lung by keeping it out of the respiratory cycle 3) Stabilizing the “in-between” alveoli by applying PEEP

The basic goals of the Open Lung Approach, on the other hand, are:

Open the whole lung with the required pressure

Keep the lung open with PEEP levels above the closing pressure

Maintain optimal gas exchange at the smallest possible pressure levels to optimize CO2 removal

This approach may utilize much higher plateau pressures than recommended by ARDSNet

Modes of ventilation which have been explored in ARDS patients include:

1) Inverse ratio ventilation

2) Airway pressure release ventilation

3) High frequency oscillatory ventilation

2) Prone Positioning:

This has been extensively studied in people. The added benefit of this is unknown in veterinary patients. Potential benefits include:

Improvement in ventilation-perfusion matching

More effective regional ventilation patterns

Reduced compression of small airways from the chest and heart

3) Use of Neuromuscular Blockers:

These have been studied in several large studies of patients with ARDS. A recent meta-analysis published in 2021 showed that NMBs were associated with significantly improved PaO2/FiO2 ratios at 48 hours with a reduced incidence of barotrauma and a lower 21 to 28-day mortality, with no effect on 90-day mortality.

Prognosis

ARDS remains a challenging condition to manage in the ICU. Mortality rates in people remain at approximately 35–40% despite improved management and changes in mechanical ventilation practices. Several therapeutic strategies for ARDS in recent years have been evaluated, including pharmacologic interventions and mechanical ventilation techniques. High flow oxygen therapy also became more widely used in humans with ARDS during the recent pandemic. Of these multitude of approaches evaluated, the most promising one that has been shown repeatedly to improve outcomes in people with ARDS remains the concept of low tidal volume (Vt) ventilation (also known as lung protective ventilation).

There is limited veterinary literature on ARDS in small animals and the overall prevalence of ARDS in dogs and cats is largely unknown. There are some small retrospective studies evaluating the clinical course of disease and a handful of case reports, and some studies that document necropsy findings in these patients. A 2007 study evaluating positive pressure ventilation in dogs and cats reported ARDS in 16% of patients ventilated for hypoxemia unresponsive to oxygen supplementation. In that study, patients with ARDS had the lowest survival rates of all pulmonary disease processes (8.33%). Overall survival to discharge across all these studies remains low (0-16%). More recently, a few case reports have documented patients surviving with newer modes of ventilation such as airway pressure release ventilation, or the use of neuromuscular blockade.

Prognostic indicators for survival to discharge and successful weaning from mechanical ventilation have been well described in people with ARDS, but very little information on this exists in dogs and cats, given the relatively small number of patients surviving to discharge that have been described in the veterinary literature. In a retrospective study evaluating mechanically ventilated dogs and cats, the oxygenation index, which is a measure of oxygenation that accounts for the mean airway pressure in a ventilated patient along with PaO_2 and FiO_2 was found to predict the likelihood of weaning in this general population, although the specific utility of this marker in the subset of dogs and cats with ARDS was not noted. The oxygen saturation index (which replaces the PaO_2 with SpO_2 in the oxygenation index) was also found to be predictive of likelihood of weaning in this study and has been shown correlated with oxygenation index in human ARDS patients.

Conclusion

ARDS is a severe, life-threatening condition that is increasingly recognized in small animals and can be precipitated by a number of diverse risk factors, both pulmonary and extrapulmonary. Management of ARDS relies on aggressive respiratory support and treatment of underlying diseases, but despite this, the prognosis for dogs and cats with ARDS remains poor.

References:

Bernard GR, Artigas A, Brigham KL, Carlet J, Falke K, Hudson L, Lamy M, LeGall JR, Morris A, Spragg R, The Consensus Committee (1994) The American-European Consensus Conference on ARDS: definitions, mechanisms, relevant outcomes, and clinical trial coordination. Am J Respir Crit Care Med 149:818–824Havemeyer

Wilkins PA, Otto CM, Baumgardner JE, Dunkel B, Bedenice D, Paradis MR, Staffieri F, Syring RS, Slack J, Grasso S, Pranzo, Esq G. Acute lung injury and acute respiratory distress syndromes in veterinary medicine: consensus definitions: The Dorothy Russell Havemeyer Working Group on ALI and ARDS in Veterinary Medicine. Journal of Veterinary Emergency and Critical Care. 2007 Dec;17(4):333-9

Ferguson ND, Fan E, Camporota L, Antonelli M, Anzueto A, Beale R, Brochard L, Brower R, Esteban A, Gattinoni L, Rhodes A. The Berlin definition of ARDS: an expanded rationale, justification, and supplementary material. Intensive care medicine. 2012 Oct;38:1573-82

Ferrando C, Suarez-Sipmann F, Mellado-Artigas R, Hernández M, Gea A, Arruti E, Aldecoa C, Martínez-Pallí G, Martínez-González MA, Slutsky AS, Villar J. Clinical features, ventilatory management, and outcome of ARDS caused by COVID-19 are similar to other causes of ARDS. Intensive care medicine. 2020 Dec;46:2200-11.

Pierrakos C, Karanikolas M, Scolletta S, Karamouzos V, Velissaris D. Acute respiratory distress syndrome: pathophysiology and therapeutic options. Journal of clinical medicine research. 2012 Feb;4(1):7.

Derwall M, Martin L, Rossaint R. The acute respiratory distress syndrome: pathophysiology, current clinical practice, and emerging therapies. Expert Review of Respiratory Medicine. 2018 Dec 2;12(12):1021-9.

Bos LD, Ware LB. Acute respiratory distress syndrome: causes, pathophysiology, and phenotypes. The Lancet. 2022 Sep 4.

Hopper K, Haskins SC, Kass PH, Rezende ML, Aldrich J. Indications, management, and outcome of long-term positive-pressure ventilation in dogs and cats: 148 cases (1990–2001). Journal of the American Veterinary Medical Association. 2007 Jan 1;230(1):64-75.

Boiron L, Hopper K, Borchers A. Risk factors, characteristics, and outcomes of acute respiratory distress syndrome in dogs and cats: 54 cases. Journal of Veterinary Emergency and Critical Care. 2019 Mar;29(2):173-9.

Balakrishnan A, Drobatz KJ, Silverstein DC. Retrospective evaluation of the prevalence, risk factors, management, outcome, and necropsy findings of acute lung injury and acute respiratory distress syndrome in dogs and cats: 29 cases (2011–2013). Journal of veterinary emergency and critical care. 2017 Nov;27(6):662-73.

Parent C, King LG, Van Winkle TJ, Walker LM. Respiratory function and treatment in dogs with acute respiratory distress syndrome: 19 cases (1985-1993). Journal of the American Veterinary Medical Association. 1996 May 1;208(9):1428-33.

Cagle LA, Hopper K, Epstein SE. Indications and outcome associated with positive-pressure ventilation in dogs and cats: 127 cases. Journal of Veterinary Emergency and Critical Care. 2022 May;32(3):365-75.

Edwards TH, Erickson Coleman A, Brainard BM, DeFrancesco TC, Hansen BD, Keene BW, Koenig A. Outcome of positive-pressure ventilation in dogs and cats with congestive heart failure: 16 cases (1992–2012). Journal of Veterinary Emergency and Critical Care. 2014 Sep;24(5):586-93.

Zambon M, Vincent JL. Mortality rates for patients with acute lung injury/ARDS have decreased over time. Chest. 2008 May 1;133(5):1120-7.

Acute Respiratory Distress Syndrome Network. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. New England Journal of Medicine. 2000 May 4;342(18):1301-8.

GARY STAMP MEMORIAL LECTURE: HIGH FLOW OXYGEN THERAPY: BEYOND TREATMENT OF HYPOXEMIA

Céline Pouzot-Nevoret [1]

[1] SIAMU, VetAgro Sup, Université de Lyon, Unité de Recherche APCSe, SIAMU (ICU), VetAgro Sup, Marcy l'Etoile, France

Learning objectives:

- To list the physiological effects of HFOT

- To describe the clinical applications of HFOT in dogs and cats

- To calculate and use clinically applicable index as ROX index to predict success or failure of HFOT

Proceeding:

High flow oxygen therapy (HFOT) entails the delivery of heated and humidified oxygen at flow rates exceeding the patient's peak inspiratory flow rate. Unlike conventional oxygen delivery techniques (COT), such as nasal cannula or oxygen cages, HFOT delivers a precisely regulated blend of oxygen and air, ensuring optimal respiratory support while minimizing discomfort and drying of the respiratory mucosa.

Few data are available in dogs, and even less in cats. Current practices describe that HFOT is delivered via specific nasal cannulas, at flow rates between 1 to 2 L/kg/min. Those specific nasal cannulas should ideally be 50% or less of the diameter of the nares. Nowadays, the beneficial use of HOT in hypoxemic patients makes no doubts, and HFOT has been showed to rapidly improve oxygenation parameters in dogs nonresponding to conventional oxygen therapy.

However, HFOT is explored in human medicine for other indications than hypoxemia, and some data are now available in dogs and cats. After a quick description of the physiological effects of HFOT and use in hypoxemic patients, this lecture will review other uses of HFOT and describe clinically applicable index that can be used by the clinician to predict failure or success of HFOT in hypoxemic patients.

Indications of HFOT beyond correction of hypoxemia

Post-extubation period

Post-extubation respiratory insufficiency is a current complication following weaning from mechanical ventilation. In human medicine, HFOT has been associated with reduced post-extubation respiratory failure and increased PaO2 compared to conventional oxygen therapy (COT) and has been showed to be non-inferior to non-invasive ventilation regarding reintubation rate.

In dogs, the use of HFOT in the recovery period has been studied by Jagodich et al. (2020) in 5 brachycephalic dogs with signs of upper airway obstruction. Application of HFOT reduced dyspnea scores in this population, and interface was well adapted to the brachycephalic face.

No data are available on the post-extubation effects of HFOT in a population of dogs and cats after mechanical ventilation, but human literature and preliminary results in dogs encourage the use of this technique.

During procedural sedation/anaesthesia

Sedation/anaesthesia in dyspneic patients can be challenging, delaying diagnostic or therapeutic procedures. In people, the use of HFOT during sedation has shown to reduce the risk of hypoxemia and increase the minimum oxygen saturation, whatever the procedure, FiO2, or risk-profile of the patient.

In dogs, HFOT has been described in 4 dogs during bronchoscopy, showing no complication, but couldn't show prevention of hypoxemia. In our institution, we performed a pilot study on dogs and cats undergoing bronchoscopy and bronchoalveolar lavage, showing that HFOT was feasible and safe and that dogs and cats under HFOT demonstrated significantly lower decrease in SpO2 and higher SpO2 during the procedure compared to the COT group. Another prospective study showed significantly higher mean PaO2 in the HFOT group (n=10) compared to the control group (n=10) during bronchoscopy, with no major adverse effects.

Congestive heart failure (CHF)

In addition to providing oxygen support, HFOT has been proposed to reduce pulmonary congestion via reductions in cardiac preload and afterload, and has been shown to be effective in improving respiratory rate, SpO2, lactate and arterial blood gas parameters compared to COT in patients with acute CHF.

One case report of use of HFOT in a cat with congestive heart failure has been published, but use of HFOT in CHF needs more data in dogs and cats.

Other applications

In human medicine, HFOT has been successfully used in other applications like carbon monoxide intoxication, asthma, acute hypercapnic exacerbation of chronic pulmonary disease, opening the possibility of exploring new indications in our population of patients.

How to evaluate the probability of success or failure of HFOT?

Considering the ease to use and the potential advantages of HFOT, the main risk is to delay intubation. The S:F ratio and the respiratory-oxygenation index (ROX), calculated by dividing the patient's S:F ratio by their respiratory rate, are monitored in humans on HFOT to guide timely escalation to mechanical ventilation when indicated.

In one prospective study on 88 dogs, ROX and SF ratio were adequately predictive of HFOT success when averaged over treatment hours 0-16 with acceptable AUC (0.72 and 0.77 respectively, $p<0.05$). HFOT responders had significantly higher SF ($P=0.002$) and ROX ($P=0.005$) than HFOT non-responders.

ROX and SF ratio need more exploration of their use but seem to be valuable tools for evaluation of patients under HFOT.

References:

Duble E., Utility of the respiratory rate-oxygenation (ROX) index as a predictor of treatment response in dogs receiving high-flow nasal cannula oxygen therapy, J Vet Emerg Crit Care, 2023, S1-S45. https://doi.org/10.1111/vec.13336

Jagodich T.A., Preliminary evaluation of the use of high-flow nasal cannula oxygen therapy during recovery from general anesthesia in dogs with obstructive upper airway breathing. J Vet Emerg Crit Care, 2020, 30; 487-492. doi: 10.1111/vec.12971.

Pouzot-Nevoret C., Use of high-flow oxygen therapy in a cat with cardiogenic pulmonary edema. JFMS Open Rep; 2023, 9(2); 20551169231195767. doi: 10.1177/20551169231195767.

Rosa de Jaureguizar Tesas M., The use of High-Flow Nasal Oxygen Therapy in 4 dogs undergoing bronchoscopy, Front Vet Sci ; 2023, 10 ; 1088103. doi:10.3389/fvets.2023.1088103

Whitney J., Clinical review of high-flow nasal oxygen therapy in human and veterinary patients. Front Vet Sci, 2023, 10:1070881. doi: 10.3389/fvets.2023.1070881. Other references on request

GARY STAMP MEMORIAL LECTURE: VENTILATOR WAVEFORMS: WHAT CAN I LEARN FROM THEM?

Céline Pouzot-Nevoret [1]

[1] SIAMU, VetAgro Sup, Université de Lyon, Unité de Recherche APCSe, SIAMU (ICU), VetAgro Sup, Marcy l'Etoile, France

Learning objectives:

After this lecture, the attendee, by analyzing the ventilator waveforms, should be able to:

- Recognize modification of resistance and compliance of the respiratory system

- Detect a leak in the respiratory circuit

- Recognize patient-ventilator asynchrony

Proceeding:

Mechanical ventilation is a crucial aspect of critical care medicine in veterinary practice, particularly for dogs and cats encountering respiratory distress due to various underlying conditions. While the basic principles of mechanical ventilation remain consistent across species, advances in technology have enabled more sophisticated analysis methods to enhance patient care. One of these advances is the utilization of waveform analysis, which, together with blood gas analysis and inspection of the patient, provides valuable insights into lung mechanics, monitoring of the disease, ventilator troubleshooting and patient-ventilator interaction and asynchrony.

This lecture will explore the significance and applications of mechanical ventilator waveform analysis in the management of respiratory disorders in dogs and cats in a critical care setting.

Generation of mechanical ventilator waveforms

Mechanical ventilator waveform analysis involves the interpretation of graphical representations of pressure, flow, and volume over time during the respiratory cycle. They are divided into scalars (one parameter is plotted over time), or loops (2 parameters are plotted simultaneously). With modern ventilators, different waveforms can be displayed simultaneously, and the clinician can choose the most relevant ones. Usually, the scalar that represents the dependent variable (example: volume/flow if the patient is ventilated in pressure-controlled mode) will most directly reflect the patient's respiratory mechanics and should be followed.

Use of waveforms to determine ventilation modes

Different mode of ventilation can be applied in patients, based on patient's characteristics and clinician preference. Shape of the pressure and flow scalar will help determine the ventilation mode. In a pressure-controlled mode, the pressure scalar will have a square shape and the flow scalar an exponential decay shape, whereas in a volume-controlled mode, the pressure scalar will have an exponential rise shape and the flow scalar will have a square or a descending ramp shape.

Analysis of waveforms can also help the clinician to determine is the breaths are mandatory (typical pressure, volume and flow scalar as described above), spontaneous (sine wave appearance) or supported, and if the patient triggered the breath or not.

Use of waveforms to determine respiratory mechanics

One of the primary applications of waveform analysis is in respiratory monitoring. By assessing waveform patterns, clinicians can identify abnormalities such as airway obstruction, dynamic hyperinflation or change in lungs compliance.

Evaluation of the pressure waveform, especially during an inspiratory pause, is of great interest for evaluation of a patient's lung mechanics. For example, evaluation of the peak inspiratory pressure (PIP), plateau pressure (Pplat – measured during an inspiratory hold), positive end-expiratory pressure (PEEP) will give information on resistance and compliance of the respiratory system and allow calculation of static and dynamic compliance. Compliance and resistance of the system can also be evaluated on the pressure-volume loop, thanks to the change in area and/or in orientation of the loop.

These measures will allow the clinician to follow the evolution of the lung disease.

Optimization of Ventilator Settings

Waveform analysis plays a vital role in optimizing ventilator settings to improve patient outcomes. By analyzing pressure-volume loops, clinicians can determine the most appropriate tidal volume, detect overdistension or collapse of alveoli or detect a leak in the circuit. More precisely, the inspection of the lower inflection point (LIP) may help to set the PEEP, as the LIP reflects the point at which a number of collapsed conduction and/or gas exchange units open. Keeping the PEEP above the LIP will help reducing atelectrauma.

On the other hand, the upper inflection point (UIP) reflects the point where overdistention of the lung will start. Keeping the PIP below the UIP may limit the volutrauma.

This personalized approach minimizes the risk of ventilator-induced lung injury while ensuring adequate gas exchange.

Detection of Patient-Ventilator Asynchrony

Patient-ventilator asynchrony is a common complication of mechanical ventilation that can compromise patient comfort and lead to ineffective ventilation. Waveform analysis allows for the early detection of asynchronies such as ineffective triggering, auto triggering, or double triggering ("breath stacking"). By

recognizing these patterns, clinicians can adjust ventilator parameters or consider alternative ventilation strategies to enhance patient-ventilator synchrony and minimize respiratory effort.

Conclusion:

Mechanical ventilator waveform analysis is a valuable tool in the management of respiratory disorders in dogs and cats. By providing real-time feedback on lung mechanics and patient-ventilator interaction, waveform analysis enables clinicians to optimize ventilator settings, monitor respiratory status, and improve patient outcomes. Continued research and technological advancements in this field hold promise to further enhance the quality of care provided to veterinary patients requiring mechanical ventilation.

References:

References available upon request

Nurse & Tech Stream, Saturday 1 June 2024

CALM YOUR RAAS DOWN! – UNDERSTANDING THE RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM AND ITS INHIBITORS

Angela Elia [1]

[1] Pieper Veterinary Group, Pieper Veterinary Group, Middletown, CT, United States

Learning objectives:

- Explain what the RAAS is, how it is activated, and what it does within the body.

- Name the organs involved in the RAAS.

- Name the hormones and enzymes involved in the RAAS and what they do.

- Explain how RAAS inhibitors work.

Proceeding:

What is the RAAS System?

The RAAS is a hormone cascade system that works to regulate blood pressure, systemic vascular resistance, and fluid/electrolyte balance.

What Organs are involved in the RAAS system?

The reach of the RAAS in the body is vast. Organs involved in the system include the brain, lungs, heart, liver, and kidneys.

What Hormones and Enzymes are involved in the RAAS system?

Renin: Hormone that is secreted by the juxtaglomerular cells of the kidneys. It is released in response to low blood flow/pressure in the kidney.

Angiotensinogen: Alpha-2 globulin that is secreted by the liver and stored in blood plasma. When it comes into contact with Renin which cleaves its protein into Angiotensin I.

Angiotensin I: Inactive while in circulation until it comes into contact with Angiotensin Converting Enzyme (ACE) which turns Angiotensin I into its activated form – Angiotensin II.

ACE- Angiotensin Converting Enzyme: Made by endothelial cells. Circulates in plasma and can be found in many tissues but are most active in the lungs.

Angiotensin II: Major product of the RAAS system. Potent vasoconstrictor. It also stimulates the release of ACTH, Aldosterone and ADH.

Angiotensin III and Angiotensin IV: Degraded versions of angiotensin II have the same, but lessened effects.

ACTH: Adrenocorticotropic hormone secreted by the anterior pituitary gland. ACTH stimulates the adrenal cortex to release cortisol.

Cortisol: Produced by the adrenal glands. Hormone that works to create a catabolic state within the body, allowing for the immediate creation of glucose to meet energy needs. Helps retain sodium and water in the kidney.

Aldosterone: The body's main mineralocorticoid hormone. Produced in the adrenal cortex. Can cause vasoconstriction, increases sodium and water retention in the kidney.

ADH: Anti-diuretic hormone/vasopressin. Made in the hypothalamus and released by the posterior pituitary. Preserves intravascular volume by decreasing water loss and increasing water conservation. Is also a potent vasoconstrictor.

How is the RAAS system activated?

Following an initial drop in blood pressure, renal blood flow slows. The decrease in blood flow activates the RAAS system in an attempt to restore blood pressure to normal. The RAAS system begins its activation with the release of Renin from the juxtaglomerular cells in the kidney.

What happens when the RAAS system stays activated?

Although crucial to the correction of acute conditions such as hypovolemic shock, chronic RAAS activation can cause significant negative changes in the body. Chronic adrenergic stimulation leads to increases in afterload, ventricular arrythmias and cardiac remodelling. Angiotensin II and Aldosterone have been shown to facilitate cardiac remodelling and fibrosis. Chronic increases in sodium and water retention increase preload. Increased preload and afterload lead to volume and pressure increases that are detrimental to cardiac function. Negative changes to cardiac function increase the severity of heart disease, which, in turn, keeps the RAAS system chronically activated, creating a cycle of cardiac damage. Chronic exposure to Angiotensin II and Aldosterone have also been shown to have damaging effects to the kidneys. By activating inflammatory cells, increasing glomerular pressure, and the extracellular matrix, both of these hormones contribute to interstitial fibrosis and glomerulosclerosis.

What drugs inhibit the RAAS system?

Renin Inhibitors: These substances bind directly to Renin, inhibiting it from synthesizing Angiotensin II. Available Drugs: Aliskiren

ACE Inhibitors: These drugs prevent the activation of Angiotensin I into Angiotensin II. Available drugs: Benazepril, Enalapril

Angiotensin II Receptor Blockers: block the binding of Angiotensin II to its receptors. Available Drugs: Losartan, Valsartan

Aldosterone Antagonists: Blocks the effects of aldosterone in the kidney. Increases renal sodium excretion which can lower blood pressure. Available drugs: Spironolactone

References:

Silverstein D, Hopper K. Small Animal Critical Care Medicine. 2nd Ed. St. Louis: Saunders; 2015.

Battaglia, Steele AM. Small Animal Emergency and Critical Care for Veterinary Technicians. 4th ed. St. Louis: Elsevier; 2021.

Norkus, CL. Veterinary Technician's Manual for Small Animal Emergency and Critical Care. 2nd Ed. Hoboken: John Wiley & Sons; 2019

WHEN THE KIDNEYS GO ON STRIKE: THE GREAT NEPHRON REBELLION

Natasha Summerfield [1]

[1] University Zurich, ICU Animal hospital Zurich, Zurich, Switzerland

Learning objectives:

- Understand the role of ICU and ER nurses in the detection, triage, and management of acute kidney injury (AKI) in dogs and cats

- Gain insights into the physiology of the kidney and the pathophysiology of AKI

- Explore treatment options for AKI, focusing on nursing considerations for optimal patient care

Proceeding:

Introduction

The kidney, a vital organ, plays an important role in maintaining homeostasis within the body. Its functions extend beyond the filtration of waste products and electrolyte balance. The kidney regulates blood pressure, produces erythropoietin, and modulates acid-base balance.

Understanding AKI:

Pathophysiology

Acute Kidney Injury refers to the abrupt decline in renal function over a short period. It encompasses pre-renal, intrinsic renal, and post-renal causes and is associated with high morbidity. The mortality rate for dogs undergoing medical care or hemodialysis is as high as 45% to 60%.

Pre-renal factors (damage before the kidney). Shock in its various forms can lead to pre-renal damage.

Intrinsic renal damage (damage directly within the kidney) may result from toxins, such as grapes and raisins, chemicals and drugs, or infections such as pyelonephritis.

Post-renal causes (after the kidney), like urinary obstructions such as Feline lower urinary tract disease, hinder urine flow and cause a back-up of urine and a collection of toxins that should be filtered out in a normal situation.

Stages of AKI

AKI may be staged in different categories based on blood values and urine output. Azotemia with Oligo/anuria is usually associated with a later-stage AKI that needs emergency treatment and is associated with reduced survival rates. The International Renal Interest Society separates Acute Kidney injury into five different stages.

Presentation

Patients may arrive with signs such as lethargy, vomiting, nausea, diarrhoea, and anorexia. They may be poly/an or oliguric. There are also less common signs such as seizures, syncope, ataxia, and dyspnoea. The nurse will measure vital parameters on admission/triage, connecting the history with the clinical picture, applying their ability to critically think, aiding in swift treatment for the emergency patient.

Treatment Options for Dogs and Cats

1. Fluid Therapy: Adequate hydration is key, the patient's fluid therapy must be tailored to the underlying cause and patient's condition. To monitor for fluid overload, nurses can monitor ins and outs where a urinary catheter is in situ and otherwise, the nurse can monitor via 'free catch' when taking out for a walk. Incontinence pads and litter trays may also be weighed. This all can help in understanding, albeit roughly, what your patient is producing. The patient should also be weighed regularly using the same scales at each weigh-in.

2. Nutritional Support: Meet the patient's specific dietary needs. Adjust feeding plans to minimize renal workload, ideally, using a specially formulated renal diet. The use of feeding tubes may be necessary in our patients; it is the job of the nurse to communicate the patients' requirements and advocate for their well-being. Force-feeding can be extremely stressful to any patient, but especially so when nausea is present. These patients commonly benefit from the placement of a nasogastric tube, which can be performed with minimal sedation by a trained nurse. Nutritional support in AKI has been shown to increase nitrogen level balance in humans.

3. Pharmacological Interventions: Medications may include diuretics, anti-emetics, and analgesics, tailored to individual cases. Depending on the case, the patient may also need support regulating their potassium level with glucose, insulin, calcium gluconate and terbutaline, especially in life-and-death situations where hyperkalaemia is present. Specific therapies can be administered for some disease processes, such as ethylene glycol toxicity and leptospirosis.

4. Dialysis: Hemodialysis can be considered in severe cases. Of course, the availability of resources will determine this, and cost is frequently a hindrance. Nursing care during this phase will include: caring for indwelling central catheters and monitoring the patient whilst undergoing dialysis (vital parameters, keeping the patient warm and calm).

Nursing Considerations: Your Vital Role

- During triage, quickly get a relevant medical history, assess the patient, and promptly notify the veterinarian of any concerning symptoms that may suggest AKI.
- Monitor renal parameters closely, including urine output, weight, creatinine, and BUN levels. Collecting and assessing blood gas samples.
- Monitoring patient mentation as well as ECG monitoring, blood pressure and respiration.

- Ensure adequate pain management and comfort for the patient, knowing what analgesics can be used for AKI patients.
- If urinary catheters are in place, understanding the risk and hygiene levels that must be needed.

References:

Rimer, D. et al. 'Acute kidney injury in dogs: Etiology, clinical and clinicopathologic findings, prognostic markers, and outcome', Journal of veterinary internal medicine / American College of Veterinary Internal Medicine, 36(2),2022, pp. 609–618.

Scheinkestel, C.D. et al. 'Prospective randomized trial to assess caloric and protein needs of critically Ill, anuric, ventilated patients requiring continuous renal replacement therapy', Nutrition , 19(11-12), 2003 pp. 909–916.

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

PATIENT HANDOVERS

Eleanor Haskey [1]

[1] Royal Veterinary College, London, United Kingdom

Learning objectives:

- Define the term 'patient handover'

- Explain the importance of an effective patient handover and the consequences of a bad handover

- Perform a standardised structured patient handover in the clinical setting

- Identify barriers which may result in an ineffective handover and suggest ways to reduce these

Proceeding:

Patient handovers

Patient handovers are a complex activity performed in veterinary and human healthcare whereby there is a temporary or permanent transfer of responsibility and accountability of care. In the veterinary clinic there are numerous occasions when patient handovers and transfer of accountability and care are required. This may be for an individual patient such as perioperative/intraoperative handovers, or a group of patient handovers during/at the end of shifts, handovers to other teams/departments and handovers to other clinics (first opinion to OOH to referral). In addition to the differences in specialties and subspecialities, the principles of achieving a successful handover can be applied across the board.

It is well documented in the medical literature that structured handovers improve information transfer and staff satisfaction, whilst reducing adverse events such as medical errors/harm to a patient, morbidity, mortality, hospital stay, inefficient use of resources and cost. It should be acknowledged that many veterinary professionals are not formally taught how to deliver an effective patient handover during their training, despite handovers playing an integral role in ensuring communication across the team is clear and accurate. There is also a lack of veterinary related evidence and currently no veterinary consensus or guidelines for patient handovers. This creates the opportunity for veterinary professionals to draw on similarities and best practice documented in the medical literature with consideration given to the following aspects of a patient handover:

Who should be involved (outgoing and incoming nurse team)

When the handover should take place (at shift changeover)

Where it should take place (cage side)

How it should happen (face to face)

What information needs to be handed over (standardised format)

There is a range of structured formats which are currently used across the world in order to pass on the pertinent pieces of information about the patient, however the World Health Organisation have endorsed the use of the ISBAR (identification/signalment of the patient, situation, background, assessment, recommendation) structured handover format. Additional information that may be required includes code status, a specific to-do list with timeline and ownership (including follow-up of any pending investigations) and anticipated issues and contingency plans.

Ineffective handovers are associated with interruptions and distractions, multi-tasking, fatigue, ambiguous team membership or roles, passive listening, time pressures, language and training. Some of these issues are easily resolved for example, setting out ground rules during patient handovers, having clear structure, building a process that aligns with the clinic/team/situation and team training. In addition to the format of the handover, there are other behaviours that can impact the success of information transfer between teams such as clear and slow verbal projection, note taking during rounds, encouraging clarification/asking questions, and promoting a respectful and supportive environment. Another exciting opportunity for the veterinary profession is the integration of technology such as electronic medical patient record systems, to strengthen the patient handover process.

Whilst we do not know the full impact of handovers and their relation to adverse events in the veterinary field, there is likely room for improvement in how we perform patient handovers. A good place to start is by reviewing current practices within your veterinary clinic to identify areas of strength and weakness. Implementing some small changes such as the structured format, no interruptions, the opportunity to ask questions/seek clarification at the end. Veterinary nurses and technicians are the perfect advocates in the clinic to encourage steps towards performing a structured safer handover in order to improve continuity of patient care.

WIDENING OUR HORIZONS: HOW VN'S ARE LOOKING TOWARDS HUMAN CENTRED NURSES TO IMPROVE PATIENT CARE

Lindsey Ashburner [1]

[1] University of Edinburgh, Edinburgh, United Kingdom

Learning objectives:

- Define 'One Health'

- What is our role as veterinary nurses in One Health?

- Identify opportunities for learning and collaboration

Proceeding:

Introduction

The concept of One Health recognises that the health and wellbeing of people, animals, and the environment are inextricably linked. One Health is not new, but it has become more important in recent years. As the world's population grows, professionals across each of these areas must collaborate to bring about change. Collaborative work is essential to ensure adequate healthcare is available to all, that public health and animal health and welfare are safeguarded, that food is produced sustainably, and that the natural environment is protected.

Role of the veterinary profession in One Health

The veterinary profession has a long history of recognising and encouraging the concept of One Health and veterinary surgeons and nurses have identified that their day-to-day work frequently incorporates One health approaches. Examples of these include the following:
Antimicrobial resistance: Antimicrobials are essential to veterinary and human medicine to treat disease, but inappropriate use could leave them ineffective. Continued availability of existing antimicrobials and the development of new ones are essential to maintain the health and welfare of humans and animals. Their responsible use needs to be at the forefront when treating our patients and public awareness should be promoted.
Mental health and wellbeing: The recognition that dogs and other animals can provide support to people in many ways and positively impact our mental, physical, and emotional health. We as veterinary nurses need to use our skillset and empathy to support all members of our community to champion this human-animal bond.

Zoonotic diseases: Worldwide, nearly 75% of all emerging human infectious diseases in the past 30 years originated in animals. Preventative healthcare such as vaccinations and deworming as well as good husbandry practices and hygiene are important to reduce the risk of these communicable diseases. Owner and public awareness is vital to encourage these practices.
Non-communicable diseases: These are known as chronic diseases that cannot be directly transmitted between people or animals. It is recognised that humans and companion animals have numerous disease processes in common, including cancer, obesity, cardiac, respiratory and endocrine diseases. A collaboration in health research has the potential to advance the understanding of these mutual diseases through a multidisciplinary approach to health.
Environmental sustainability: Nature is the bedrock of the living world, and we are totally dependent on natural services for the provision of food , water, energy, medicines, and genetic resources. However, our natural world has been significantly degraded through human activities. We as veterinary nurses need to consider the links between veterinary healthcare and ecosystems, how we can practice more sustainably and how we can influence and lead in our wider community.
Injuries: Animals, both wild and domesticated can injure humans with potentially deadly impacts. The best way to manage such injuries is to prevent them happening in the first place. This can be done but understanding, and where necessary, modifying animal behaviours or advising humans on how to act around animals. Humans may also physically harm animals. Whilst most of these injuries are accidental, in some cases they may not be. Where serious animal abuse has occurred in a household there may be an increased likelihood that some other form of family violence is also occurring. Whether we are faced with a patient or a human victim of abuse, it's important that no one steps outside their area of expertise but that, where possible, concerns are reported accordingly.

Conclusion

The concept of One Health is dynamic and continuously evolving as new knowledge is acquired, much like the veterinary nursing profession itself. It is hoped that if we, as veterinary professionals, can better educate ourselves on the issues of One Health, we can become trusted and reliable sources of information for others, especially within our local community.

References:

Capua, I. and Cattoli, G., One Health (r)Evolution: learning from the past to build a new future. Viruses 2018, 10(12), 725. doi: 10.3390/v10120725.

Destoumieux-Garzon, D., Mavingui, P., Boetsch, G., Boissier, J., Darriet, F. et al., The One Health Concept: 10 years and a long road ahead, 2018, Frontiers of Veterinary Science 5, 14.

Gibbs, E.P.J., The evolution of One Health: a decade of progress and challenges for the future. Veterinary Record 2014,174, 85–91. doi: org/10.1136/vr.g143

Jones R. and Jeffrey A., One Health for Veterinary Nurses and Technicians: An Introduction, 2023, Oxfordshire: CABI.

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

NEONATAL NURSING

Angela Elia [1]

[1] Pieper Veterinary Group, Middletown, CT, United States

Learning objectives:

- Describe the physiologic differences between neonate and adult felines and canines

- List the difference in vitals and how they change as the neonate ages

- Describe the special considerations for the neonatal physical exam and triage

- List the most commonly used sites for venous catheterization in the neonate

- Describe the ideal environment needed for neonates to thrive in clinic

Proceeding:

Neonatal Development Stages

Neonatal Period: This is the first stage of puppy development that begins at birth. At this stage, puppies and kittens only have the sense of taste and touch, and they rely completely on the mother.

Transitional Period: Occurs from 2-4 weeks of age. Sensory development continues with hearing and sense of smell. Teeth start to come through and eyes will begin to open. This is the age where personality beings to develop. Babies will begin interacting with littermates, move their tails, stand, and walk a little.

Socialization Period: Occurs from 3-12 weeks. This is the most important stage in development. It is vital to understand this stage as most animals are separated from their mothers during this period and will rely on owners for development in this stage. This is the period to introduce young animals to items such as vacuum cleaners, street noises, etc. Social skills will either need to be taught through interactions with other animals or through play with owners.

Physiological Considerations for the Neonate

Neonates have no gag reflex. This reflex develops at around 10 days of age.

Cardiovascular physiology can vary greatly from that of an adult. Neonates have lower blood pressures and stroke volumes, but higher heart rates and greater cardiac output. This elevated heart rate decreases as the animal develops increased parasympathetic influences (vagal tone) at 4 weeks of age. Atropine and dopamine will have little effect on cardiac function until 4-5 weeks of age.

The neonate is born with limited capacities for gluconeogenesis and glycogenolysis because of the immaturity of the newborn's liver. In addition, initial limited hepatic glycogen stores, small muscle mass, lack of adipose tissue, and decreased use of free fatty acids as an alternative energy source place neonates at great risk for developing hypoglycemia in the face of even the briefest fast. Impaired gluconeogenesis caused by the delayed maturation and induction of the rate-limiting gluconeogenic enzymes has been shown to result in hypoglycemia in human infants and is suspected in the same condition in kittens and puppies.

Neonates cannot urinate or defecate without assistance. When with the mother, she will stimulate them both before and after feedings. Neonate feces should resemble mustard. It can be bright yellow with or without “seed” like material. These “seeds” are fatty excrement and are considered normal.

Maintenance of normal physiologic functions is related to temperature in puppies and kittens. In puppies that become chilled, the heart rate may drop precipitously. A newborn with a rectal temperature of 96° F has a heart rate somewhere between 200 and 250 beats per minute (bpm). Once the rectal temperature reaches 70° F, the heart rate quickly drops to only 40 bpm. A decreased heart rate may result in inappetence, dehydration, and loss of suckling reflexes. In addition, nursing bitches may refuse to nurse and care for cold puppies and even push them away. When body temperature falls below 94° F, a gastrointestinal (GI) ileus develops, and a chilled puppy will stop trying to nurse. If chilled puppies are not rewarmed before force feeding, regurgitation and subsequent aspiration pneumonia can result.

Neonatal Vitals

The Neonatal Triage

It is important to realize that with all of the differences mentioned, neonatal triages are going to be markedly different than triaging an adult animal. It is important to remember the following:

Always wear PPE when handling a mother and/or her neonates

Gather information about this birth and any previous litters

Gather information about feeding schedule, type of food being fed

Husbandry information like heating sources, whelping areas, bedding used is also important

Do not forget to ask about the health and behavior of the littermates if available

The Neonatal Physical Exam

After gathering information about the neonate’s environment and care, we can focus on the neonatal physical exam. During any physical exam on a neonate, it is important to identify:

Confirmation of sex

Urogenital abnormalities

Cleft palate

Open fontanelle

Hydrocephaly

Blood sugar levels

Temperature – remember PROBE COVERS CAN TEAR THE RECTUM OF A NEONATE and should never be used.

Neonatal Care in the ER and ICU

It is important to have some equipment available to treat neonatal patients. Most often needed are warming devices and oxygen supplementation systems. Incubators are ideal, but anesthesia induction boxes, climate-controlled Snyder cages and other methods may suffice for care. As a neonatal nurse, it is important to remember that a neonate's condition and vitals may change much faster than with an adult patient. It is vital to monitor hydration, weight changes, mentation, temperature and adhere to a strict feeding schedule.

Neonatal Venipuncture

Neonatal venipuncture can range from more than challenging to nearly impossible. It is important to be creative but careful with selection of venipuncture and catheterization sites. The most commonly used IVC site for the neonate is the jugular vein. This is often the most visible vein and can be the most comfortable for the patient. Your chances of getting a better blood return for lab sampling also increases with use of the jugular vein. It is important to keep in mind that neonates can only handle micro sampling, especially if dehydrated. You may only get a few drops per sample, so using them in blood glucose or other machines that need minimal sample size such as i-stat or other handheld analyzer are preferred. If no venous access can be gained, Intraosseous catheterization may be necessary. Intraosseous catheters can be placed in the proximal femur, but other potential sites include the proximal humerus and proxiomedial tibia. Use of an IO catheter has similar results in rehydration and drug availability as that of an IVC.

Neonatal Nutrition

Neonates have minimal fat reserves and a limited ability to generate glucose. Shortly after being born, neonates deplete their stores of glycogen, making nursing shortly after birth essential. Neonates need constant replenishment of nutrients making even minimal fasting dangerous. The caloric requirement of a neonate is 133 cal/day during the first week of life, 155 cal/kg/day in the second week of life, 175-200 cal/kg/day for the third week and 220 cal/kg/day for the fourth week. There are several commercially made milk replacement formulas available for use. Careful preparation of powdered formulas by following package directions exactly is imperative to prevent osmotic diarrhea. Feeding can be done through bottles if the neonate readily suckles and has no palate abnormalities. Even with a strong suckle, it is easy for neonates to aspirate from being bottle fed. Orogastric feeding tube use is often the safer, more efficient, and preferred method of feeding. When correctly performed, it can prevent aspiration, allow more accurate measurement of food ingestion, and allows for reliable medication administration.

The Neonatal Medical Record

Even though it is often overlooked, it is important to keep detailed records of neonatal treatments and monitoring results. It can become overwhelming when caring for multiple neonates, but it is essential to keeping track of patient progress. Medical records should include progress notes for events such as activity level, mentation, ability to suckle/eat and several weights checks throughout the day. Marking neonates with ribbons, collars, skin markers, etc. may be necessary to identify each individual. Significant events that would warrant alert of your clinician are:

Change in mentation or activity level

Listlessness

Any nasal or ocular discharge

Regurgitation

Milk coming from the nose while feeding/nursing

Refusal/aggression from mother

Discharging the Neonatal Patient

Neonatal discharges are often complex and revolve mostly around client education. Clients will often need written instructions and in person demonstrations of things like feeding, medicating, and toileting a neonate. Husbandry directions and supplies must be understood and gathered before the neonates arrive back home. Teaching clients how to record weights and feedings is also important so they can identify failure to thrive at the earliest onset. If the client plans to reunite the neonate with the littermates and/or the mother, it is imperative that they be monitored for refusal of care. The mother may reject a neonate after hospitalization by refusing to feed it, pushing it out of the whelping area and even being aggressive towards it causing severe injury or death. If any of these occur, the owner will have to continue care of the neonate until weaning can occur.

References:

Traas, A.M. (2008). Resuscitation of canine and feline neonates. Theriogenology, 70, 343-348.

Davidson Autumn P. Management of the neonate in dogs and cats. Merck Veterinary Manual; Dec 2022.

Silverstein D, Hopper K. Small Animal Critical Care Medicine. 2nd Ed. St. Louis: Saunders; 2015.

Battaglia AM, Steele AM. Small Animal Emergency and Critical Care for Veterinary Technicians. 4th ed. St. Louis: Elsevier; 2021.

Norkus, CL. Veterinary Technician's Manual for Small Animal Emergency and Critical Care. 2nd Ed. Hoboken: John Wiley & Sons; 2019

Plunkett SJ. Emergency Procedures for the Small Animal Veterinarian. 2nd ed. St. Louis: Saunders; 2013.

Leadership Stream, Saturday 1 June 2024

WELLNESS ACTION PLAN: HOW TO KEEP YOUR TEAM HAPPY AND HEALTHY

Laura Cole [1]

[1] Queen Mother Hospital for Small Animals, Royal Veterinary College, Hertfordshire, United Kingdom

Learning objectives:

By the end of the lecture delegates will be able to:

- Understand different personality types and learning styles

- Understand the importance of mentorship in the workplace

- Implement a wellness action plan in their practice

- Recognise poor mental wellbeing in themselves and others

Proceeding:

Wellbeing and mental health of veterinary professionals is a topic of increasing interest within the veterinary community. A large percentage of veterinary professionals, particularly young veterinarians, report experiencing burnout as well as serious psychological distress (Volk, et al 2022). Causes include but are not limited to: inability to cope with moral stressors, team conflict and inadequate mentorship (Reinhard, et al 2022).

Veterinary practices, both private and academic, often comprise large multidisciplinary teams with diverse personalities and character types. Getting to know your team, particularly how they prefer to receive information can be invaluable in promoting team cohesion and minimising conflict. There are various personality assessments and learning style tests available online, including DOPE 4 bird personality test (Stephenson, 2019), colour tests (Erikson, 2019) and Honey and Munford learning styles (1986). These assessments can be used to help individuals understand themselves as well as other team members. Unlike the other tests, Honey and Munford (1986)'s four learning styles (activist, reflector, theorist, pragmatist) are not fixed personality traits but acquired preferences that are adaptable and can change based on circumstances. Understanding individual learning styles can help manage stressors and minimise work conflict.

Mentoring, an underutilised initiative in the veterinary sector may have a role in reducing burnout. Studies in human healthcare have shown that carefully implemented mentorship programmes may have a role in reducing burnout in physicians, residents, and nurses (Leung, et al 2021; Drybye, et al 2019; Morioka, et al 2017). Effective mentoring can help increase self-awareness, self-autonomy and strengthen underutilised skills. However, mentor-mentee pairing should be considered carefully to

prevent potential difficulties associated with mentorship, including conflict of interest, imbalance of power and unrealistic expectations (Burgess, et al 2018).

Mind, a UK based mental health charity have developed a Wellness Action Plan (WAP) initiative (Mind, n.d), inspired by an evidence based system used for people recovering from mental health issues; Wellness Recovery Action Plan® (WRAP®, n.d) `This tool, composed of questions with free text, allows the individual to reflect on what keeps them well at work, what triggers poor mental health, how it may be recognised and what warning signs may there be, should their mental health be affected. It further allows the individual to consider how they would be affected should a mental health problem arise and what a line manager can do to help address a mental health problem should they experience one. This initiative encourages individuals to be self-aware and by sharing the document with line managers can open a dialogue to allow them to better understand the individuals' needs and experiences. This will allow the manager to recognise poor mental health sooner and allow them to provide extra support under certain conditions when needed. For example, should someone struggle with verbal instruction under high stress situations, ensuring instructions are written under these conditions. It is important this document is held confidently, and the individual agrees to whom the document is shared with, and done so in a confidential manner, such as a password protected document.

The beneficial effect of WRAP® on employment success has been studied in individuals with psychiatric disabilities. Based on interviews with individuals the findings of this study showed that WRAP® strategies facilitated success in employment (Olney, et al 2017). A Wellness Action Plan, based on the Mind initiative was introduced in a postgraduate emergency and critical care residency training programme at one of the authors' place of work four years ago, and has subsequently been expanded to other residency training programmes. The initiative is voluntary and has received around 80% uptake. The Wellness Action plan was distributed to residents at the beginning of their training and repeated annually. The hope was the WAP will promote self-awareness in individuals and help line managers better understand individual needs and stressors as well as proactively provide mental health support. Challenges met during the first launch included: asking individuals to answer things they may not know about themselves, and difficulties in line managers actioning specific points and therefore being at risk of appearing to "ignore" the document and finally issues around confidentiality. Wider distribution of the initiative and studies are needed to determine the impact of this wellness initiative.

References:

Volk JO, Schimmack U, Strand EB, et al.(2022) Veterinary Mental Health and Wellbeing and How to Improve Them. Learnings from the Merck Animal Health Veterinarian Wellbeing Study III. 2022. Page 19. https://www.merck-animal-health-usa.com/wp-content/uploads/sites/54/2022/02/2021-PSV-Veterinary-Wellbeing-Presentation_V2.pdf. Accessed January 28th 2023.

Burgess A, van Diggele C, Mellis C. Mentorship in the health professions: a review. Clin Teach. 2018 Jun;15(3):197-202. doi: 10.1111/tct.12756. Epub 2018 Jan 10. PMID: 29318730.

Reinhard, A. R. (2023). Early Career Veterinary Well-being and Solutions to Help Young Veterinarians Thrive. Advances in Small Animal Care.

Stephenson RN. DOPE 4 bird personality test: dove, owl, peacock, eagle. https://richardstep.com/self-tests-quizzes/dope-bird-personality-test-printable/. Published 11 April 2012. Accessed 14 January 2024.

Leung, V. W., Konci, X., & Meterissian, S. (2021). Is there a role for formal mentorship programs in reducing burnout in surgical residency?: A literature review. International Journal of Surgical Education.

Morioka, H., & Kitaoka, K. (2017). Investigation of the influence of mentors in the workplace environment of nurses, including associated occupational stress and willingness to continue working. J Wellness Health Care, 41(2), 23-39

Dyrbye, L. N., Shanafelt, T. D., Gill, P. R., Satele, D. V., & West, C. P. (2019). Effect of a professional coaching intervention on the well-being and distress of physicians: a pilot randomized clinical trial. JAMA internal medicine, 179(10), 1406-1414.

Erikson, T.(2019) Surrounded by idiots, St Martins Essentials, NY Honey, P. and Mumford, A. (1986a) The Manual of Learning Styles, Peter Honey Associates. Mind Wellness Action Plan, [accessed 26th January 2024]. Available from: https://www.mind.org.uk/media/12145/mind-wellness-action-plan-workplace.pdf The Wellness Recovery Action Plan (WRAP) [(accessed on 26 January 2024)]. Available online: https://copelandcenter.com/wellness-recovery-action-plan-wrap

SELF-EFFICACY – THE MISSING LINK IN LEARNER ASSESSMENTS?

Rita Hanel [1]

[1] Veterinary Emergency Group, Raleigh, United States

Learning objectives:

- Describe the concept of self-efficacy
- Discuss the impact of self-efficacy on computer-based learning
- Discuss how self-efficacy can impact imposter syndrome
- List educational approaches to improving self-efficacy

Proceeding:

Among the various theories that attempt to explain the processes that drive and regulate behavior, one of the most prominent is Badura's (1977) Social Cognitive Theory. Social Cognitive Theory posits that a combination of external social systems and internal self-influence factors motivate and regulate behavior. Self-efficacy, one of these self-influencing factors, is thought to be a major contributor and has been defined as "the belief in one's capabilities to organize and execute a course of action required to produce given attainments". Self-efficacy beliefs influence how individuals think, feel, motivate themselves, and behave. Trainees with high levels of self-efficacy are more likely to set challenging goals, persevere in the face of obstacles, and exhibit resilience in the demanding environment of medical education. Several studies have demonstrated a significant positive relationship between academic self-efficacy and academic performance among university students.

The practice of veterinary emergency medicine can be rigorous and stressful. When training an individual to perform at a high level, self-efficacy plays a crucial role in shaping one's development and success. Medical excellence requires competence in various clinical skills, decision-making, and patient care. To execute efficiently and effectively in an emergent setting on these competencies requires confidence and belief in one's abilities to do so, making self-efficacy a major contributor to performance.

The increased utilization of computer-based learning means that students must learn to self-regulate their learning. This incorporates self-monitoring, self-evaluation, goal setting, and planning, all of which can contribute positively to academic achievement. In studies investigating learning a second language and academic writing, it was found that learners who have a higher sense of motivation and self-efficacy are more likely to engage in such self-regulation and be successful in their performance.

Imposter syndrome is another domain where the evaluation of self-efficacy may be of benefit. In a study evaluating the presence of imposter syndrome in data science students, perfectionism, low self-efficacy, and higher anxiety were noted to be high predictors. One international study evaluating the prevalence of imposter syndrome in veterinarians found that 68% of the 941 practicing veterinarians that responded, using a survey-based scale, met or exceeded the score for imposter syndrome. Given the stress of emergency medicine and the need for rapid goal-oriented decision making, the early identification of areas in which imposter syndrome may be undermining performance may be of benefit.

Although it can be impacted by a myriad of factors, including past experiences, verbal persuasion, observation, and indirect or direct mastery of subject matter, knowledge of its presence and influence may assist with teaching and mentoring veterinarians with respect to clinical performance. Several evidence-based approaches can be employed to promote self-efficacy and support the development of future healthcare professionals, including: 1) the provision of mastery experiences, where trainees are provided opportunities to succeed in challenging tasks and clinical scenarios, 2) vicarious learning through observation and interaction with experts who demonstrate clinical skills and decision-making to inspire trainees, 3) mentorship, peer to peer learning, and collaborative group learning, as well as 4) verbal persuasion offering positive reinforcement and encouragement to highlight strengths and enhance confidence.

In this seminar, we will discuss the concept of self-efficacy as well as the author's experience utilizing it in an emergency doctor training program.

References:

Available upon request

NON-TECHNICAL SKILLS – HOW CAN WE INCORPORATE THIS INTO RESIDENCY TRAINING?

Duana McBride [1]

[1] VetLed, London, United Kingdom

Learning objectives:

- Define non-technical skills (NTS) required by ECC specialists

- Understand the importance of NTS in regards to patient safety and wellbeing of veterinary teams from the perspective of ECC specialists

- Discuss how to incorporate and standardise NTS training into residency programs – what is being done now? What more can we do?

- Address the challenges which may be faced

Proceeding:

NTS are defined as cognitive, social and personal resource skills that complement technical skills, and contribute to safe and efficient task performance. Most common NTS include communication skills, teamwork, leadership, decision making and situation awareness. All of these skills being encapsulated under the definition of human factors, which also include organisational factors and ergonomics, can impact on patient safety, staff/learners' wellbeing, and growth of an organisation or hospital. Deficiencies in NTS has been well described as contributing to medical errors. NTS training has been associated with improved patient outcomes: for example incorporating team training had reduced a critical care teams' time to initiating ECMO; crisis resource management training reducing ICU complication rates, and even improving success rates of CPR from 19 – 67% in one study. With this evidence in mind NTS training is becoming a standard component of human medical training programs.

In small animal veterinary medicine, deficiencies in NTS have also been identified as underlying causes of medical errors and negligence claims, with client communication, team communication followed by drug administration errors being most commonly reported. Although the importance of NTS training in veterinary medicine is becoming more apparent, it has not yet become standard training in undergraduate or post graduate veterinary training.

With a consensus of increased training needs for ECC veterinarians in North America, a competency framework for post graduate training for ECC veterinarians was developed, in which communication, teamwork, conflict resolution, leadership, crisis resource management, time management, floor management, record keeping and selfcare were identified as important NTS for ECC veterinarians. With

this in mind, it is even more timely to consider the important components of NTS required by ECC specialists, and how we can incorporate this into residency training programs.

This panel discussion with experts in the field of ECC residency training, will bring forth the important components of NTS required by ECC residents and specialists; what does NTS training actually look like; and discuss how we can effectively and feasibly incorporate this into residency training programs.

References:

Cooper S, Endacott R, Cant R. Measuring non-technical skills in medical emergency care: a review of assessment measures. Open Access Emerg Med 2010;2:7-17 Kohn L. To err is human: an interview with the institute of medicine's Linda Kohn. Journal Comm J Qual Improv 2000;26(4):227-234

Noyes J, Stewart S, Gabarro J, Welch P. Development of a veterinary emergency open standard competency framework using a competency-based model of medical education. JVECC 2021;31:727-741

Oxtoby C, Ferguson E, White K, MossopL. We need to talk about error: causes and types of error in veterinary practice. Vet Rec 2015;doi:10.1136/vr.103331

Pimental I, Mata N, Bezerra I, et al. Nontechnical skills training in intensive care units: Protocol for a systematic review and meta-analysis. PLoS ONE 2023; 18(1): e0280132

PSYCHOLOGICAL SAFETY

Duana McBride [1]

[1] VetLed, London, United Kingdom

Learning objectives:

- Define psychological safety and how this applies at an organisational, team and individual level.

- Understand how psychological safety is an integral part of safety culture and just culture.

- Develop strategies to ensure psychological safety in the workplace.

- Develop strategies to ensure psychological safety in veterinary education, including internships and residencies.

Proceeding:

Psychological safety is a shared belief that it's OK to take risks, to express ideas and concerns, to speak up with questions, and to admit mistakes — all without fear of negative consequences. It is the understanding and practice that mistakes will be worked on together as a learning opportunity.

Psychological safety has been studied at an organisational level in improving worker engagement, team collaboration, improving job satisfaction and creating high performing teams in many industries. In the medical field, improved psychological safety is linked to patient safety, with improved patient care, interdisciplinary communication and reduced staff turnover; all being part of a hospital's 'safety culture'.

It is evidenced that when we have more active and positive emotional states, our learning and retention is enhanced. Despite this understanding, there is still a large gap between psychological safety as imagined, and the perceived psychological safety of learners in medical education. Psychological safety is well studied and embedded in simulation-based training and debriefing, however there is still a gap within clinical education, especially for interns and residents due to hospital structures and hierarchy.

Psychological Safety and Just Culture

Just Culture refers to a workplace culture that recognises that competent professionals make mistakes. Just Culture focuses on systems-based issues where there is a shared accountability between the organisation (practice) and the individual. A just culture involves a reporting culture and learning culture, where team members cooperate, share their fears, acknowledge mistakes, and strive to continually improve. Therefore, psychological safety is important in developing a healthy workplace culture.

How can we create a psychologically safe environment?

Psychological safety requires a culture which acknowledges fallibility, and a learning culture which models curiosity. It requires openness and safety in speaking up, not just with errors and mistakes but with ideas and questions too. It is important to establish a workplace code of conduct that mistakes are not to be shamed or hidden but are opportunities for sharing, growth, and learning for everyone.

At a leadership level, it is important to create a non-hierarchical environment. Leaders can do this by demonstrating behaviours which are congruent with psychological safety, such as acknowledging their own fallibility when an error occurs, asking questions with genuine curiosity, and involving team members in clinical discussions and debriefings, for example asking, 'what could I have done to make work easier for you today?'.

Part of creating a culture of psychological safety involves creating a safe environment for speaking up. Speaking up like any skill can be developed in team members through training, (e.g. simulation), but it's also important to train all levels of staff in non-judgemental listening with genuine curiosity.

When undertaking simulation-based training and simulation or clinical debriefing, it is important to state ground rules to create a psychologically safe environment for learners.

Psychological safety can be measured in the workplace by periodically conducting anonymous surveys to measure team perceptions on sharing ideas, making mistakes, and feeling included. These surveys can also be considered before and after implementation of tools for psychological safety in a workplace.

Outcome of psychologically safe environments

Studies have shown that a psychologically safe work environment results is better client satisfaction, lower staff turnover, reduced disciplinaries and greater dissemination of learning across the practice. In one NHS trust (NHS MerseyCare) where a Just and Learning Culture was implemented, within less than three years disciplinary cases amongst staff members were reduced by 75%, there was a significant increase in patient safety and a positive increase in staff satisfaction survey results.

References:

Annual Perspective: Psychological Safety of Healthcare Sta¬. PSNet. (2022, October 5). Clark, T.R. (2020).

The 4 stages of psychological safety: Defining the path to inclusion and innovation (illustrated). Berrett-Koehler Publishers. Hofmeister E, Love L.

Patient safety culture is needed in veterinary medicine. JAVMA 2023;261(12):1908-1912 McClintockA, Fainstad T, Blau K, Jauregui J.

Psychological safety in medical education: A scoping review and synthesis of the literature. Medical Teacher 2023;45(11):1209-1299 McKay C, Vaisman J.

Psychological safety, purpose, path, and partnership reduce associate veterinary desire to leave current employment. JAVMA 2023;261(10):1518-1524 O'Donovan R, Van Dun D, McAuliffe E.

Measuring psychological safety in healthcare teams: developing an observational measure to complement survey methods. BMC Medical Research Methodology 2020;20:203 Tsuei S, Lee D, Ho C, Regehr G, Nimmon L.

Exploring the construct of psychological safety in medical education. Academic Medicine 2019;94(11):528-535

Oral Abstracts, Original Study, Thursday 30 May 2024

COMPARISON OF RADIOGRAPHS AND ULTRASOUND FOR DIAGNOSIS OF RIB FRACTURES IN CANINE CADAVERS

Harding C[1], Menard J[1], Knight CG[1], Sukut S[2], Finch A[1], Hillstead M[1], Boysen SR[1]

[1] University of Calgary, Faculty of Veterinary Medicine, Calgary, Canada
[2] Western College of Veterinary Medicine, Saskatoon, Canada

Introduction:

Though controversial, recent evidence suggests ultrasound is superior to radiographs in diagnosing rib fractures in humans. The present study aimed to determine the sensitivity and specificity of point-of-care ultrasound (POCUS) and digital radiography for detection of rib fractures in canine cadavers using necropsy as the reference standard. We hypothesized that sensitivity, but not specificity, would differ between imaging modalities.

Methods:

Ethics were obtained. Nine canine cadavers were randomly assigned to a fracture or control group. Rib fractures were created by making a small skin incision, scoring the superficial rib surface with bone cutters, and using a chisel and hammer to complete the fracture(s). Randomization allowed ribs to have more than one fracture. Similar skin incisions were made in all cadavers and subsequently sutured. An expert (>20 years POCUS experience) and novice sonographer (final-year vet student) blinded to fracture location and number scanned cadavers bilaterally. Post-sonographic three-view thoracic radiographs were assessed by a blinded expert (board-certified radiologist) and novice (final-year vet student). The time to sonographically assess cadavers and interpret radiographs, and the number and localization of rib fractures was recorded. D'Agostino & Pearson and One-way ANOVA/Freidman's were used to test normalcy and compare continuous variables between groups, respectively, and categorical variables were analyzed using Chi-square ($P<0.05$ = significance). Sensitivity and specificity were calculated using necropsy as the reference standard. Data reported as mean +/- SD or median (min-max).

Results:

Fifty rib fractures were created. Overall sensitivity and specificity for fractures were 83%, 99.74%, and 82%, 99.22% for ultrasound and radiographs, respectively, with no statistical difference between groups. Time to scan or read radiographs for novice and expert was 64.7+/-12.7 and 26.4+/-7.9 mins, and 9.2 (7.7-15) and 3.1 (2.2-8.2), respectively. There was a statistically significant difference in time between groups ($P<0.0001$), with Dunn's post-hoc analysis identifying the difference between the radiologist and both sonographers and between the student radiograph reader and student sonographer.

Conclusions:

POCUS and radiographs have equally good to excellent sensitivity and specificity in identifying rib fractures in canine cadavers. Excluding the time to obtain radiographs, it takes much longer to identify rib fractures with ultrasound than to read radiographs.

E-mail: roseva.harding@ucalgary.ca

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

COMPARISON OF POINT-OF-CARE ULTRASOUND AND BLINDED ANATOMICAL LANDMARK THORACOSTOMY TUBE INSERTION IN CADAVER DOGS

K.G. Gillett[1], S.B. Boysen[1], C.H. Harding[1], A.F. Finch[1], M.H. Hillstead[1], J.M. Menard[1]

[1] University of Calgary, Calgary, Canada

Introduction:

Small-bore wire-guided thoracostomy tubes (SBWGTT) may result in serious complications including iatrogenic lung perforation. We aimed to determine if POCUS-guided SBWGTT placement improves ease of placement, confidence in placement, and decreases complication rates compared to blind landmark techniques. We hypothesized that POCUS-guided placement would outperform blind landmark placement in all categories.

Methods:

This was a cross-over randomized cohort study. Animal and human ethics were obtained. A total of thirty 2nd and 3rd year DVM students were enrolled. Students completed an online training module, practiced POCUS-guided vascular access on a chicken breast phantom model, and POCUS-guided SBWGTT using a pork rib & bucket model. Each student was then randomized into 2 of 4 cadaver groups: POCUS-guided (U) and landmark-guided protocols (L) for either pleural effusion (PE) or pneumothorax (PN). Students then placed SBWGTT's into 22 canine cadavers with bilateral iatrogenic pleural effusion and pneumothorax. Placement time, student confidence in SBWGTT placement and ease of placement (using weighted scales), and complications (determined by necropsy) were recorded. D'Agostino and Pearson were used to test normalcy, and a Kruskal Wallis, Fisher's exact test or one-way ANOVA with Tukey's correction used for statistical analysis.

Results:

Data were non-parametric and parametric. One SBWGTT was missing at necropsy. LPE SBWGTT placement (median: 5.5 min IQR: 4.3-6.1) was faster than UPN (9.5 min IQR: 7.2-10.9, p= 0.0118), and LPN (4.4 min IQR: 3.5-5.6) was faster than UPN (p= 0.0205) and UPE (7.1 min IQR: 5.1-10.9, p= 0.0004). Eleven SBWGTT's were placed extra-pleurally (3 LPN, 3 LPE, 1 UPE, 4 UPN) with no significant difference between groups. Four UPE SBWGTTs had intrathoracic complications (retained broken guidewire, two lung penetrations, one tube placed < 1 cm intra-pleurally), which was not statistically significant between groups (Fisher's exact test p> 0.005). Novices felt more confident SBWGTTs were intra-pleural when using POCUS; UPN (mean 7.25 ± 2.41) vs. LPN (4.39 ± 2.65, p= 0.0232), and LPE (4.61 ± 2.68) vs. UPE (7.12 ± 2.86, p= 0.0326).

Conclusions:

Compared to blind landmark placement, POCUS-guided SBWGTT placement performed by novices was slower, had similar complication rates, and greater confidence scores.

E-mail: julie.menard@ucalgary.ca

A LOW-COST SONOGRAPHIC CANINE SIMULATOR FOR PERICARDIOCENTESIS TRAINING

M. Hillstead[1], J Menard[1], S.R. Boysen[1], N. Perewernycky[2], K. Gillbert[1]

[1] University of Calgary Faculty of Veterinary Medicine, Calgary, Canada
[2] University of Calgary Faculty of Biomechanical Engineering, Calgary, Canada

Introduction:

Pericardiocentesis (PC) is often taught in the clinical setting, which can be stressful. By contrast, PC simulation allows clinicians to train in a risk-free context. This study aimed to construct a low-cost sonographic simulator to improve novice confidence in performing PC. We hypothesize that clinicians experienced in performing PC will assess the simulator's sonographic appearance and tactile performance as realistic and that novice veterinarians' confidence in performing PC will improve with simulator practice.

Methods:

Human ethics were obtained. The right hemithorax was simulated using ballistic gel, silicon foam and a three-dimensional printed spine with ribs. A tennis ball within a balloon, both filled with water, simulated pericardial effusion (PE). After performing ultrasound-guided PC on the simulator, experts (> 5-10 PC procedures and > 3 years of emergency experience) assessed the simulator using a five-point Likert scale survey (1; strongly agree, 5; strongly disagree) for realism regarding sonographic appearance of PE and surrounding structures and tactile feel of catheter advancement. Before and following simulator practice, interns completed a survey (4-point Likert scale) assessing their confidence performing and steps required to perform ultrasound-guided PC. Results were tested for normality using the D'Agostino-Pearson test. Parametric data is presented as mean +/- standard deviation and non-parametric as median and interquartile range. Pre and post-confidence levels were compared using paired T-tests/Wilcoxon tests depending on normalcy results.

Results:

Experts rated sonographic appearance of PE as 2.00+/- 0.5, tactile feel of catheter advancement into the simulated PE as 2.56+/- 0.9, and comparability of performing PC on the simulator to PC on a live canine as 2.00 [2.00-2.00]. Interns had a significant increase in their confidence to perform pericardiocentesis in a live patient ($p = 0.03$), sonographic visualization and guidance of the catheter ($p = 0.02$), and correct placement of the catheter stylet within the simulated pericardial space ($p = 0.02$).

Conclusions:

The constructed simulator realistically simulates naturally occurring PE seen in dogs and improves novice operator confidence in performing PC. Further research is required to know if simulation-acquired skills apply to the identification and treatment of canine pericardial effusions in the clinical setting.

E-mail: Madison.hillstead@ucalgary.ca

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

POINT-OF-CARE ULTRASOUND OF GASTRIC ANTRUM TO ESTIMATE GASTRIC CONTENT AND VOLUME IN CRITICALLY ILL DOGS

P.A. Vidal[1 2], L. Bouvet[2 3], B. Allaouchiche[2 4 5], A. Nectoux[1 2], C. Pouzot-Nevoret[1 2]

[1] VetAgro Sup campus vétérinaire de Lyon, Université de Lyon, Marcy l'Etoile, Lyon, France
[2] UPSP 2021.A101, Pulmonary and Cardiovascular Aggression in Sepsis APCSe, Lyon, France
[3] Hospices Civils de Lyon, Hôpital Femme Mère Enfant, Lyon, France
[4] Hospices Civils de Lyon, Centre Hospitalier Lyon-Sud, Lyon, France
[5] Université Claude Bernard, Lyon1, Lyon, France

Introduction:

Delayed gastric emptying is a common complication in critically ill dogs. This can have a negative impact on recovery and can lead to several complications. In human medicine, point-of-care ultrasound measurement of antral area (AAPOCUS) is used to non-invasively assess gastric residual volume (GRV). The objectives of this prospective observational study were to evaluate the feasibility and the interobserver reliability of AAPOCUS, and to determine the relationship between AAPOCUS measurement and GRV.

Methods:

Dogs hospitalized in the ICU of VetAgro Sup (Lyon, France) with a nasogastric tube between October 2021 and December 2023 were prospectively enrolled. In the first part of the study, to determine inter-observer reliability, two operators performed 3 gastric antrum measurements per dog on 13 dogs. The intraclass correlation coefficient was calculated to evaluate inter-observer reliability, and the concordance correlation coefficient was also calculated to estimate the reproducibility between the 2 observers. In the second part, to determine the relationship between AAPOCUS and GRV, 3 measurements were obtained at 3 time points by the same operator: before aspiration of the GRV; after aspiration and after administration of an enteral nutrition bolus. During AAPOCUS, dogs were placed in right lateral recumbency, with the transducer positioned on the midline just caudal to the xiphoid. A linear multiple regression analysis was performed to model the relationship between the gastric volume administrated during enteral nutrition and the logarithm of the antral area. The model was built from the volume ingested in animals with an empty antrum after aspiration, in order to have the most accurate model possible. The R^2 value corresponding to the goodness of fit of this model was calculated.

Results:

Interrater reliability was high, with intraclass correlation coefficient of 0.99 (95%CI: 0.96 to 0.99), and concordance correlation coefficient of 0.97 (95%CI: 0.91 to 0.99). Twenty-two dogs were included in the second part of the study, leading to 333 AAPOCUS. Median GRV and median volume of nutrition administrated were respectively 2 (IQR: 0.65 to 5) ml and 61 (IQR: 39.25 to 85.25) ml. Antral area was significantly increased after enteral nutrition. This result confirms that the antral surface area is a marker of gastric volume. The prediction of gastric volume was as follows: gastric volume = 22 x log (antral area, cm^2) +2.29 x body weight (kg) - 1. The R^2 was 0.56.

Conclusions:

As described in human medicine, POCUS assessment of antral area is feasible in critically ill dogs and can estimate gastric volume using a mathematical model.

E-mail: dr.pierreandrevidal@gmail.com

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

NASOGASTRIC FEEDING TUBE POSITION CONTROLLED USING POINT-OF-CARE ULTRASOUND COMPARED TO X-RAYS: A PRELIMINARY STUDY ON 39 CASES

A. Nectoux[1 2], E. Faucher[1 2], P.A. Vidal[1 2], B. Allaouchiche[2 3], C. Pouzot-Nevoret[1 2], L. Bouvet[2 3]

[1] VetAgro Sup, SIAMU, Marcy l'Etoile, France
[2] APCSe, Université de Lyon, Marcy l'Etoile, France
[3] Hospices Civils de Lyon, Pierre Bénite, France

Introduction:

Early enteral nutrition is a key point in critically ill patients management, mostly using feeding tubes. Nasogastric tube (NGT) is useful to remove and quantify gastric residual volume. Combination of esophageal and gastric POCUS images is an efficient technique to confirm NGT position in human medicine. Mid-neck point-of-care ultrasound (POCUS) showed excellent sensitivity and specificity to confirm the esophageal position of NGT in dogs and cats. The aims of this study were to assess the feasibility and the accuracy of POCUS to confirm the gastric position of NGT compared to radiographs, in critically ill dogs and cats.

Methods:

Animals hospitalized in the ICU of VetAgro Sup (Lyon, France) with indication of NGT placement were prospectively enrolled from April 2023 to March 2024. After NGT placement, POCUS images were recorded by one operator, blinded of NGT placement modalities and before radiographic confirmation. Direct visualization of the tube in the stomach was assessed with the sub-xiphoid view (STO) using a 5MHz microconvex probe. Indirect position of the tube was assessed by visualization of echoic contrast in the stomach after rapid inflation of 15mL of air (AIR) via the NGT. After POCUS, a right lateral thoracic radiograph was performed. The position of the NGT and time to obtain images were compared using Wilcoxon non-parametric test.

Results:

Fifty-eight animals were included (37 dogs and 21 cats). Radiographs confirmed that 1/58 tube was placed in the respiratory tract. Of the 57 digestive tubes, 43 ended in the stomach and 14 in the esophagus with 3/14 before the 7th rib space. Only 22/43 NGTs were detected with the STO view. To confirm a gastric location of the NGT tip, the best sensitivity and specificity were obtained using the combination of STO and AIR with 67% (95% CI 51-81) and 73% (95% CI 45-92), respectively. Radiographs were obtained in a median of 11 [5-15] minutes and POCUS images in a median of 6 [5-8] minutes (p=0.0008).

Conclusions:

In critically ill dogs and cats, POCUS is not reliable for confirming the gastric ending of an NGT.

E-mail: alexandra.nectoux@vetagro-sup.fr

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

THE EFFECT OF PACKED RED BLOOD CELL TRANSFUSIONS ON AMMONIA LEVELS IN DOGS

E. Gomez-Soto[1], H. Enevoldsen[2], E. Llewellyn[1], J. Wolf[1]

[1] Hospital for Small Animals of the Royal (Dick) School of Veterinary Studies, Midlothian (Edinburgh), United Kingdom
[2] The Ralph Veterinary Referral Centre, Marlow, United Kingdom

Introduction:

Ammonia concentration within stored packed red blood cells (pRBC) is known to increase over time. Hyperammonemia causing severe clinical signs secondary to pRBC transfusions have been reported in human medicine; however, there is minimal data regarding the clinical significance in veterinary medicine. This study aims to evaluate plasma ammonia concentrations and associated clinical signs following pRBC transfusion in dogs.

Methods:

Prospective study. Dogs requiring a pRBC transfusion with owner's consent for a small volume of additional blood to be collected were included in the study. A neurological examination was performed before and after the pRBC transfusion. Prior to transfusion, PCV and ammonia concentration were measured in each dog and pRBC unit. PCV and ammonia concentration were measured post-transfusion at 1 hour and 18-24 hours. Signalment, presumptive diagnosis, age of the pRBC unit and the pRBC dose (ml/kg) were also recorded. Parametric variables are expressed as mean ± standard deviation and non-parametric variables as median and range. Pre-transfusion and post-transfusion ammonia levels were compared and associations between pRBC unit age, pRBC ammonia concentration and post-transfusion ammonia concentrations were evaluated.

Results:

Eighteen dogs were enrolled. The mean pRBC unit age and ammonia concentration were 22.12 days (±9.86) and 161.1umol/L (±85.99), respectively. Mean pRBC transfused volume = 12.61ml/kg (±3.27). The median ammonia concentrations (reference range 0-98) of all dogs pre- and 1-hour post-transfusion were 0umol/L (0-52) and 2umol/L (2-40), respectively. There was no significant difference between pre-transfusion and maximal ammonia concentration post-transfusion ($p= 0.57$). pRBC unit age significantly correlated with the pRBC ammonia concentration ($r = 0.86$, $p<0.001$). No correlations between ammonia concentrations of the pRBC units and 1h- and 18-24h post-transfusion were found ($r= 0.24$, $p= 0.37$ and $r= -0.54$, $p= 0.09$ respectively). No deterioration of neurological clinical signs was noticed in any dog post-transfusion. None of the dogs enrolled had hepatic dysfunction.

Conclusions:

Ammonia concentration in canine pRBC units increases over time during storage reaching high levels before the expiration date. In dogs with presumed normal hepatic function, ammonia concentrations do not significantly increase after pRBC administration.

E-mail: e.gomez-soto@sms.ed.ac.uk

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

INFLUENCE OF ESTIMATED COSTS ON EUTHANASIA DECISION FOR CATS PRESENTED WITH TRAUMA TO AN EMERGENCY CENTER IN NEW ZEALAND: A RETROSPECTIVE ANALYSIS

K. Houben[1], F.A. Avendaño Veas[2], W.K.A. Tan[1], I.D. Yozova[1]

[1] School of Veterinary Science - Tāwharau Ora, Massey University, Palmerston North, New Zealand
[2] School of Agriculture and Environment, Massey University, Palmerston North, New Zealand

Introduction:

Reported mortality rates in cats with trauma ranges from 8-20%. Previous studies suggest the influence of the cost of care on decisions for euthanasia. The aim of this study was to investigate the influence of estimated cost in the decision for euthanasia over treatment in cats presenting after trauma in an afterhours first opinion and referral emergency veterinary hospital in New Zealand (Manawatu region).

Methods:

Signalment, clinical parameters, patient outcomes, veterinary recommendation for euthanasia, mention of financial constraint by clients and welfare euthanasia (without payment by client) were extracted from medical records of cats presenting for trauma between September 2013 and February 2023. Trauma severity was scored using the Animal Trauma Triage score (ATT) and allocated into non-severe (ATT ≤4) and severe (ATT ≥5) trauma groups. Estimated costs were calculated retrospectively for all cases. Statistical analysis included Shapiro-Wilk test for normality, Kruskal Wallis rank sum test and Wilcoxon rank sum test for association between cost and trauma severity. The statistical effect of the variables cost, trauma severity, age and euthanasia recommended on the response variable euthanasia was assessed through a generalized linear mixed effects model.

Results:

1179 cats met the inclusion criteria of which 25 cats died due to their condition and 240 cats were euthanized. Among non-survivors (n=265), 36% were euthanized due to financial constraints and 7% on a welfare basis. The generalized linear mixed effect model established that estimated cost and ATT independently and the interaction between both had significant effects on euthanasia (p-value <0.001 for all three), even after adjusting for confounding factors such as age and veterinarian recommendation for euthanasia. Among non-survivors, cats with severe trauma had significantly higher estimates compared to cats with non-severe trauma (p-value <0.001).

Conclusions:

Our study demonstrated that in a predominantly first-opinion emergency setting, estimated cost has a significant effect on the mortality in cats presenting for trauma. The effects of cost should be incorporated into survival analysis for cats with trauma.

E-mail: houbenkaat@gmail.com

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

INTRAVENOUS ADMINISTRATION OF VITAMIN K1 IN DOGS AND CATS PRESENTED FOR ANTICOAGULANT RODENTICIDE POISONING: A RETROSPECTIVE STUDY OF 59 CASES

C. Dartencet[1 2], C. Pouzet-Nevoret[1 2], M. Poupon[1], M. W. Kim[1 2], B. Allouchiche[2 3], A. Nectoux[1 2]

[1] VetAgroSup, Marcy L'Etoile, France
[2] VetAgroSup, APCSe, Marcy L'Etoile, France
[3] Hospices Civils de Lyon, Pierre-Bénite, France

Introduction:

Anticoagulant rodenticide poisoning is a common life-threatening condition in veterinary medicine. Anticoagulant rodenticides inhibit vitamin K1 2,3-epoxide reductase. Emergency treatment consists in vitamin K1 and blood transfusion. Anaphylactoid reactions have been described with intravenous (IV) injection of vitamin K1. In some countries, lecithin solubilizing excipient allows IV administration of vitamin K1 (VITAMINE K1 INJECTABLE, TVM®). Our objectives were to describe side effects of IV vitamin K1 administration, and to compare outcomes between vitamin K1 administration only or combined with exogenous coagulation factors.

Methods:

Dogs and cats presented between January 2010 and January 2022 for anticoagulant rodenticide poisoning at the ICU of VetAgro Sup, were retrospectively enrolled. Diagnosis was based on toxin detection in patient blood or urine (LC-MSMS technique), or rapid normalization of coagulation times with clinical improvement after IV vitamin K1. We identified two groups: patients who received vitamin K1 ± packed red blood cells or autotransfusion (without coagulation factors) (VITK1) and patients who received vitamin K1 and whole blood or fresh frozen plasma (containing coagulation factors) (CF). Outcomes studied were: physical exam findings, packed cell volume (PCV), coagulation times, length of hospitalization and survival to discharge.

Results:

Fifty-nine patients were enrolled. All except one patient, received IV vitamin K1 at 5mg/kg. No patients had any side effects after IV vitamin K1 administration. All patients had normalization of their coagulation times within 24 hours. Dogs in the CF-group had lower PCV compared to dogs in the VITK1-group (17% *vs* 32%, p=0.0011). Between the VITK1-group and the CF-group, there was no significant difference in survival to discharge (96% *vs* 85%, p=0.22, respectively) and length of hospitalization for cats ((2 days [2; 2.5]; 2 days [2; 3], p=0.92, respectively). Dogs in the VITK1-group had shorter length of hospitalization compared to dogs in the CF-group (2 [1; 2.8] *vs* 2 [2; 3] days (p=0.028), respectively).

Conclusions:

Intravenous administration of vitamin K1 with lecithin solubilizing excipient appears to be safe, with no reported adverse effects. In AR-poisoned patients, IV vitamin K1 without exogenous coagulation factor administration appears to rapidly normalize coagulation times, with good survival and short length of hospitalization.

E-mail: camille.dartencet@vetagro-sup.fr

OUTCOME PREDICTION AND SURVIVAL IN CRITICALLY ILL DOGS WITH ACUTE KIDNEY INJURY UNDERGOING CONTINUOUS RENAL REPLACEMENT THERAPY

H. Raskansky[1], Y. Bruchim[1], R. Nivy[1]

[1] Tipul Nimratz Ben Shemen, Ben Shemen Youth Village, Israel

Background:

Continuous renal replacement therapy (CRRT) is routinely used in humans with acute kidney injury (AKI) but studies in dogs are scarce. The objective of this study was to assess the utility of the APPLEfull score for outcome prediction in dogs undergoing CRRT.

Methods:

Medical files of 30 dogs were retrospectively reviewed. The APPLEfull score was calculated upon admission, and receiver-operator characteristic curve (ROCC) was constructed to assess its utility for outcome prediction. The CRRT dose intensity was determined using KDIGO recommendations and adjusted individually.

Results:

Presumptive etiologies for AKI included heatstroke, pyelonephritis, hemoabdomen, trauma, hemolysis, pancreatitis, snake bite, and splenic torsion. Anuria/oliguria developed in 21/30 dogs, despite diuretic therapy. Median (IQR) serum creatinine at CRRT initiation, at discharge, and 1-month post-discharge was 9.2 mg/dL (7.1 mg/dL), 3.4 mg/dL (1 mg/dL) and 1.5 mg/dL (1 mg/dL), respectively. The median (IQR) number of CRRT sessions was 2 (2), with a median (IQR) cumulative treatment duration of 46 (86.5) hours. The prescribed median CRRT dose was 29 mL/kg/hr (IQR, 18.5 mL/kg/hr), with a median Kt/V of 3.39 (IQR, 1.49). Eleven dogs survived to discharge and 3-months post-treatment. The area under the ROCC for the APPLEfull score as survival predictor was 0.87 (95%CI, 0.74-0.99), with an optimal cut-off point (<33.5) yielding a sensitivity/specificity (95%CI) of 100% (74%-100%) and 63% (41%-81%), respectively.

Conclusions:

In this cohort of dogs, CRRT demonstrated comparable efficacy to previous reports of dogs undergoing medical or renal replacement therapy. The APPLEfull score proved a clinically useful ancillary tool for outcome prediction herein.
E-mail: hillipomi@gmail.com

PRELIMINARY EVALUATION OF THE OXYGEN RESERVE INDEX AS A SURROGATE FOR THE ARTERIAL PARTIAL PRESSURE OF OXYGEN IN ANESTHETIZED DOGS

F. Zanusso[1], L. Bellini[1]

[1] University of Padova, Legnaro, Italy

Introduction:

The oxygen reserve index (ORi) is a continuous non-invasive parameter measured by multi-wavelength pulse CO-oximetry, which detects hemoglobin absorption in arterial and venous blood. Unlike conventional pulse oximetry, which solely measures arterial oxygen saturation and plateaus at arterial partial pressure of oxygen (PaO_2) >100 mmHg, multi-wavelength pulse CO-oximetry estimates oxygenation in mild hyperoxemia when venous oxygen is not fully saturated. In particular, ORi was developed in humans to estimate PaO_2 between 100 and 200 mmHg, making it useful for titrating long-term oxygen supplementation. This study assesses the ability of ORi to serve as a surrogate for PaO_2 and to quantify non-invasively the ratio between PaO_2 and inspired fraction of oxygen (FiO_2) in anesthetized dogs.

Methods:

In this retrospective analysis, values of ORi between 0.1 and 0.9 were considered from a prior study involving 37 adult anesthetized and mechanically ventilated dogs. Simultaneously collected measurements of PaO_2 and FiO_2 with ORi values were also analyzed. Linear regression analysis was performed, to calculate the correlation (r^2) between ORi and PaO_2 and to establish an equation for estimating PaO_2 (PaO_2est) from ORi value. Pearson correlation coefficient (r) was calculated between PaO_2est and PaO_2 and between ORi/FiO_2 and PaO_2/FiO_2. The agreement between PaO_2est and PaO_2 was assessed using Bland–Altman analysis.

Results:

A total of 85 ORi measurements ranging from 0.1 to 0.9 were analyzed. Mean PaO_2 value was 146.8±30.6 mmHg. Value of FiO_2 was 0.32±0.07. A moderate positive correlation was found between ORi and PaO_2 (r^2=0.35, p< 0.0001). Correlation was moderate (r=0.59) between PaO_2est and PaO_2 and strong (r=0.72) between ORi/FiO_2 and PaO_2/FiO_2. Bland–Altman agreement analysis between PaO_2 and PaO_2est showed a mean bias of −0.002 mmHg, with 95% limit of agreement of −48.2 to 48.2 mmHg.

Conclusions:

An ORi value between 0.1 and 0.9 might represent a surrogate of PaO_2 in anesthetized mechanically ventilated dogs receiving supplemental oxygen, although it may show a clinically significant over- or under-estimation of the level of oxygenation. The ORi/FiO_2 ratio might early identify alterations in gas exchange and guide oxygen titration in anesthetized dogs, although arterial blood gas analysis remains ideal for assessment of oxygenation.

E-mail: francesca.zanusso@studenti.unipd.it

EVALUATION OF SERUM MIRNA EXPRESSION AS BIOMARKERS OF BRACHYCEPHALIC OBSTRUCTIVE AIRWAY SYNDROME SEVERITY IN FRENCH BULLDOGS

S.M. Cutler[1], K. Blacklock[1], S. Guizard[1], D. Shaw[1], R. Clark[1], D. Dunbar[1], A. Fawkes[1], E. Llewellyn[1]

[1] University of Edinburgh, Edinburgh, United Kingdom

Introduction:

Determination of brachycephalic obstructive airway syndrome (BOAS) severity in dogs is complex and currently focuses on subjective assessments of conformation, frequency of appropriate clinical signs and exercise tolerance tests. MicroRNAs (miRNAs) are small, non-coding RNAs that are important in the regulation of post-transcription gene expression and there is growing evidence of miRNAs as useful biomarkers for several diseases. The aim of this study is to determine whether serum miRNAs are reliable predictive biomarkers of BOAS severity in French bulldogs.

Methods:

A prospective study involving serum samples collected from three cohorts of client-owned dogs: French bulldogs with clinical signs of BOAS requiring surgical intervention (n=15; Group 1); French bulldogs with no clinical signs or history of BOAS undergoing general anaesthesia (n=15, Group 2); and healthy, non-brachycephalic dogs (n=15; Group 3; control group). Clinical assessment of BOAS severity for each French bulldog included Cambridge Respiratory Functional (CRF) Grading Scheme assessment, airway examination grading and owner completion of a questionnaire on the frequency of BOAS-related respiratory/gastrointestinal clinical signs. Serum miRNA was isolated and sequenced using next-generation sequencing (NextSeq 2000 platform). miRNA analysis was performed using two nf-core pipelines to generate a counting matrix with reads mapped against two reference databases, miRbase and mirdeep2; and aligned against a reference canine genome. Differentially expressed miRNAs (> ±1.5-fold change; $p<0.05$) were identified and compared between the 3 cohorts.

Results:

Median CRF Scores for Group 1 and Group 2 were: 2 (range 1-3) and 1 (all scores 1), respectively (chi-square, p=0.001). Airway examinations of all dogs in Group 1 (15/15) were grade 1, 47% (7/15) of dogs in Group 2 were grade 1 and 53% (8/15) grade 0 (chi-square, p = 0.002). The counting matrix comprised 451 miRNAs, with 7 miRNAs differentially expressed between the 3 groups, including one novel miRNA. miRNA-122 expression was upregulated in Group 1 compared to Group 2. Upregulation of miRNA-1, miRNA-125a, miRNA-125b, miRNA-143, and miRNA-145 in Group 1 compared to Group 3 was also identified.

Conclusions:

miRNAs are differentially expressed in French bulldogs and show promise as biomarkers of BOAS severity with the potential for primary/referral clinicians to encourage responsible breeding.

E-mail: simonecut08@gmail.com

DETERMINATION OF HIDDEN FLUID VOLUME IN CANINE INTENSIVE CARE PATIENTS - HIDDEN FLUIDS IN CRITICALLY ILL DOGS: THE HEROS TRIAL

C. Guillet[1], I. Hennink[1], B. Vidondo[2], K. N. Adamik[1]

[1] Department of Clinical Veterinary Medicine, Vetsuisse Faculty, University Bern, Bern, Switzerland
[2] DCR-VPH, Vetsuisse Faculty, University Bern, Liebefeld, Switzerland

Introduction:

Undocumented "hidden" fluid volume (HFV) is a major contributor of total intravenous (IV) fluids in human critical care, potentially leading to fluid overload. Comparable data on HFV in dogs remain unexplored. This study aimed to quantify the HFV and its sources in critically ill dogs, presenting preliminary results from an ongoing study.

Materials and Methods:

This single-center, prospective, observational study included dogs admitted to the emergency room and hospitalized for at least 36h and up to 72h in the Intensive Care Unit. The volume of all types of fluids administered IV was precisely recorded. HFV included all non-prescribed IV fluids, such as line flushes, medication vehicles, and diluents.

Results:

The study included 63 dogs, with a mean bodyweight (BW) of 17 kg (range, 1.3-44.0) and a median age of 6 years (range, 0.3-15). The distribution of disease groups was as follows: sepsis (21 dogs), medical (18 dogs), neurologic (13 dogs), surgery (9 dogs), and trauma (2 dogs). The mean $APPLE_{full}$ score was 29 (range, 16-54). From admission (day 1) to day 2, BW changed by a mean of 0.0% (range, -10% to +12%), and from day 2 to day 3 by a mean of 0.2% (range, -9% to +8%). None of the dogs had evidence of peripheral edema. Across days 1 to 3, the percentage of HFV relative to the total IV volume in 24h (%HFV) (median, range) was 4.5% (0.1-67.8), 6.5% (1.5-54.1), and 5.9% (1.5-56.8). Dogs with a BW <10kg (n=21) received a higher %HFV (median, range) compared to dogs >10 kg (n=42) at day 1 (14.0% [0.3-67.8] vs. 3.7% [0.1-25.6]; P=0.010), day 2 (8.5% [2.3-54.1] vs. 5.5% [1.5-26.9]; P=0.096), and day 3 (8.7% [3.4-56.8] vs. 4.8% [1.5-19.6]; P=0.036). In dogs receiving drugs via constant rate infusion(s), %HFV was higher than in dogs that did not (day 1-3, P=0.008; P=0.002; P=0.075). There was no evidence of an association between the disease category or the $APPLE_{full}$ score and the %HFV.

Conclusions:

BW <10kg and drug administration via constant rate infusion were important contributors to higher HFV which must be considered when treating critically ill small-breed dogs.

E-mail: katja.adamik@unibe.ch

EFFECTS OF ACUTE INTRAVASCULAR VOLUME OVERLOAD ON ENDOTHELIAL GLYCOCALYX THICKNESS IN HEALTHY CATS – PRELIMINARY RESULTS

I.D. Yozova[1], J.S. Munday[1], F.A. Avendaño Veas[2], M. Irie[3], N.Thomson[1]

[1] School of Veterinary Science - Tāwharau Ora, Massey University, Palmerston North, New Zealand
[2] School of Agriculture and Environment, Massey University, Palmerston North, New Zealand
[3] Shikoku Veterinary Medical Center, Miki, Kagawa Prefecture, Japan

Introduction:

Previous studies suggest that the endothelial glycocalyx is negatively affected by volume overload. This study assessed the effects of transient intravascular volume overload on endothelial glycocalyx thickness in healthy cats, using side-stream darkfield videomicroscopy and the Glycocheck-TM software.

Methods:

This was a single-centre prospective blinded cross-over experimental study on twenty healthy cats. Cats were sedated with gabapentin PO and butorphanol SC and induced and maintained with propofol CRI during each timepoint. They were randomized to either receive 30 ml/kg of LRS over 10 min (intravascular volume overload group) or not (control) and crossed over after a washout period of 7 days. Sublingual mucosa videomicroscopy images were acquired at T1 (baseline), T2 (immediately after intervention), T3 (3h after end of intervention) and T4 (24h after end of intervention) with three measurements performed per timepoint. The perfused boundary region (PBR), an inverse estimate for endothelial glycocalyx thickness, for vessels with diameter of 5-25, 5-9, 10-19 and 20-25 µ, respectively, was then automatically calculated with the Glycocheck-TM. A linear mixed model was used for normally distributed data and a generalized linear mixed model for not normally distributed data analysis.

Results:

There were no statistically significant differences between groups in all timepoints. In the intravascular volume overload group, there was a significant decrease in PBR 5-25 µ between T2 and T4 ($p=0.016$) and T3 and T4 ($p=0.003$), decrease in PBR 5-9 µ between T1 and T4 ($p=0.007$) and T2 and T4 ($p=0.045$), decrease in PBR 10-19 µ between T2 and T4 ($p=0.012$) and T3 and T4 ($p=0.011$), respectively. There was a significant decrease in PBR 5-25 µ between T3 and T4 ($p=0.011$), increase in PBR 10-19 µ between T1 and T3 ($p=0.031$) and decrease between T3 and T4 ($p=0.032$) in the control group.

Conclusions:

The significant differences between time points in the intervention group suggest that acute intravascular volume overload could lead to discrete transient changes in endothelial glycocalyx thickness. However, as there were no significant differences between groups and a few significant differences within the control group, it cannot be certain these differences were due to the intervention in this preliminary analysis. This is the first study evaluating effects of intravascular volume overload on the microcirculation in cats.

E-mail: i.yozova@massey.ac.nz

Oral Abstracts, Nurse & Technician Case Reports, Friday 31 May 2024

FROM FLAMES TO CARE: MECHANICAL VENTILATION IN A DOG AFTER SMOKE INHALATION

M. Decellieres, J. Combet-Curt [1], M.W. Kim[1], C. Pouzot-Nevoret[1]

[1] VetAgro Sup, Marcy l'Etoile, France

Introduction:

Smoke inhalation in small animals is poorly described but neurological and respiratory signs have been reported. Respiratory irritants and toxins in inhaled smoke can burn the skin, eyes, and upper respiratory tract. Respiratory failure and other complications can develop, necessitating intensive nursing care.

Synopsis:

A 7-month-old intact male Boston terrier, was referred to SIAMU, the ICU at VetAgro Sup, due to worsening respiratory distress post-smoke inhalation five days earlier. Severe inspiratory distress due to upper airway obstruction, immediate endotracheal intubation at admission. Despite intubation, the dog remained in respiratory distress and mechanical ventilation was initiated.

An arterial catheter, a urinary catheter, and a nasogastric tube were placed to monitor blood pressure, ensure comfort and hygiene, and facilitate aspiration of residual gastric volume to minimize the risks of aspiration. Aspiration was conducted as needed for secretions, alongside nebulizations to clear his airways followed by respiratory physiotherapy. Due to frequent obstruction of the upper airways, several suctioning or life saving re-intubation were performed.

Youpi received specific care involving applying ointment around his mouth to ensure his tongue stayed moist, administering eye drops to prevent ulcers, and maintaining the cleanliness of his urinary catheter. Physiotherapy for his limbs aimed to prevent joint stiffness and promote functional recovery. Finally, he received a skincare for burns, using disinfectant wipes and ointment.

A large 10.5 cm airway plug of debris was removed during a ETT changeover, allowing rapid improvement of respiratory mechanics and successful weaning 36 hours after start of ventilation. The patient improved rapidly and was discharged after 5 days of hospitalization.

Conclusions:

Due to a high risk of moderate to major complications, patients under mechanical ventilation require intensive nursing care. This is the first case report of a dog surviving to discharge following mechanical ventilation to treat severe respiratory distress, tracheal mucosal necrosis, and airway obstruction due to smoke inhalation.

E-mail: maxence.decellieres@vetagro-sup.fr

TEMPORARY TRACHEOSTOMY TUBE MANAGEMENT IN A DOG WITH BOAS

J. Salama[1], M. Kim[1], C. Pouzot-Nevoret[1], A. Nectoux[1]

[1] VetAgro Sup - SIAMU, Marcy l'étoile, France

Introduction:

Brachycephalic breeds are more prone to a series of upper airway abnormalities known as Brachycephalic Obstructive Airway Syndrome (BOAS). BOAS causes life-threatening complications. Most BOAS animals have noisy breathing (and respiratory discomfort) and less frequently gastrointestinal signs (regurgitation). BOAS can be treated both medically and surgically. In cases with severe concurrent laryngeal collapse, a temporary tracheostomy tube may be placed. Such patients require intensive nursing care to minimize complications such as wound infection and acute airway obstruction.

Synopsis:

A 3-year-old male castrated French Bulldog presented with severe inspiratory respiratory distress, stridor, cyanosis, and obtundation. The main diagnostic differential was severe BOAS. Due to the severe respiratory distress, the dog was anesthetized and intubated. Tracheobronchoscopy revealed abnormalities consistent with BOAS with severe secondary inflammation. After stabilization, an extubation attempt was unsuccessful and a temporary tracheostomy tube was placed and the dog was successfully woken up from anesthesia. A nasogastric tube was placed to assist with patient management. To avoid the risk of infection or obstruction, temporary tracheostomy tubes require strict hygiene and frequent nursing care. Local skin cleaning was performed every 1 to 4 hours. To humidify and aid the removal of secretions, nebulization and tube suctioning were regularly performed. In addition, the internal cannula of the tracheostomy tube was frequently changed depending on the amount of secretions. Regular nasal care was necessary due to the presence of purulent discharge. After 7 days the dog's respiratory and overall condition improved, allowing surgical correction of his BOAS. After an improvement of tracheobronchoscopic lesions, the tracheostomy tube was removed and Pablo was discharged from hospital.

Conclusions:

Severe obstructive BOAS is a life-threatening condition that can cause severe respiratory distress. Such cases may require stabilization with a temporary tracheostomy before surgical correction. To minimize complications, protocolized intensive nursing care must be provided.

E-mail: johanna.salama@vetagro-sup.fr

PUPPY WITH COMPLEX TRAUMA TO MULTIPLE BODY PARTS AND SYSTEMS CAUSED BY DOG ATTACK

S. Aadel Tångås[1]

[1] Blå Stjärnans Djusjukhus (Blue Star Animal Hospital), Gothenburg, Sweden

Introduction:

Handling a challenging case due to the complexity of injuries involved, having to make difficult choices regarding the priority of treatment, stabilization efforts, monitoring, and nursing care. The focus is on the initial 36 hours of stabilization and treatment.

Synopsis:

Puppy, 6 months, badly bitten by 4 larger dogs. Presented to the emergency barely responsive in lateral recumbency, panting heavily with bleeding wounds covering large parts of body, extremities and skull. Further assessment showed severe neck pain and bleeding from nose, oral cavity and right conjunctival sac. Emphysema was present over the frontal bone, moving synchronously with breathing, crepitations around right zygomatic bone and over sinuses. Tachycardic with weak non-synchronized femoral pulse. Pale, sticky mucous membranes, prolonged CRT. Possible penetration into thoracic cavity on the right side, possible fracture to the right front leg. PVC immediately placed, opioids and oxygen is administered. Patient in evident shock and was rushed to ICU, lateral x-ray of chest/neck were done on route. In the ICU work begun trying to stabilize the dog with warm bolus of crystalloids, oxygen, additional analgesia and antibiotics. Patient underwent initial wound care (cleaning, debridement and dressings) and further assessment of its injuries. Patient was closely monitored continuously including blood work, non-invasive blood pressure, ECG, POX and urine catheter. The following day patient underwent further surgical repair, x-rays and CT-scan.

Conclusions:

This case presented numerous challenges. The patient was strongly affected by pain, making it difficult to estimate the extent of its injuries. The combination of skull trauma, panting, and pain made it challenging to trust the vital parameters of the dog. We had to analyze these parameters separately and together to understand the patient and its needs. Determining what was due to pain/injury/stress, identifying which parameters indicated what, and prioritizing nursing care were essential considerations. For example, decisions had to be made about placing the patient's head high to lower intracranial pressure or low to reduce the risk of aspiration? Was the patient panting due to pain, injury or stress?

E-mail: sofia.johansson1990@hotmail.se

Poster Abstracts

Original Study

PREVALENCE OF FELV PROVIRUS IN A FELINE BLOOD DONOR POPULATION: A COMPREHENSIVE ANALYSIS

H. Ferreira[2], B.M. Correia[1], A.P. Magalhães[1], L.M. Rocha[1], R.F. Ferreira[2 3], I. Cardoso[2], I. Mesa-Sánchez[4]

[1] NIV-Veterinary Research Centre, Porto, Portugal
[2] Animal Blood Bank, Porto, Portugal
[3] Multidisciplinary Unit for Biomedical Research, Porto, Portugal
[4] Animal Blood Bank, Barcelona, Spain

Introduction:

Feline leukemia virus (FeLV) is a gamma-retrovirus affecting domestic cats. Depending on immune response, exposure to FeLV may result in clearance of the infection (*i.e.*, abortive infection); persistent provirus-positive aviremic infection without clinical signs or persistent viral shedding (*i.e.,* regressive infection); or extensive viral replication and shedding and development of disease (*i.e.*, progressive infection). Cats that receive a blood transfusion from feline blood donors with regressive infection can develope active FeLV infections, some with a progressive outcome and the development of fatal FeLV-associated disease (*i.e.*, non-regenerative anemia, T-cell lymphoma, or lymphoblastic leukemia). The present study aimed to investigate the prevalence of regressive infection in a feline potential blood donor population in Portugal and Spain.

Methods:

Blood samples of apparently healthy, indoor, domestic cats selected to be potential blood donors were tested for FeLV. Rapid tests (WITNESS FeLV-FIV, Zoetis or SNAP FIV/FeLV Combo Test, IDEXX) were used for a pre-donation quick screening. Later, potential donors who tested negative for the rapid test underwent a FeLV ELISA test (Gemini Stratec, Novatec). Donors who were negative for both previous tests were tested for FeLV provirus PCR (LightCycler 480II, Roche). Asymptomatic cats with negative FeLV p27 antigen tests (rapid tests and ELISA), but positive PCR for proviral DNA, were classified as cats with regressive infection.

Results:

Overall, 10,716 blood samples from 4,897 cats were tested between November 2021 and December 2023. A total of 2.7% (130/4,897) cats were positive for FeLV p27 antigen tests and therefore excluded from the screening program. To the remaining 4,767 cats, FeLV provirus PCR evidenced 2.5% (119/4,767) positive results.

Conclusions:

FeLV regressive infection prevalence is high in potential feline blood donors. Feline leukemia virus proviral DNA testing is considered essential to reduce the risk of FeLV transmission to blood recipients, regardless the use of p27 antigen tests.

E-mail: barbararibeiroc21@gmail.com

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

NOREPINEPHRINE INFUSION DID NOT IMPACT SUBLINGUAL MICROCIRCULATION IN A PORCINE HEMORRHAGIC SHOCK MODEL

M.W. Kim[1 2], T. Schuller[2 3], T. Victoni[2 4], R. Lac[2 5], B. Allaouchiche[2 3], G. Hoareau[6], C. Pouzot-Nevoret[1 2], M. Magnin[2 5]

[1] SIAMU, VetAgro Sup, Marcy L'Etoile, France
[2] Université de Lyon, UR APCSe, Marcy L'Etoile, France
[3] Université de Lyon, Hospices Civils de Lyon, Centre Hospitalier Lyon Sud, Lyon, France
[4] Department of Toxicology, Vetagro Sup Campus Vétérinaire, Marcy L'Etoile, France
[5] Université de Lyon, Vetagro Sup, Unité de Physiologie, Marcy L'Etoile, France
[6] University of Utah School of Medicine, Salt Lake City, United States

Introduction:

Hemorrhagic shock demands blood product-centered resuscitation. Vasopressor use is controversial: it can support macrocirculation and reduce transfusion requirements, but induced vasoconstriction may diminish tissue perfusion. We aimed to assess the effects of a norepinephrine CRI on sublingual microcirculation in an experimental porcine hemorrhagic shock model.

Methods:

This study has institutional ethical approval (APAFIS#21171-2018052215097237 v6). Eight pigs were anesthetized, instrumented, then two-phase massive hemorrhage was induced by withdrawing 20 ml/kg of blood over 20 minutes then 15 ml/kg over the next 40 minutes. Hypotension was maintained post-hemorrhage for 1.5 hours. Subsequently, all pigs received a fluid and autologous whole blood transfusion whilst "norepinephrine" group pigs were also administered a norepinephrine CRI (0.2 µg/kg/min). MAP was maintained above 65 mmHg in both groups by fluid or autologous whole blood boluses based on predetermined hemoglobin thresholds. Sublingual microcirculation was assessed using a sidestream dark field imaging device and proprietary software. Calculations included the De Backer score (DBs) and proportion of perfused vessels (PPV). Pigs were humanely euthanized upon completion of the study.

Results:

Induction of hemorrhagic shock produced marked disturbances with an increase in heart rate (baseline median 79 bpm [IQR 72 – 99], shock 176 bpm [140 – 182], p = 0.001), decreases in MAP (baseline 84 mmHg [81 – 94], shock 49 mmHg [45 – 51], p = 0.001), and cardiac index (baseline 1.0 L/min/m² [0.8 – 1.2], shock 0.7 [0.4 – 0.8], p = 0.009). PPV (-0.07%/min [CI -0.14; -0.01], p = 0.0255) and DBs (-0.11n/mm [CI -0.2; -0.03], p = 0.03) also decreased during this period. Total infused fluid (including whole blood) was

significantly lower in the norepinephrine compared to the control group (4125 mL [3825 – 4585] vs. 2078 mL [1912 – 2304], p = 0.03). There were no significant differences in sublingual microcirculation measures between groups.

Conclusions:

In our experimental hemorrhagic shock model, norepinephrine infusion did not significantly alter sublingual microcirculation.

E-mail: mark.kim@vetagro-sup.fr

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

PUPILLOMETRY AND PARASYMPATHETIC TONE ACTIVITY INDEX TO DETECT SYMPATHETIC NERVOUS SYSTEM ACTIVATION DURING HEMORRHAGE

M.M. Magnin[1], A.C. Chapelle[1], S.J. Junot[1], T.C. Schuller[1], P.P. Pétillon[1], M.B. Brindejonc[1], R.L. Lac[1], B.A. Allaouchiche[1], G.H. Hoareau[2], T.V. Victoni[1], M.K. Kim[1], C.P.N. Pouzot-Nevoret[1]

[1] VetAgro Sup, Marcy L'Etoile, France
[2] University of Utah, Salt Lake City, United States

Introduction:

Hemorrhage activates the sympathetic nervous system (SNS), which helps maintain tissue perfusion but may mask the presence of bleeding. Assessing SNS activation could aid detection of occult hemorrhage. Our objective was to evaluate the utility of pupillometry and *Parasympathetic tone activity index* (PTA) in assessing SNS activation during hemorrhagic shock.

Methods:

Sixteen pigs were utilized. Following anesthesia and instrumentation, massive hemorrhage was induced by withdrawal of 35 ml/kg of blood: 20 ml/kg over 20 minutes, then 15 ml/kg over the next 40 minutes. Hypotension was maintained post-hemorrhage for 1.5 hours. Pupil diameter and pupillary reflexes were measured every ten minutes using a Neurolight device (IDMED, Marseille, France). PTA (MDoloris device), heart rate (HR), and mean arterial pressure (MAP) were also recorded. Measurement changes over time and associations with the blood withdrawal volume were analyzed using mixed linear models and Pearson correlations. The results are presented as effect size (regression or correlation coefficient) and 95% confidence interval of the effect size. P-value < 0.05 were considered significant.

Results:

During blood withdrawal, a significant decrease in MAP (-0.6 mmHg/min [-0.7; -0.6], P < 0.0001), a significant increase in HR (1 bpm/min [0.9; 1.2], P < 0.0001) and pupillary diameter (0.02 mm/min [0.01; 0.02], P < 0.0001) were observed. Several parameters significantly correlated with the volume of blood withdrawn: decrease in MAP (r = -0.67, [-0.77; -0.55], P < 0.0001), increase in HR (r = 0.66, [0.53; 0.75], P < 0.0001) and increase in pupillary diameter (r = 0.32, [0.14; 0.48], P = 0.0009). During the hypotension phase, a significant but slight change in MAP (-0.05 mmHg/min, [-0.08; -0.02], P = 0.001) and pupillary diameter (0.004 mm/min, [0.002; 0.005], P < 0.0001) were observed, but HR did not vary significantly (0.02 bpm/min, [-0.04; 0.09], P = 0.50). Large variations in PTA were observed, preventing analysis. Pupillary reflexes were absent throughout.

Conclusions:

The increase in HR and pupil diameter could reflect activation of the SNS. PTA values are not useful based on the data presented. Further studies are needed to evaluate the diagnostic performance of pupillometry in clinical settings.

E-mail: mathieu.magnin@vetagro-sup.fr

EFFECT OF FELINE MULTIPLE DONATIONS ON IRON CONCENTRATION AND ITS RESERVES

I. Cardoso[2], A. Queirós[1], A. Magalhães[1], S. Alves[1], L. Rocha[1], R. Ferreira[1 2 3], I. Mesa-Sanchez [4]

[1] NIV-Veterinary Research Centre, Porto, Portugal
[2] Animal Blood Bank, Porto, Portugal, Porto, Portugal
[3] Centro de Estudos de Ciência Animal (CECA), Universidade do Porto, Porto, Portugal
[4] Animal Blood Bank, Barcelona, Spain

Introduction:

Feline blood banking is becoming more important and crucial for ECC. Awareness regarding donor welfare should be a pillar for any organized donor program by ensuring a safe donation procedure. Cumulative effects of blood donations on iron markers are still not well reported. The objective of this study was to retrospectively evaluate the levels of iron markers in feline blood donors.

Methods:

A total of 103 cats enrolled in a blood bank program were considered, with 510 donations over a period of 1-4 years and a minimum of 2 donations per year. Cats presented different breeds, sex, and age between 1-10 years. Blood samples were analyzed to determine serum iron concentration (SI), serum ferritin (SF) and unsaturated iron-binding capacity (UIBC). Transferrin saturation percentage (%SAT) was calculated using the formula (SI/TIBC) x 100, with total iron-binding concentration capacity (TIBC) being determined by adding SI and UIBC. The parameters were measured before the first donation (T0) and immediately after each donation. The analysis of variance (ANOVA) test was used to compare a) based on the length of enrollment in the donation program - Group 1 (≤2 years) and Group 2 (2 to 4 years); b) based on the total number of donations - Group 3 (3 and 4 donations), Group 4 (5 and 6 donations), and Group 5 (>7 donations). T0 values were also compared between each group.

Results:

The following group characteristics (mean/SD) were identified: respectively, Group 1 and 2 donated during 1.4±0.47 and 2.7± 0.5 years a total of 4±1.3 and 6±1.3 times; Group 3, 4 and 5 donated respectively every 6.2±4.4, 4.2±2.2 and 4.4±2.3 months. Values at T0 were SI 86.54±25.54 µg/dL; SF 5.47±3.79 ng/mL; UIBC 268.49±58.08 µg/dL; %SAT 24.80±7.89 %. No significant differences were found between groups at baseline and post donations.

Conclusions:

Based on these results, iron markers do not seem to change in cats undergoing multiple donations, suggesting preservation of iron reserves and no cumulative iron depletion. Prospective studies with standard predefined donation frequencies, and groups with higher donation frequency and longer study period would help to confirm these findings, to ensure donor welfare commitment in blood donor programs.

E-mail: anaqueiros@bsanimal.com

FELINE BLOOD DONATION: DESCRIPTION AND ADVERSE REACTIONS FROM 29201 DONATION EVENTS BETWEEN 2019 AND 2023

S.S. Taylor[1], H. Ferreira[2], A. Cambra[2], I. Mesa-Sanchez[3], K. Jeevaratnam[1], G. Lo Lacono[1], R. Ferreira[4]

[1] University of Surrey, Guildford, United Kingdom
[2] Animal Blood Bank, Porto, Portugal
[3] Animal Blood Bank, Barcelona, Spain
[4] Universidade do Porto, Porto, Portugal

Introduction:

Feline blood transfusion is required for the treatment of various illnesses in cats. Donor adverse reactions can include cardiorespiratory, venipuncture-related and behavioral abnormalities. Study aims included describing a large number of donation events, documenting adverse reactions and identifying risk factors for reactions.

Methods:

Retrospective analysis of donation event records from the Portuguese Blood Bank with recording of donor signalment, donation volume, sedation status, donor gender, breed, weight, hemoglobin and adverse reactions (acute within two hours of donation and caregiver reported via telephone questionnaire five days after donation). Risk factors for adverse reactions (volume of blood collected, weight, sedation status, blood-type, age, gender, hemoglobin level, breed) were examined by stratifying data according to groups exposed to relevant predictors and calculating odds ratios with 95% and 99% confidence intervals.

Results:

The study included 7812 individual cats and 29201 donation events between 2019 and 2023. Median age at donation was 50 months (range 12-131), body weight 4.3kg (3-12.9) and most frequent breed was 'Common European' (66.9% of donors). Median volume of blood collected was 9.3ml/kg (range 3.3-13.7). Most cats (27925/29201; 95.6%) were sedated. Adverse effects were uncommon (84/29201, 0.3%, 2.88/1000 donor events) and most commonly cardiorespiratory (22/84 cats, 0.75/1000 donor events) or behavioral (18/84, 0.62/1000 donor events). Of the 22 cardiorespiratory reactions most (18/22) received treatment: fluid therapy post-donation (13/18), butorphanol and oxygen (2/18), referral to the primary veterinarian (2/18), oxygen and feeding (1/18). All but two cats referred to the primary veterinarian (later diagnosed with underlying pathology) were discharged without further treatment. The only risk factor significantly associated with adverse reactions was conscious donation, with conscious donors 4.4 times

more likely to have an adverse reaction. Data analysis showed some indication that lower body weight might be associated with adverse reactions, but the evidence is limited.

Conclusions:

Feline blood donation is associated with a low rate of adverse reactions. A prospective randomized trial should be performed to confirm if sedation can be effective in decreasing adverse reactions. Caregiver education on home care post-donation may reduce behavioral adverse reactions.

E-mail: sam.taylor@icatcare.org

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

EVALUATION OF HEMOSTASIS INCLUDING VISCOELASTIC TESTING IN A CANINE HEMORRHAGIC SHOCK MODEL

T. Baird[1], J. Guillaumin[1], K. Zersen[1]

[1] Colorado State University, Fort Collins, United States

Introduction:

Hemostasis disorders associated with hemorrhagic shock with or without trauma is a poorly understood complex disorder.

Methods:

A canine model of pressure-targeted hemorrhagic shock followed by re-transfusion of shed blood was performed. Dogs were anesthetized and instrumented with pulmonary arterial, venous, and peripheral arterial catheters. After 10 minutes of blood pressure stabilization (T1), dogs were hemorrhaged to a mean arterial blood pressure of 40±5 mmHg for 10 minutes (T2), then resuscitated with 100% of shed blood (T3). Complete blood count, prothrombin time (PT), partial thromboplastin time (PTT), fibrinogen, antithrombin, D-Dimers level, and VCM-Vet™ parameters clot time (CT), clot formation time (CFT), alpha angle (Angle), maximum clot formation (MCF), amplitude at 10 and 20 minutes (A10 and A20, respectively), and clot lysis index at 30 and 45 minutes (LI30 and LI45, respectively) were performed at T1, T2 and T3.

Results:

Eight dogs were enrolled. Mean blood volume removed was 36.3±11.6 mL/kg. All dogs survived. CT (min) was 3.75 [2.10–7.30], 4.2 [2.20–6.00] and 2.05 [1.30–6.40] while CFT (min) was 4.45 [2.40–10.3], 3.05 [2.00–9.20] and 3.20 [1.30–9.40] at T1, T2, and T3, respectively ($p=1.000$ for both). Median angle (degree) was 46.5 [24.0–61.0], 53.0 [28.0–62.0], and 54.5 [35.0–71.0] at T1, T2, T3, respectively ($p<0.001$). Median A10 and A20 (unit) were 16.5 [10.0–26.0], 20.5 [11.0–29.0], 19.0 [10.0–32.0] ($p<0.001$) and 24.0 [15.0–35.0], 26.5 [16.0–36.0] and 25.5 [14.0–37.0] ($p<0.001$), at T1, T2, T3 respectively. MCF was 30.0 [20.0–44.0], 33.0 [23.0–44.0], 35.0 [19.0–66.0] at T1, T2, T3, respectively ($p=1.000$). Median Li30 (%) and Li45 (%) were 100 [66–100], 100 [98–100], 100 [39–100] and 100 [73–100], 100 [88–100], 100 [45–100] at T1, T2, T3 respectively ($p <0.001$ for both). Median values for angle, A10, A20, MCF, Li30 and Li45 were within normal limits for all time points. PTT was significantly prolonged ($p <0.001$). Hematocrit and platelet count were decreased over time ($p<0.05$). No other significant change was seen.

Conclusions:

Our hemorrhagic shock model showed significant prolongation of PTT, angle, A10, A20, and shortening of Li30 and Li45 over time, despite remaining in reference range, highlighting complex hemostasis disturbances in that model.

E-mail: julien.guillaumin@colostate.edu

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

CLINICAL AND POINT-OF-CARE ULTRASOUND FINDINGS IN A CASE SERIES OF CATS WITH HIGH-RISE SYNDROME

H. Swanstein[1], K. Gommeren[2], R. Langhorn[1], S. Boysen[3]

[1] University of Copenhagen, Copenhagen, Denmark
[2] University of Liege, Liege, Belgium
[3] Faculty of Veterinary Medicine, University of Calgary, Calgary, Canada

Introduction:

To describe clinical and point-of-care ultrasound (POCUS) findings in cats with high-rise syndrome.

Methods:

Client-owned cats presenting for high-rise syndrome within a five-month period were retrospectively enrolled. All data passed normality testing (Shapiro-Wilk test) and are reported as mean (+/-SD).

Results:

Fourteen cats were included; all fell from one to nine stories. Clinical findings included mean heart rate 203 (+/-30) beats/minute, respiratory rate 53 (+/-23) breaths/min, temperature 38.1 (+/-0.6) ^{O}C, fractures (6/14; femoral, spinal, tibial, or pelvic), skin lacerations (6/14), animal trauma triage scores of 0-7 (n=14), and respiratory distress (2/14). POCUS diagnosed pneumothorax in 5/14 cats; three unilateral and two bilateral. Sonographic findings included loss of lung sliding (5/5), lung point (5/5) and asynchronous curtain signs (2/5). Bilateral pneumothorax and asynchronous curtain signs were identified in the two cats with respiratory distress. Coalescent B-lines were sonographically identified in six cats and ventral lung consolidation in one cat. Abdominal POCUS was unremarkable. The subxiphoid window caudal vena cava collapsibility index was 55 (+/-23) %. Radiographs were obtained in 9/14 cats: five with sonographic findings of pneumothorax, three with coalescent B-lines, and one with lung consolidation. Pneumothorax was radiographically confirmed in three cats, one of which had had thoracocentesis performed. A diffuse bilateral interstitial pattern was radiographically identified in one cat with coalescent B-lines and unremarkable in two others. A diffuse bilateral moderate bronchointerstitial pattern was identified in the cat with sonographic lung consolidation. Twelve cats survived to discharge, and two were euthanized due to prognosis and/or financial constraints.

Conclusions:

This case series reports abnormalities identified on POCUS in cats with high-rise syndrome, supported in most cases by radiographs obtained following patient stabilization. This is the first feline study to report the sonographic combination of absence of lung sliding and detection of lung point and/or asynchronous curtain signs to diagnose pneumothorax in cats. B-lines were also detected in a few cats that had normal radiographic findings, suggesting that, as for dogs, POCUS may be more sensitive at detecting contusions compared to radiographs. A larger study using a reference standard for contusion and pneumothorax is required to confirm these findings.

E-mail: hugo.swanstein@sund.ku.dk

VALIDATION OF A HUMAN FRET-VWF73 ASSAY FOR ASSESSMENT OF ADAMTS-13 ACTIVITY IN HEALTHY DOGS

C. Crinò[1], R. Jepson[1], T. Greensmith[1], S. Cortellini[1]

[1] The Royal Veterinary College, Hatfield, United Kingdom

Introduction:

Cutaneous and renal glomerular vasculopathy (CRGV) is a thrombotic microangiopathy (TMA) of unknown etiology, often fatal in azotemic dogs. In people, a common cause of TMA is thrombotic thrombocytopenic purpura, caused by deficiency of a disintegrin and metalloproteinase with a thrombospondin type 1 motif, member 13 (ADAMTS-13). No validated ADAMTS-13 activity assay is available for use in dogs. The aim of this study was to validate a human fluorescence resonance energy transfer (FRET-VWF73) assay for measurement of ADAMTS-13 activity in dogs.

Methods:

A prospective laboratory assay validation study was performed on citrated platelet-poor plasma (PPP) obtained from residual blood samples from 20 healthy blood donors. Two methods of ADAMTS-13 inactivation were performed to obtain low ADAMTS-13 activity samples (whole blood at room temperature for 72 hours before PPP separation [LOW1]; PPP exposed to 54°C for 1 hour [LOW2]). Intra- and inter-assay repeatability tests on high (HIGH) and low samples, effect of storage (fresh refrigerated sample, and after storage at -80°C for 3 and 9 months), and dilutional parallelism (with sodium chloride 0.9% and inactivated PPP) were performed.

Results:

Coefficient of variation (CV) for the intra-assay test (n=2 repeats) for HIGH (mean ± standard deviation [SD]: 164.3 ± 9.1 IU/dl), LOW1 (mean ± SD: 115.5 ± 2.2 IU/dl) and LOW2 (mean ± SD: 118 ± 4 IU/dl) were 5.6%, 1.9% and 3.4% respectively. For the inter-assay test (n=5 repeats), CV for HIGH (mean ± SD: 134.9 ± 15.6 IU/dl), LOW1 (mean ± SD: 94.7 ± 28.3 IU/dl) and LOW2 (mean ± SD: 118.3 ± 0.3 IU/dl) were 13.4%, 27% and 2.8%, respectively. Effect of storage test showed a CV at 3- and 9-months storage of 23.9% and 9.8%, respectively. Dilutional parallelism was not observed with either diluent used.

Conclusions:

Based on these findings, we were unable to validate the human FRET-VWF73 assay in dogs, precluding its clinical use. Results from the second batch of samples were unusually low compared to other batches. No obvious reasons for this could be identified. Future studies may investigate use of canine-specific substrates FRET-VWF73 or validation of canine ADAMTS-13 enzyme-linked immunosorbent assay.

E-mail: ccrino21@rvc.ac.uk

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

COMPARISON OF ULTRASOUND PROBE LOCATION AND SONOGRAPHIC FINDINGS USED FOR THE EVALUATION OF PNEUMOTHORAX IN CANINE PATIENTS: A CADAVER PILOT STUDY

A. Finch[1], J. Menard[1], C. Harding[1], M. Hillstead[1], S.R. Boysen[1], V. Madden[2]

[1] University of Calgary, Calgary, Canada
[2] Life Line Vet Care, Calgary, Canada

Introduction:
The objective of this canine cadaver study was to compare previously described sonographic findings and thoracic windows used to diagnose pneumothorax in dogs.

Methods:
Study population: Intubated frozen-thawed cadavers undergoing positive-pressure ventilation (PPV). Inclusion criteria: Absence of pneumothorax based on lung sliding (LS) and/or B-lines at the most gravity independent sites (MGIS) and a normal curtain sign (CS). Exclusion criteria: Absent LS and B-lines at the MGIS and/or the presence of a lung-point/abnormal CS. No pneumothorax, unilateral and bilateral pneumothorax groups were created, the latter by infusing air (3ml/kg) under ultrasound guidance. Four blinded sonographers (two experts, two novices) assessed the chest tube site (CTS) and caudo-dorsal border (CDB) for LS and B-lines, and the CS for asynchronous and double CS in sternal PPV cadavers. When pneumothorax was suspected, operators searched for a lung-point. Positive or negative pneumothorax findings at the CTS, CDB, CS, and combined CTS+lung-point, and CDB+CS+lung-point (PLUS) were recorded. Post-study right and left horizontal beam radiography was used as the pneumothorax reference standard and to quantify pneumothorax volume (scant, mild, moderate, large). Results were analyzed by Fisher's exact or Chi-square test ($P < 0.05$ = significance).

Results:
Five cadavers were eliminated and 8 enrolled [22.3kg (range 30.1-13.5 kg)]. Pneumothorax was created in 11/16 hemithoraces, classified as scant (2/11) mild (6/11), and moderate (3/11). Combined accuracy, sensitivity, and specificity of all operators was 28%, 0%, 90% for both CTS and CTS+lung-point; 66%, 50%, 100% for CDB; 50%, 27%, 100% for CS; and 69%, 55%, 100% for PLUS, respectively. There was a significant difference in identification of pneumothorax between the CTS and CDB (P=0.0003), and CTS and CS (P=0.0242) and between CTS+lung-point and PLUS for all operator comparisons (P=0.0001).

Conclusions:
Preliminary evaluation of these data suggest there is a difference between the CDB and CTS to detect

pneumothorax in canine cadavers, with the CDB being more accurate (66% vs. 28%, respectively). There is a difference in PLUS compared to CTS+lung-point, with PLUS being more accurate (69% vs 28%, respectively). Validation of these findings in live spontaneously breathing dogs, and patients with larger volume pneumothorax requires further investigation.

E-mail: ashley.finch37@gmail.com

BLOOD COMPONENT CONSUMPTION PROFILE OF VETERINARY CENTERS ORDERING FROM A COMMERCIAL BLOOD BANK

R.F. Ferreira[1], R. Soares[1], I. Cardoso[1], J. Araújo[2], I. Mesa-Sanchez [3]

[1] Animal Blood Bank, Porto, Portugal
[2] Animal Blood Bank, Liége, Belgium
[3] Animal Blood Bank, Barcelona, Spain

Introduction:

The use of blood transfusions differs between veterinary clinics, and access to blood products is expected to influence transfusion decisions. No previous studies addressed the consumption profile of clinics with permanent availability of hemocomponents ordered from blood banks. The aims of this study are: 1. To quantify the blood products consumed by clinics that partnered with a blood bank; 2. To confirm the expected increased use of hemocomponents, after ensuring a quick, reliable, and permanent access; 3. To describe clinics consumption profile by species and blood component.

Methods:

Records from orders placed by clinics in Europe and Asia to a commercial blood bank were used. For the goal 1. data from 2023 orders from those clinics ordering regularly for at least 5 years was used, and clinics were grouped as small (<15 vets working, according to the staff information on their website), medium (15-29 vets) and large (≥30 vets). veterinary centers with more than 30 units ordered per year (after the first year) during more than 4 years were included for goal 2., allowing the calculation of order growth rates per year. Finally, for goal 3. involved a descriptive statistical analysis of every unit ordered in 2023.

Results:

In 2023, small (n=40), medium (24) and large (31) clinics, ordering for more than 5 years, ordered respectively 114±72, 167±80 and 252±196 units (mean±SD). In the 2nd, 3rd, 4th and 5th years ordering from the blood bank, the growing rates of 120 clinics evaluated were respectively 123±110%, 26±61%, 29±49% and 35±97% (mean±SD). From the total of 32,509 units ordered in 2023, 69.3% were canine (41.5% pRBC, 26.6% plasma, 0.8% platelets concentrate and 0.3% other components) and 30.7% feline (24.8% pRBC, 5.9% plasma).

Conclusions:

This study allowed to estimate the annual hemocomponents needs by clinic size. It also highlighted the predictable growth of consumption after starting to use blood products coming from a dedicated blood bank, meaning easy access to these products potentiates their use. The data obtained allow every clinician to understand if their clinic's reality matches the data obtained from consolidated practices on transfusion potential.

E-mail: ruiferreira@bsanimal.com

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

CLINICAL FINDINGS AND BLOOD GAS ANALYSIS IN COMPANION RABBITS WITH GASTRO-INTESTINAL STASIS: A RETROSPECTIVE STUDY OF 43 CASES

A.A. Antoine[1], D.A. Alarcon[1], C. Debie[1], C.K. Kennedy[1], K.G. Gommeren[1]

[1] Veterinary medicine faculty, Liege, Belgium

Introduction:

Little information is available regarding venous blood gas analysis in companion rabbits. Previous publications showed modified blood glucose or sodium concentrations in rabbits with gastrointestinal stasis. Common underlying conditions for gastrointestinal stasis include digestive (e.g., cecal bloat, gastric or duodenal obstruction, and sub-obstruction) and extra-digestive causes (e.g., renal disease, dental malocclusion, other). This retrospective study aimed to 1) compare blood gas results from clinically ill rabbits with internal references from 12 healthy rabbits; 2) evaluate associations between patient data, blood gas results and prognosis; 3) and evaluate whether these findings differed between rabbits with stasis suspected secondary to digestive (D) or extra-digestive (ED) causes.

Methods:

Rabbits presented for gastrointestinal stasis (decreased appetite and/or output of feces) during a one-year period were included. Patient data were recorded; blood gas analysis was performed after admission. Rabbits were grouped in D/ED based on complementary exam results and response to treatment. Outcome (survival) was recorded. Kruskal-Wallis testing was performed to compare data from sick rabbits versus internal references. Logistic regression assessed association between patient data, blood gas results and prognosis. Stepwise models evaluated associations between patient data, blood gas results, prognosis and grouped D versus ED causes. Spearman correlations were used to assess association between glucose, sodium and urea concentrations.

Result:

Forty-three rabbits were included: 23 D and 20 ED. Overall, rabbits with gastrointestinal stasis had increased glucose ($p<0.0001$), urea ($p=0.012$), hematocrit ($p=0.035$), anion gap ($p=0.041$), and lower chloride ($p=0.037$) than the internal reference. Group D rabbits were more likely to survive (16/23) versus ED (7/20) ($p=0.027$). After controlling for group D causes, higher body condition scores (odds ratio=0.14) and higher lactate concentrations (odds ratio=1.73) were associated with survival and non-survival, respectively. There were no associations between blood glucose, sodium and urea concentrations, and none of these was associated with outcome.

Conclusions:

Digestive causes of gastrointestinal stasis and higher body condition scores on admission are associated with increased survival. Increased lactate is associated with decreased survival. Unlike previous studies, we found no associations between glucose, sodium and urea concentrations.

E-mail: kris.gommeren@uliege.be

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

DIFFERENCE OF CORRECTED SERUM CHLORIDE AND MEASURED SERUM CHLORIDE IN THE DIFFERENTIATION BETWEEN CARDIOGENIC AND NON-CARDIOGENIC EFFUSIONS IN DOGS

M. Croce[1], T. Vezzosi[2], F. Marchesotti[1], E. Zini[1], O. Domenech[1]

[1] Anicura Istituto Veterinario Novara, Granozzo con Monticello, Italy
[2] University of Pisa, San Piero a Grado, Italy

Introduction:

Cavitary effusions in dogs represent a diagnostic challenge, with potential causes ranging from inflammatory diseases to neoplasia, trauma, coagulopathies, hepatic disease, protein-losing nephropathies, enteropathies, and right-sided congestive heart failure (R-CHF). In R-CHF, activation of the renin-angiotensin-aldosterone system and anti-diuretic hormone release result in venous congestion, fluid retention, and sodium (Na) and chloride (Cl) hemodilution. Correcting serum Cl based on mid-reference Na ([Na+]) and measured serum Na (m[Na+]) allows for assessing its concentration, removing the dilutional effect of water retention.

Methods:

This retrospective observational study included 76 dogs: 27 with cardiogenic effusion secondary to R-CHF(CARDIO-EFF), 28 with non-cardiogenic effusion (NC-EFF), and 21 healthy controls. Right-sided congestive heart failure was defined based on specific clinical signs, including the presence of ascites and/or pleural effusion associated with jugular venous distension, hepatomegaly, and dilated caudal vena cava. Laboratory variables, including m[Na+], m[Cl−], and c[Cl−], were collected, with c[Cl−] calculated using the formula [(mid-reference [Na+]/m[Na+])×m[Cl−]], and Δ[Cl−] determined. Dogs with endocrinopathies, gastrointestinal and kidney diseases, or those receiving diuretics were excluded. Dogs with NC-EFF exhibited various underlying conditions such as hepatopathies, neoplastic diseases, hypoalbuminemia, and chylothorax. Dogs with CARDIO-EFF showcased conditions like pulmonary hypertension, cardiac tamponade, dilated cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, and pulmonary stenosis. Healthy controls comprised pets undergoing blood sampling before blood donation or elective surgeries.

Results:

The study revealed that m[Na+] was significantly lower in dogs with CARDIO-EFF compared to controls and NC-EFF ($p<0.0001$). Δ[Cl−] was significantly higher in dogs with CARDIO-EFF compared to controls and NC-EFF ($p<0.0001$). The optimal Δ[Cl−] cutoff to detect CARDIO-EFF was >1.2 (area under the curve=0.82; $p<0.0001$; sensitivity=77%, specificity=87%).

Conclusions:

In conclusion, quantifying Δ[Cl−] demonstrated good diagnostic accuracy in differentiating CARDIO-EFF and NC-EFF in dogs, providing clinicians with a valuable parameter for formulating an effective initial diagnostic plan for cavitary effusions.

E-mail: marta.croce@anicura.it

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

MANAGEMENT OF GLASS INGESTION IN 21 DOGS

H. L. Matson[1], A. Le Gal[2], K. Humm[1], S. Cortellini[1]

[1] Royal Veterinary College, Hatfield, United Kingdom
[2] Dick White Referrals, Newmarket, United Kingdom

Introduction:

Foreign body ingestion is a common reason for presentation of dogs to the emergency veterinarian, however there are currently no reports of glass ingestion in dogs. The aim of this study was to describe the management and clinical outcome of dogs with confirmed glass ingestion.

Methods:

This was a retrospective study of dogs presenting to 2 referral hospitals with confirmed glass ingestion between January 2007 and January 2023. Signalment, presentation, diagnostic imaging findings, case management and outcome were recorded. Referring practices were contacted for follow-up information. Descriptive statistics only were used, and data presented as median (range).

Results:

Twenty-one presentations and 20 dogs were included; one dog presented twice 3 years apart for eating different glass objects. Cocker spaniels (n=4), Labrador retrievers (n=3) and crossbreeds (n=2) were the most common breeds. There were 12 neutered males, 4 entire males, 2 neutered females and 2 entire females. Median age was 3 years 2 months (2 months–14 years 4 months), median weight was 20kg (4.3-44kg). Most commonly consumed items were jars containing foodstuffs (n=7), followed by glass casserole dish (n=2), with fragment sizes ranging from 0.5-6.8cm. Two dogs presented with alimentary tract perforation (one esophageal and one jejunal). Ten cases were medically managed only, 6 underwent surgery (of which 4 had glass removed successfully), 4 endoscopy (of which 1 had glass removed successfully), and one both endoscopy and surgery which had glass removed successfully. Two dogs were initially managed medically prior to performing gastrotomy due to lack of radiographic progression. Ultimately, 6/21 had successful removal of the glass objects. The largest fragment that was successfully medically managed was 2.4cm; the smallest fragment that was successfully surgically removed was 2cm. Of the remaining 15 dogs, follow-up of one-month post-discharge was available for 13 dogs; diarrhea was the only complication reported (n=2). All dogs survived to discharge.

Conclusions:

Medical management of canine glass ingestion is possible in selected cases and was successful in the majority of dogs (88%), however care should be taken as gastrointestinal perforation can also occur.

E-mail: hmatson1@rvc.ac.uk

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

CARDIOVASCULAR EFFECTS OF THREE DIFFERENT INTRAMUSCULAR DOSES OF MEDETOMIDINE-VATINOXAN IN HEALTHY DOGS INDUCED WITH PROPOFOL AND ANESTHETIZED WITH SEVOFLURANE

H.A. Turunen[1], S. St. Clair[2], N. Zimmerman[3], W. Muir[2]

[1] Vetcare Oy, Helsinki, Finland
[2] QTest Labs, Columbus, Ohio, United States
[3] Dechra, Northwich, United Kingdom

Introduction:

Medetomidine-vatinoxan combination (Zenalpha®) is indicated to provide restraint, sedation and analgesia during non-invasive, non-painful or mildly painful procedures and examinations intended to last no more than 30 minutes. Safe use of medetomidine-vatinoxan as a pre-anesthetic has not been evaluated, and therefore, it's use as preanesthetic is currently contraindicated in Europe. The objective was to characterize the cardiovascular effects of three different intramuscular doses of medetomidine-vatinoxan in dogs anesthetized with sevoflurane.

Methods:

This was an experimental, cross-over laboratory study with 6 healthy pre-instrumented Beagle dogs. Each dog received intramuscular injection of medetomidine 0.25 mg/m^2 and vatinoxan 5 mg/m^2 (LOW), medetomidine 0.5 mg/m^2 and vatinoxan 10 mg/m^2 (MIDDLE), or medetomidine 0.75 mg/m^2 and vatinoxan 15 mg/m^2 (HIGH) as premedication (T0), followed by intravenous propofol approximately 20 minutes later (T20) administered to effect to allow intubation. Anesthesia was maintained with sevoflurane for 55 minutes (T25-T80). Heart rate (HR), cardiac index (CI), stroke volume index (SVI), systemic vascular resistance index (SVRI) and direct mean arterial pressure (MAP) were recorded prior to premedication and then every 5 to 10 minutes. Each of the variables were analyzed via a mixed linear model repeated measures analysis with $p < 0.05$ considered significant.

Results:

During anesthesia (T25-T80) HR increased, and SVI, SVRI and MAP decreased significantly with all studied medetomidine-vatinoxan doses in comparison to last timepoint prior to anesthesia (T15). With MIDDLE dose CI was significantly lower from T60 to T80 in comparison to T15. The changes in CI during anesthesia were not statistically significant with other studied doses. Lowest group mean arterial pressures during anesthesia were 66 ± 5 mmHg (mean ± SD) at T60 with LOW, 71 ± 6 mmHg at T65 with MIDDLE, and 66 ± 4 mmHg at T60 with HIGH. Two dogs with LOW dose had transient period of hypotension at the end of

anesthesia that resolved without treatment. Lowest MAP recorded was 57 mmHg at T80. All recoveries were uneventful.

Conclusions:

Medetomidine-vatinoxan dosing did not affect the incidence or magnitude of cardiovascular changes during sevoflurane-anesthesia in healthy dogs. Cardiovascular effects during anesthesia were virtually identical with all studied doses.

E-mail: heta.turunen@vetcare.fi

VETERINARY ONCOLOGIC PATIENTS IN ICU: PREVALENCE AND PROGNOSTIC FACTORS

C. Mattavelli[1], A. Guillén[1], K. Humm[1], S. Cortellini[1]

[1] The Royal Veterinary College, Hatfield, United Kingdom

Introduction:

Veterinary oncologic patients might require hospitalization in the Intensive Care Unit (ICU). However, outcomes of these patients are unknown. The aims of this study were to describe the oncologic population in a veterinary ICU and identify possible prognostic factors.

Methods:

Prospective, observational study at a veterinary teaching hospital between December 2022 – January 2024. All patients admitted to the ICU were recorded. Those with a definitive diagnosis or a strong suspicion (based on diagnostic imaging results and disease progression) of neoplastic disease had clinicopathologic data collected as well as the reason for admission to the ICU. Survival to discharge and 90 days post-discharge were recorded, as well as date of death. Standard inferential statistics and multivariate linear regression analysis were performed.

Results:

In the study period, the prevalence of neoplastic patients in the ICU was 13.6%, with 38 cats and 114 dogs being diagnosed with, or strongly suspected of having, neoplasia. A definitive diagnosis was obtained in 106 patients (73.7% cats, 68.4% dogs). Hematologic neoplasia was the most frequent cancer in cats (64.3%), while carcinoma was the most frequent in dogs (35.9%), followed by sarcoma (25.6%). The most frequent reasons for admission in the ICU were respiratory distress (31.6% cats, 20.2% dogs) and cardiovascular instability (21.1% cats, 20.2% dogs). Survival to discharge was 56.6% (44.7% cats, 60.5% dogs) and 90-day survival was 27.2% (21.1% cats, 29.4% dogs). Routine admission, primary disease different from neoplastic, surgical treatment, receiving chemotherapy, higher ionized calcium concentration and lower quick SOFA score were predictors of survival in dogs; while in cats, routine admission, surgical treatment and receiving chemotherapy were predictors of survival.

Conclusions:

Neoplastic disease was common in our ICU population. Prognosis for critical oncologic patients was guarded. Dogs and cats presented similar prognostic factors.

E-mail: cmattavelli22@rvc.ac.uk

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

A SYSTEMATIC LITERATURE SEARCH AND STRUCTURED REVIEW ON THE MEASUREMENT OF ANTIMICROBIAL RESISTANCE AND SUSCEPTIBILITY TESTING IN URINE SAMPLES OF DOGS AND CATS

S. Mitze[1], M. Costa[1], R. Humphry[1]

[1] Scotland's Rural College (SRUC), Centre for Epidemiology and Planetary Health, Inverness, United Kingdom

Introduction:

The main objectives of this work were to evaluate which antimicrobial susceptibility testing (AST) methodology is currently used, its interpretation, reporting of resistance and the estimation of the prevalence of antimicrobial resistance (AMR) in urine samples of dogs and cats.

Materials and Methods:

A systematic literature search and structured review was performed by following the Preferred Reporting Items for *Systematic reviews* and Meta-Analyses (PRISMA) guidelines, and by adapting them where necessary. PubMed® was used for identification of articles published between the years 2010 and 2022 inclusive. Studies were included if the article was in English, urine of cats and dogs were used, applied AST methods were mentioned and their interpretation. The way of reporting of resistance was noted, as was the number of examined and resistant bacteria.

Results:

In total, 125 studies were identified, of which 57 were included. Urine sampling methods were infrequently reported. The reported quantity of urine tested varied widely (1µl - 1ml). The number of isolates tested per sample could not be clarified. Among phenotypic AST methods used, disk diffusion was most common (50.9%, n = 29), followed by broth microdilution (26.3%, n = 15) and agar dilution (1.7%, n = 1). Phenotypic and genotypic AST methods were commonly used together (21.1%, n = 12). The number of antibiotics and antibiotic classes tested was variable and ranged from 6-33 (median 15.8) and 4-14 (median 8.0), respectively. Minimum inhibitory concentrations (MIC) were infrequently listed. Recommendations of the Clinical and Laboratory Standards Institute (CLSI) were the most used guidelines globally while European Committee on Antimicrobial Susceptibility Testing (EUCAST) guidelines and veterinary breakpoints were rarely used. Examined antibiotic resistance and virulence factor genes were ranging from 1-35 (median 13) and 3-16 (median 9), respectively.

Conclusions:

In summary, this study detected a significant variability in the preanalytical phase, AST methodology used, its interpretation, genotypic analysis, and reporting of resistance. Conclusions about the prevalence of AMR in urine of dogs and cats could not be drawn.

E-mail: stefanie1mitze@hotmail.com

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

EXAMINING THE IMPACT OF BLOOD DONATION FREQUENCY ON THE SEDATION DOSAGE ADMINISTERED TO FELINE DONORS

A.F.P. Cambra[1], H.C.M. Ferreira[1], S.S. Taylor[2], I. Mesa-Sanchez[3], R.F. Ferreira[1 4]

[1] Animal Blood Bank, Porto, Portugal
[2] University of Surrey, Guildford, United Kingdom
[3] Animal Blood Bank, Barcelona, Spain
[4] Centro de Estudos de Ciência Animal (CECA), Universidade do Porto, Porto, Portugal

Introduction:

Creating a cat-friendly environment and using low-stress handling techniques reduce the stress associated with blood donation. However, sedation during the donation process is still frequently required to avoid stronger physical restraint and sudden movements. Higher needs for sedation are frequently associated with increased stress levels. This study aims to evaluate the relationship between the sedative dose needed to perform blood donations and its frequency in cats, as an indirect indicator of donor tolerance and stress.

Methods:

Records of all feline blood collections from a blood bank, performed from January 2019 to October 2023, were analyzed. The study sample included sedated donations and, when applicable, subsequent non-sedated donations. Donors who had received prior anxiolytic medication were excluded. The sedation protocol involved placing an IV catheter and administering an initial fixed dose of diazepam (0.54mg), ketamine (2.71mg), and butorphanol (0.14mg) mixture, with additional half dose(s) bolus if needed based on donors' response to handling and level of anxiety (assessed by donors' behavior and body language). Donors were grouped by the number of donations performed and median sedation dose was compared. Descriptive statistical analysis was conducted and differences between groups were evaluated.

Results:

The sample included 7,750 feline donors, corresponding to 27,874 (98.9%) sedated donations and 319 (1.1%) non-sedated donations. Among all donors, only 120 (1.6%) that were sedated for the first donation did not require sedation for subsequent donations. The median number of donations was 2 (1-19) and the median weight was 4.2 kg (3.0-11.0 kg). The median sedation dose was 0.03 ml/kg (0.00-0.17 ml/kg), corresponding to 0.11 mg/kg of diazepam, 0.54 mg/kg of ketamine and 0.03 mg/kg of butorphanol. There were no statistically significant differences in the median sedation dose needed among various groups, except for a minor decrease observed between the first and the third donations (p=0.047).

Conclusions:

The sedation doses used for blood donors did not increase as the number of donations increased. The decrease observed between the first and third donations is unlikely to have clinical relevance as it is not reasonably measurable. We may postulate that the stress level and donor intolerance do not increase over time.

E-mail: andrecambra@bsanimal.com

RANGE OF REFERENCE FOR IONIZED MAGNESIUM AND IMPACT OF INTRAOPERATIVE FLUID THERAPY IN HEALTHY DOGS

F. Perini[1], F. Evangelista[1], A. Briganti[1]

[1] University of Pisa, Pisa, Italy

Introduction:

The aim of this study was to extrapolate a range of reference for ionized magnesium and assess the impact of one hour of intraoperative fluid therapy in healthy dogs.

Methods:

For establishing the reference range, dogs undergoing preanesthetic clinical evaluation were enrolled. Among these, patients undergoing elective surgical or diagnostic procedures lasting 1 hour were recruited. Venous blood gas analysis was conducted before anesthesia (T0). Physical examination and blood exams were performed to confirm ASA I status. Following sedation with a mix of dexmedetomidine, methadone and ketamine, fluid therapy with Ringer Lactate started at a rate of 3 mL/kg/h. A second venous blood gas analysis was conducted after 1 hour of fluid therapy (T1), and a third was performed 1 hour after extubation and discontinuation of fluids (T2). The reference range for ionized magnesium was calculated using the 2.5th and the 97.5th percentiles of the values recorded a T0. A one-way ANOVA for repeated measures with a Dunnett's test as post hoc analysis was used to compare values recorded at T0 with T1 and T2. P values < 0.05 were considered significant.

Results:

Blood gas analysis at T0 was performed on 120 dogs. The mean value for Mg^{2+} was 0.66 ± 0.14 mmol/L, and the reference range was 0.44 - 1.01 mmol/L. Sixty-five dogs underwent an anesthetic procedure. Hematocrit, hemoglobin, BUN, creatinine, pH, lactate and HCO_3^- significantly decreased at both T1 and T2. $PvCO_2$ and Ca^{2+} significantly increased at T1 and T2, while Mg^{2+}, K^+ and glucose increased at T2.

Conclusions:

The reference range for ionized magnesium in healthy dogs was found to be 0.44-1.01 mmol/L. An increase in Ca^{2+}, Mg^{2+}, and K^+ was observed, possibly due to the direct effect of fluid therapy, despite Ringer Lactate lacking magnesium, to the mild respiratory acidosis developed during anesthesia and to the active warming during recovery. The decrease in BUN, creatinine, lactate, hematocrit, and hemoglobin could be

attributed to the diluting effect of fluid therapy. Further studies are necessary to elucidate the effect of fluid therapy on pathological patients.

E-mail: francesca90p@gmail.com

ANCILLARY TREATMENTS WITH THROMBOLYTICS OR MEDICATIONS AIMED AT IMPROVING FUNCTIONAL RECOVERY OR BOTH IN ACUTE FELINE AORTIC THROMBOEMBOLISM

C.C. Ray[1], J. Wolf[2], J. Guillaumin[1]

[1] Colorado State University, Fort Collins, United States
[2] University of Florida, Gainesville, United States

Introduction:

Locomotion improvement without serious complications is the main goal of treatment for feline aortic thromboembolism (FATE). A study showed no outcome differences with systemic 1-hour tissue plasminogen-activator (tPA) infusion. tPA continuous rate infusion (CRI) and other ancillary therapies have not been studied.

Methods:

This is a retrospective, bicentric study. Inclusion criteria were cardiogenic FATE cats receiving tPA CRI and/or medications aimed at improving functional recovery (i.e., pentoxifylline and/or cyproheptadine). Survival to discharge and ambulation status were compared to historical multicentric control groups (raw data available for this study) not receiving pentoxifylline and/or cyproheptadine: 1-hr-tPA (1 mg/kg over 1-hour, n=20) or placebo (n=20) control groups. All continuous data are presented as median (minimum-maximum).

Results:

Seven cats (57% male) were enrolled, 83% with bilateral FATE. Median age was 6 years (5.3-10). Median weight was 5.4kg (4.1-6.2). Admission rectal temperature, pulse rate, respiratory rate, non-affected lactate, affected limb lactate, creatinine and potassium were 98.5°F (95.8-101.8), 220 beats-per-minute (180-280), 46 breaths-per-minute (30-72), 3.1 mmol/L (1.1-10.9), 12.1 mmol/L (7.05-15.50), 1.3 mg/dl (0.6-2.1) and 3.7 mmol/L (3.5-4), respectively. Significant differences were found between our population and age of the placebo group (11 years (3-15), p=0.02) and rectal temperature of the 1-hr-tPA group (96.4°F (90-102), p=0.04). Five cats (71%) received tPA-CRI (0.1-0.2 mg/kg bolus then 0.1-0.2 mg/kg/hr for 1 mg/kg total). One additional cat received pentoxifylline (60-100mg PO, q12hr) and cyproheptadine (2mg PO, q12hr). The remaining cat received only pentoxifylline. Out of the five cats receiving tPA-CRI, four received pentoxifylline and three received cyproheptadine in addition to tPA-CRI and pentoxifylline. Reperfusion injury and acute kidney injury were documented in 50% and 29% of the cats, respectively, not different from the 1-hr-tPA group (58% and 42%, p=1.00 and 0.35, respectively), and the placebo group (46% and 58%, p=1.00 and 0.66, respectively). All study cats (100%) had an improvement in locomotion,

compared to 60% (1-hr-tPA, p=0.08) and 40% (placebo, p=0.07) in control groups. Four cats survived to discharge (57%), compared to 45% (1-hr-tPA, p=0.68) and 30% (placebo, p=0.36) in control groups.

Conclusions:

In this pilot study, all cats receiving the described medication regained locomotion without worsening of complications.

E-mail: julien.guillaumin@colostate.edu

Poster Abstracts

Case Reports

ACUTE LINGUAL ARTERY THROMBOSIS IN A CAT WITH HYPERTROPHIC CARDIOMYOPATHY

F. Blunschi[1], J. Ortlieb[1], T. Jaresova[1], C. Vollmar[1], M. Schneider[1], S. Schaub[1], E. Hassdenteufel[1]

[1] Small Animal Clinic, Justus-Liebig University Giessen, Giessen, Germany

Background:

Thromboembolism is a common complication in cats suffering from cardiomyopathy and mostly affects the hind limbs. This case describes a case of lingual artery thrombosis in a cat with hypertrophic cardiomyopathy. This localization has been described for cardiac thromboembolism in people but so far not in cats.

Case presentation:

An 8-year-old male neutered domestic shorthair cat was presented to the emergency service for acute onset of staggering gait, apathy, and anorexia. Clinical examination revealed pronounced hypersalivation and a swollen and bluish discolored tip of the tongue with an inability to retract it into the oral cavity. In addition, an arrhythmia with a systolic heart murmur (grade III/VI) and tachypnea (88/min) was detected. Neurological examination showed ataxia and blindness with absent menacing response and pupillary reflex. A cardiac focused point-of-care ultrasound revealed severe dilatation of the left atrium and auricle (LA/Ao 2.5) with spontaneous echo contrast and severe thickening of the myocardium. This was later confirmed through a complete echocardiographic examination diagnosing hypertrophic cardiomyopathy. Initial blood pressure was 60 mmHg, with pulse palpable in all four limbs. Treatment with dobutamine (2.5 µg/kg/min) constant rate infusion (CRI) was started. After blood pressure increased (140 mmHg), dobutamine CRI was discontinued. Complete workup included MRI contrast medium study of the head, revealing lack of accumulation of contrast medium in the tongue and small hemorrhage around the choroid plexus in the fourth ventricle on the left side. Color flow Doppler assessment showed a low degree of blood flow in the A. lingualis to the base of the tongue, with no detectable blood flow in the rostral part. Anticoagulant therapy was started with enoxaparin (1 mg/kg) subcutaneously, which was later changed to a CRI (3 mg/kg/day). Due to the progression of tongue swelling with severe swallowing difficulties, the cautious prognosis of tongue preservation and functionality, and the advanced heart disease, the owners elected euthanasia.

New/Unique Information:

This case documents a novel localization for feline thromboembolism due to an underlying cardiac disease. Cardiac evaluation may be warranted in cats with acute lingual swelling and discoloration without apparent cause.

E-mail: fabienne.blunschi@vetmed.uni-giessen.de

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

REFRACTORY HYPERKALEMIA IN A CAT WITH PRESUMPTIVE TYPE 4 RENAL TUBULAR ACIDOSIS AND PSEUDOHYPOALDOSTERONISM DUE TO SUSPECTED LIPID-INDUCED RENAL PAPILLARY NECROSIS

C. Torrente[1], P. Bou[1], M. Riba[1], D. Fernández[1], L. Bosch[1]

[1] Fundació Hospital Clínic Veterinari UAB, Universitat Autònoma de Barcelona, Cerdanyola del Vallès (Barcelona), Spain

Background:

To describe type 4 renal tubular acidosis (RTA) and acquired pseudohypoaldosteronism (APHS) in a cat with acute kidney injury presumably associated with lipid infusion therapy for permethrin toxicosis.

Case presentation:

A 1-year-old castrated male cat initially presented with acute weakness, ataxia, fasciculations, tremors, hyperthermia, and seizures due to permethrin toxicosis. Upon admission, intravenous diazepam and 20% intravenous lipid emulsion were administered. Forty-eight hours post-discharge, the cat was readmitted, displaying lethargy, anorexia, polyuria, and vomiting. At readmission, the cat exhibited depression, dehydration, and moderate hypoperfusion. Initial assessments included a complete blood count, biochemistry profile, and urinalysis. Further diagnostic work-up and abdominal point-of-care ultrasound revealed clinical findings consistent with acute kidney injury (AKI). Venous acid-base analysis confirmed severe hyperkalemia and hyperchloremic normal anion-gap metabolic acidosis. Urinalysis revealed a pH of 7, urine specific gravity of 1.010, urine osmolality of 350 mOsm/kg, and slight proteinuria. The urinary fractional excretion (FE) of sodium (1.25%; reference interval [RI]: 0.03–0.81%) was elevated while the urinary FE of potassium (0.69%; RI: 4.70–14.26%) and the urinary transtubular potassium gradient (TTKG) were extremely low (0.6; RI: 2.9-4.6), all consistent with a diagnosis of secondary or APHS and type 4 RTA (voltage-dependent hyperkalemic distal RTA). Emergency medical treatment for hyperkalemia was initiated, but proved unsuccessful. Peritoneal dialysis was initiated 24 hours post-admission, alongside general supportive care. Mineralocorticoid support (continuous rate infusion of hydrocortisone) was initiated four days post-admission due to suspected aldosterone deficiency/resistance at the distal nephron. Unfortunately, hyperkalemia remained refractory despite the intensive peritoneal dialysis prescription. Finally, the cat died sixteen days post-admission with the complete histopathological examination confirming acute and severe renal papillary necrosis but absence of abnormalities on the remaining renal parenchyma (renal cortex) and also the adrenal glands.

New or unique information:

This is the first report of APHS characterized as a type 4 RTA in a cat. Furthermore, the authors hypothesize that, according to the histopathological findings, this particular presentation of AKI might be presumably secondary to the intravenous ILE use for permethrin toxicosis, not previously reported also in the veterinary literature.

E-mail: carlos.torrente@uab.es

ACUTE RESPIRATORY DISTRESS SYNDROME IN A DOG: MANAGING LUNG COLLAPSE WITH AIRWAY PRESSURE RELEASE VENTILATION

G. Frudua[1], M. Pelizzola[1], C. Fattori[1]

[1] Clinica Veterinaria San Marco, Veggiano (PD), Italy

Background:

Airway pressure release ventilation (APRV) is a form of mechanical ventilation (MV) that minimizes alveolar derecruitment in spontaneously breathing patients. APRV setting parameters include a long period (T_{high}) of high airway pressure (P_{high}) and a short period (T_{low}) of low airway pressure (P_{low}). Although APRV is a well-known strategy for the management of refractory hypoxemia in patients with acute respiratory distress syndrome (ARDS) in human medicine, there is only one reported case in veterinary literature.

Case presentation:

An 11-year-old male mixed-breed dog was presented for a 10-day history of coughing, dyslexia, and vomiting. Fever, tachycardia and a moderate expiratory dyspnea were noted on physical examination. Thoracic radiographs showed a multifocal bilateral broncho-alveolar pulmonary pattern. Blood works revealed a systemic inflammatory state. Suspecting an ARDS associated with pneumonia, antimicrobial therapy and continuous positive airway pressure ventilation with a helmet were started. Serial arterial blood gas analyses were performed, revealing a state of hypoxemia (PaO_2:66 mmHg; SaO_2:83 %; FiO_2:40%). The patient's respiratory distress progressively worsened, requiring MV. Despite pressure-assisted controlled (PAC) MV being started, the patient could not maintain SpO_2 > 85% and required continuous lung recruitment maneuvers. Based on these findings, MV setting was changed to APRV (P_{high}:13 cmH_2O; P_{low}:0 cmH_2O; T_{high}:4 seconds; T_{low}:0.6 seconds). In the following hours, oxygenation parameters improved (PaO_2:83.9 mmHg; SaO_2:92.6%; FiO_2:85%). Computed tomography (CT) confirmed radiographic findings. Two multidrug-resistant bacteria were isolated from a broncho-alveolar lavage culture, confirming pneumonia. APRV was continued for three days, with stable oxygenation parameters. A control CT scan showed a mild improvement of the pulmonary conditions. On day 5, due to the slow clinical progresses and owners' financial limitations, the dog was euthanized.

New/Unique information:

The authors suspect that, during PAC MV, the exhalation phase caused a cyclic alveolar derecruitment, determining a state of hypoxemia. APRV could have improved oxygenation parameters due to the short duration of T_{low}, which generated an intrinsic positive end-expiratory pressure that prevented alveolar

collapse. To the authors' knowledge, this is the second case reported in veterinary literature of ARDS successfully managed using APRV.

E-mail: plz.mrc@gmail.com

SEVERE ANEMIA CAUSED BY ORAL LIMNATIS NILOTICA LEECH INFESTATION IN A DOG

I. Green[1], S. Hefer[1], G. Dafna[1], S. Klainbart[1], K.Y. Mumcuoglu[2], I. Aroch[1]

[1] Koret school of veterinary medicine, The Hebrew University of Jerusalem, Rehovot, Israel
[2] The Hebrew University-Hadassah Medical School, Jerusalem, Israel

Background:

Leeches are parasitic segmented worms (phylum *Annelida*, subclass *Hirudinea*) with worldwide distribution. Leech infestation is commonly termed hirudiniasis and occurs following host contact with leech-infested water. Clinical signs vary according to the affected anatomic site. Reports of internal hirudiniasis in domestic animals are scarce. This report describes a case of severe, life-threatening anemia in a dog, caused by oral hirudiniasis.

Case presentation:

A 3.5-year-old, female Shi-Tzu dog was presented due to acute collapse and oral bleeding. Previous medical history was unremarkable. Clinical signs first appeared a week earlier with acute onset of dry cough manifested with daily episodes followed by oral bleeding. At presentation, the dog was collapsed, showing tachycardia, tachypnea and pale mucous membranes. A dark sublingual hematoma was described. Melena was noticed on rectal examination. CBC revealed severe regenerative anemia [Packed cell volume/total plasma proteins (PCV/TPP), 13/4] and leukocytosis, while platelet count, prothrombin time and activated partial thromboplastin time were within normal reference intervals. Initial treatment included packed red blood cells transfusion at 20mL/kg followed by supportive care and monitoring. The dog's clinical condition improved significantly following the transfusion. Repeated bloodwork revealed moderate anemia (PCV/TPP 22/4.6) with increased D-dimer concentration and markedly low antithrombin activity. The dog underwent general anesthesia for further oral cavity examination and upper gastrointestinal endoscopy. Three engorged leeches were observed attached to the sublingual area. The leeches were manually removed using forceps and salt. Upper gastrointestinal endoscopy showed blood remnants with no further abnormalities. The leeches were identified as *Limnatis nilotica,* widely distributed throughout the Middle East. Recovery was uneventful and the dog was discharged 24 hours post-presentation. Upon recheck, three days post-discharge, the dog was bright with normal vital signs and no evidence of cough or bleeding. PCV/TPP were stable compared to discharge (23/5.8).

Unique information:

This is the first report of naturally occurring internal hirudiniasis in domestic dogs in Israel. Decreased antithrombin activity, a novel finding with hirudiniasis, raises questions regarding the underlying mechanism. It is unclear whether this signifies a possibly clinically significant effect of active leech saliva proteins *in-vivo* or merely *in-vitro* effects on assay accuracy.

E-mail: dririsgreen@gmail.com

STATUS EPILEPTICUS AFTER ELECTROCUTION INJURY IN A DOG

J. Ortlieb[1], F. Blunschi[1], S. Muthmann[1], A. Olszewska[1], E. Hassdenteufel[1]

[1] Justus-Liebig-University Giessen, Giessen, Germany

Background:

Electric fences are usually harmless for humans and animals, supplying only a brief high-voltage impulse with a low current to scare, protect, and confine livestock. Rare reports exist of fatal encounters in people and wildlife after accidentally becoming trapped in electric fences. This case report describes the successful management of a dog suffering from status epilepticus after being entangled in an agricultural electric fence.

Case presentation:

A 1.5-year-old male Kangal-Mix was presented to the emergency service after being entangled in an agricultural electrical fence for 1-2 hours. The fence was connected to a direct current energy source supplying a 10,000-volt impulse every 1.5 seconds. On presentation, the dog was recumbent, unable to stand or walk, and showed severe hypersalivation, panting, and tachycardia (128 beats/min). The remaining physical parameters and neurological examination were normal, and no electrical burn injuries were observed on the body. ECG and thoracic radiographs showed no abnormalities. During further assessment and stabilization of the patient, sudden tonic-clonic seizures occurred. Initial benzodiazepine administration (midazolam 0.5 mg/kg IV) had no effect, and anticonvulsive therapy had to be consecutively extended (midazolam CRI 0.5 mg/kg/h, phenobarbital 16 mg/kg IV, levetiracetam 60 mg/kg IV, ketamine CRI 0.3 mg/kg/h, dexmedetomidine CRI 0.5 µg/kg/h) due to refractory seizure activity. Additionally, with progressive anticonvulsive therapy, the dog showed insufficient ventilation and mechanical ventilation had to be initiated. Due to the rapid deterioration with refractory seizure activity, an electroencephalogram (EEG) was performed while the dog was on mechanical ventilation to help guide therapy and provide prognostic information. EEG recording revealed only sporadic singular spikes, yet no more seizure activity, and the dog could be successfully weaned after a total of 30 hours on the ventilator. Anticonvulsive therapy was tapered throughout the remaining hospital stay, and the dog made a full recovery and was discharged with oral phenobarbital after a total of 7 days. Four weeks after phenobarbital was discontinued, the dog experienced a new seizure and medication was reinitiated.

New/Unique information:

This is the first case describing successful management in a dog with status epilepticus due to electrocution injury from an electric fence.

E-mail: julia.ortlieb@vetmed.uni-giessen.de

DEXMEDETOMIDINE INFUSION IN THE MANAGEMENT OF A REFRACTORY SEPTIC SHOCK IN A DOG AFFECTED BY PROTEIN LOSING ENTEROPATHY (PLE)

G. Ceccherini[1], A. D'Addetta[1], F. Romoli[1], V. Marchetti[2]

[1] BLUVET SpA, Lucca, Italy
[2] University of Pisa, Pisa, italy

Background:

The alpha-2 agonist dexmedetomidine provides benefits to treat septic shock through activating receptors in medullary vasomotor center.

Case presentation:

A 2-year-old male German Shepherd dog with a diagnosis of Protein-Losing Enteropathy (PLE) presented as emergency after collapsing at home with multiple episodes of hemorrhagic diarrhea. Dog presented in lateral recumbency and unresponsive, with signs of distributive shock, shallow breathing and sinus tachycardia with 180 beats per minute. The systolic non-invasive blood pressure (PAS) was 80 mmHg, the mean arterial pressure (MAP) was 45 mmHg, and the rectal temperature registered at 41.5°C. Stabilization efforts were initiated, including administering oxygen through a face mask. The dog received three intravenous boluses of Ringer's Lactate (30 ml/kg), followed by a single bolus of hypertonic 7.5% saline solution (5 ml/kg) and then assessment with Ringer lactate solution at 5-10 ml/Kg/h A venous blood gas analysis was performed, along with a complete blood count (CBC), comprehensive chemical panel, and thoracic and abdominal point-of-care ultrasound (POCUS). AFAST revealed presence of moderate abdominal fluid and peritoneal reactivity.

The blood work revealed:

- Moderate metabolic acidosis: Lactate (2.3 mmol/L; reference range 0-2.5 mmol/L) and bicarbonate (HCO3- 16 mEq/L).
- Neutrophil left shift: 2.01 K/µL (2.95-11.64 K/µL).
- Thrombocytopenia: 140 K/µL(150-484 K/µL).
- Hypoalbuminemia: 1.8 g/dL (2.3-4.0 g/dL).
- Hypocholesterolemia: 81 mg/dL (115-320 mg/dL)
- Hyperbilirubinemia: 2.3 mg/dL (0-0.9 mg/dL).
- Hypoglycemia: 50 mg/dL (88-120 mg/dL).

Glucose at 5% was supplemented and monitored. Amoxicillin Clavulanate at 22 mg/kg iv q8h was initiated. Despite fluids boluses, MAP did not improve and Norepinephrine at 0.1 µg/kg/min was started, but MAP remained low (<60 mmHg) and Hydrocortisone at 0.6 mg/Kg IV was initiated. Because of persistence of hypotension, dexmedetomidine infusion at 2µg/Kg/h was also added with an improvement and normalization of blood pressure parameters, MAP was assessed around 80-85 mmHg and after six hours from the start and Dexmedetomidine was discontinuated followed by Noradrenaline. Clinical condition of dog improved, blood pressure remained stable. After 4 days in intensive care unit, dog was discharged uneventfully.

New/Unique Information:

To the authors' knowledge, dexmedetomidine is used for the first time in the management of refractory septic shock in a dog with PLE.

E-mail: gianilaceccherini@virgilio.it

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

SUCCESSFUL OPEN-CHEST CARDIOPULMONARY RESUSCITATION IN A DOG WITH PYOTHORAX

E. Escande[1], N. Graziano[1], M. Lejoncq[1], S. Larcheveque[1], B. Mignini[1], K. Gommeren[1], C.R. Kennedy[1 2]

[1] Clinique Vétérinaire Universitaire, Liège, Belgium
[2] Veterinary teaching hospital, Chicago, United States

Background:

Open-chest cardiopulmonary resuscitation (OCCPR) is described in human and veterinary literature as an effective way to provide greater cardiac output with improved cerebral and myocardial perfusion compared with closed-chest cardiopulmonary resuscitation. This report describes a dog with a para-esophageal abscess and secondary pyothorax complicated by a pneumothorax requiring OCCPR and experiencing complete long-term recovery.

Case presentation:

A 5-year-old neutered male German hound was referred for management of a pyothorax. The pleural fluid was removed *via* needle thoracocentesis, and the dog was anesthetized and intubated for computed tomography (CT). A para-esophageal abscess and consolidated lung tissue were identified and ultrasound-guided aspiration was performed. Upon return from CT to the intensive care unit, the dog experienced a cardiopulmonary arrest (CPA), suspected secondary to a tension pneumothorax. Peracute sinus bradycardia (30 bpm) occurred, followed within 60 seconds by asystole and loss of a palpable pulse. OCCPR was initiated immediately through a right-sided thoracotomy by a first veterinarian; a second performed direct cardiac compressions as soon as possible; and a third provided manual ventilation at 10 breaths per minute. The pericardium was not removed. Return of spontaneous circulation was achieved within 6 minutes from asystole. The dog was transferred to surgery for exploratory sternotomy. The para-esophageal abscess was drained and removed, the thoracic cavity was lavaged, and both the sternotomy and emergency thoracotomy wounds were closed routinely. A chest tube was placed. Postoperatively, the dog was severely hypoxemic (PaO2: 32.8 mmHg), anemic (x%), hypovolemic (based on clinical and ultrasonographic findings) and hypotensive (MAP: 45 mmHg), requiring management with high flow nasal oxygen, blood product administration and crystalloid infusions. Critical care echocardiography was performed to optimize fluid management. The dog recovered over the following six days. On discharge, he was ambulatory and eating voluntarily. Six months after OCCPR, the dog continuous to do well without remaining detectable abnormalities reported by the owner or on clinical examination.

New/Unique information:

OCCPR is recommended in dogs experiencing CPA with suspected pleural space disease. Although a left-sided approach is recommended in textbooks, a right-sided approach is acceptable to avoid delays due to patient repositioning.

E-mail: etienne.escande@uliege.be

ACUTE RESPIRATORY DISTRESS SYNDROME AND TRICUSPID VALVE THROMBUS SECONDARY TO PULMONIC BALLOON VALVULOPLASTY TREATED WITH OXYGEN CAGE THERAPY AND HEPARIN

G.T. Toth[1], J. J. C. Noval Montero[2], M. Legrand[2], M. Lekane[2], K. Gommeren[2], A. C. Merveille[2]

[1] Willows Veterinary Centre & Referral Service, Shirley, Solihull, United Kingdom
[2] University of Liège, Liège, Belgium

Background:

Pulmonic stenosis can be addressed through balloon valvuloplasty (BVP) in dogs. Serious complications post BVP such as arrhythmias, thromboembolism, cardiac perforation and non-cardiogenic pulmonary edema (NCPE) have been described. The etiology of NCPE remains debated, with potential causes including a sudden increase in hydrostatic pressure within lung capillaries, increased capillary permeability, or inflammation-mediated reperfusion-ischemia injury. Acute respiratory distress syndrome (ARDS), probably secondary to NCPE, has been described in 5 dogs. Two cases survived, treated with a continuous positive airway pressure helmet (CPAP) and positive pressure ventilation (PPV), providing adequate oxygenation and alveolar recruitment. Unfortunately, CPAP isn't widely available and PPV may be cost-prohibitive. This case describes successful treatment of a tricuspid valve thrombus and ARDS post-BVP with furosemide, pimobendan, heparin and non-invasive oxygen support.

Case presentation:

A 7-month-old female whippet underwent BVP for Type A pulmonic stenosis with high pulmonary valve pressure gradient (PVPG 155 mmHg), and secondary right ventricular hypertrophy. Moderate tricuspid valve dysplasia and regurgitation was demonstrated. The procedure was uneventful, but the patient developed acute expiratory dyspnea 20 minutes post BVP. Point-of-care ultrasound and thoracic radiographs were consistent with non-cardiogenic pulmonary edema or pulmonary thromboembolism. PPV and an CT-angioscan were declined for financial reasons. High flow nasal oxygen (HFNO) support providing some positive pressure was not supported. The dog was placed in an oxygen cage at 70% FiO2 and received furosemide and pimobendan. Respiratory rate and effort improved, and FiO2 was discontinued over the following 12 hours. On the second day, a control echocardiography demonstrated a thrombus adherent to the parietal tricuspid valve leaflet, after which a constant rate infusion of heparin was administered. The dog was discharged on day three receiving clopidogrel, rivaroxaban and atenolol. Three months afterward, the patient was clinically fine, with an improved PVPG (65 mmHg) and resolved thrombus.

New/Unique Information:

This case reports successful treatment of ARDS following BVP with non-invasive oxygen using an oxygen cage. The oxygen cage offered a viable, less stressful environment compared to HFNO. Noninvasive oxygen support may have prevented hemodynamic complications associated with PPV. Valvular thrombus formation post-BVP has not been described previously.

E-mail: giulia.toth89@gmail.com

PRESUMPTIVE SEPSIS-ASSOCIATED ENCEPHALOPATHY IN A CAT AND A DOG

C. R. Sharp[1], C. Chan[1], C. J. Boyd[1]

[1] Murdoch University, Murdoch, Australia

Background:

To describe the clinical presentation, diagnosis, management, and outcome of presumptive sepsis-associated encephalopathy (SAE) in a septic cat and dog.

Case presentation:

A 2-year-old male neutered domestic shorthair cat with bacterial pyothorax and a 5-year-old male neutered cavoodle dog with septic necrosis of the falciform ligament were referred to a tertiary hospital for treatment of sepsis. Both patients met criteria for multiple organ dysfunction syndrome and developed new-onset neurological dysfunction subsequent to the development of sepsis. The cat developed generalized seizures, impaired consciousness, and proprioceptive and cranial nerve deficits. The dog developed impaired mentation, delirium behavior, proprioceptive deficits, and paroxysmal clonic movement of his left pelvic limb. The cat additionally had hepatobiliary and pulmonary dysfunction whilst the dog had cardiovascular, pulmonary, and renal dysfunction. Both patients had magnetic resonance imaging (MRI) of the brain and cerebrospinal fluid (CSF) analysis. The cat had a normal brain MRI and a normal CSF total cell count (0.6cells/µL, reference interval 0-8cells/µL) with a relative increase in neutrophils (35%) and mast cells (31%). The dog had T2W/FLAIR hyperintensities, focally in the left caudate nucleus, and bilaterally in the dorsal caudate nuclei, corona radiata, and internal capsule on brain MRI, with cytologically normal CSF. Exclusion of other etiologies and a consistent clinical progression prompted a presumptive diagnosis of SAE. Treatment of both patients followed general recommendations for sepsis, including prompt broad-spectrum intravenous antimicrobial therapy, source control, intravenous fluid therapy and vasoactive agents to normalize perfusion, analgesia, and support for other organ dysfunctions. Specific therapy for presumed SAE included anticonvulsive medications (midazolam, levetiracetam, phenobarbital) for the cat and sedative medications for the delirium behavior in the dog. Both patients survived to discharge and had neuropsychological sequelae consistent with human SAE. The cat had persistence of generalized seizures up to two years post-discharge and the dog had persistent behavioral changes.

New/Unique information:

This is the first report in veterinary medicine to describe a syndrome of neurological dysfunction in a cat and a dog, with brain MRI and CSF analysis, consistent with SAE in humans.

E-mail: c.sharp@murdoch.edu.au

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

UPPER AIRWAY OBSTRUCTION FOLLOWING RODENTICIDE INTOXICATION IN THREE DOGS

M. Pelizzola[1], G. Frudua[1], M.S. Calabria[1], E. Clementi[1]

[1] Clinica Veterinaria San Marco, Veggiano (PD), Italy

Background:

Anticoagulant rodenticide intoxication (ARI) is a common poisoning in dogs. ARI results in life-threatening coagulopathy by inhibiting vitamin K_1 epoxide reductase. Clinical signs are non-specific and depend on the hemorrhage site (usually pleural space, pulmonary parenchyma and abdomen). Upper airway obstruction (UAO) is not frequently associated with ARI.

Case presentation:

In January-February 2023, three dogs, with known ingestion or possible exposure to anticoagulant rodenticides, were referred because of UAO. Dog 1: An eight-year-old, spayed female, mixed-breed dog was presented for inspiratory dyspnea, oral bleeding, difficulty in barking and neck region swelling. Dog 2: A three-year-old, intact female, Bernese Mountain dog was presented for inspiratory dyspnea and coughing. Dog 3: A thirteen-year-old, neutered male, mixed-breed dog was presented with labored breathing. On admission, intubation and mechanical ventilation (MV) were performed due to the severity of clinical signs (Dogs 1-2) and respiratory arrest (Dog 3). The endoscopic examination showed arytenoid hemorrhage, peri-laryngeal and sublingual hematomas (Dog 1); tracheal sub-obstructive hemorrhage and intraluminal clots (Dog 2); laryngeal swelling due to intramural hemorrhage (Dog 3). Based on the history, endoscopic and clinical findings and significantly prolonged coagulation times, ARI was hypothesized. Therefore, vitamin K_1 was administered intravenously (5 mg/kg) and continued at the dosage of 2.5 mg/kg twice daily. In all dogs, rodenticide Brodifacoum was detected in blood samples by liquid chromatography-mass spectrometry. Clotting times normalized within 3 hours after vitamin K_1 administration. After 48 hours, an endoscopic evaluation revealed an improvement of the UAO, thus all dogs were weaned from MV and successfully extubated.

New/Unique information

To the authors' knowledge, there are few reported cases of UAO associated with ARI in dogs. In case of inspiratory dyspnea and upper airway hemorrhage, ARI should always be included in differential diagnosis. Intravenous vitamin K_1, intubation and MV can be considered life-saving treatments in cases of UAO due to ARI.

E-mail: plz.mrc@gmail.com

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

GENERALIZED TETANUS IN A CAT AND ITS MANAGEMENT WITH A PERCUTANEOUS ENDOSCOPIC GASTROSTOMY TUBE

G.T. Toth[1], A. Valcke[2], C. R. Kennedy[3], K. Gommeren[2]

[1] Willows Veterinary Centre & Referral Service, Shirley, Solihull, United Kingdom
[2] University of Liège, Liège, Belgium
[3] University of Illinois Urbana-Champaign, Urbana, United States

Background:

Tetanus is a rare neurological disease in cats. Intensive supportive care is required to minimize complications, such as aspiration pneumonia and malnutrition. Early enteral feeding is important to prevent intestinal villous atrophy and malnutrition. Nutritional management in reported feline tetanus cases has been limited to spontaneous feeding or assisted feeding via a nasogastric or esophagostomy tube. This case describes a class III feline tetanus patient with megaesophagus and hiatal hernia, complicated by secondary aspiration pneumonia, that was successfully treated and whose nutritional requirements were met using a percutaneous endoscopic gastrostomy (PEG) tube.

Case presentation:

An 8-month-old female entire Maine Coon presented with a 3-day history of spastic monoparesis of the right hind limb, progressing to generalized spasticity involving the entire body over the ensuing days. A clinical diagnosis of generalized tetanus was established, prompting the initiation of intensive supportive therapy, encompassing antibiotics, skeletal muscle relaxants, analgesia, and general supportive measures. During hospitalization, the patient developed megaesophagus, hiatal hernia, and aspiration pneumonia or pneumonitis confirmed with thoracic radiography. To meet nutritional requirements, and to circumnavigate the megaesophagus and hiatal hernia, a PEG tube was placed. The cat recovered slowly but uneventfully. She ate spontaneously on day 20 of hospitalization and was discharged the following day. A source for tetanus was not identified.

New/Unique information:

Patients with tetanus are at risk of aspiration pneumonia due to ileus, megaesophagus and hiatal hernia. Enteral feeding is recommended to avoid intestinal villous atrophy, malnutrition, and disrupted gastrointestinal mucosal barrier. Placing tubes via an esophageal route can be technically challenging in patients with esophageal dysfunction and may pose an additional risk for aspiration pneumonia or reflux esophagitis. PEG tubes bypass the esophagus, making them suitable for patients with generalized tetanus that require prolonged assisted enteral feeding. In cats, PEG tubes are, however, rarely placed due to the

small esophageal diameter requiring small endoscopes and trained personnel. This case report describes the successful placement and outcome of a PEG tube in a cat with generalized tetanus. Due to their benefits, PEG tubes should be considered for cats with generalized tetanus.

E-mail: giulia.toth89@gmail.com

FELINE URETHRAL OBSTRUCTION SECONDARY TO A FOREIGN BODY

M. Vernaccini[1], C. Di Franco[1], A. Briganti[1], C. Puccinelli[1], I. Lippi[1]

[1] University of Pisa, Pisa, Italy

Background:

Urethral obstruction is a common emergency presentation in male cats. Urolithiasis, plugs, and congenital/acquired abnormalities (such as urethral strictures) are the most prevalent causes, while foreign bodies are extremely rare. This case report describes urethral obstruction in a cat due to a broken urinary catheter.

Case presentation:

A 12-year-old, male neutered, European cat was presented to the the Veterinary Teaching Hospital for stranguria and hematuria, with a history of urethral obstruction resolved by catheterization 5 years before. On admission, the cat was depressed, with a painful and overdistended urinary bladder, heart rate 140bpm, respiratory rate 20bpm, and temperature 38.2°C. Abdominal point-of-care ultrasound revealed perivesical peritoneal effusion and bladder intraluminal hyperechoic material. Venous blood gas revealed metabolic acidosis and increased BUN (229mg/dL) and creatinine (18.7mg/dL). Sacrococcygeal epidural anesthesia was performed under sedation, and a urinary catheter was positioned. During catheterization, a plug was excreted from the urethra and sent for analysis, as well as a urine culture sample. The day after, an abdominal ultrasound was done, and an intraluminal linear foreign object was observed in the urethra beyond the presence of the urinary catheter. Additionally, a diagnosis of pyelonephritis of the left kidney and hydronephrosis of the right was made, based on ultrasonographic and urinary findings. The urinary catheter placed the day before was removed and a CT was performed, which confirmed the presence of a linear foreign body broken into two pieces in the urethra. A urethrotomy was necessary to find the fragments. Nephrectomy of the left kidney, cystotomy and urethrotomy were performed and the 5-year-old urinary catheter was removed. The cat was discharged after 13 days, uneventfully.

New/Unique Information:

Diagnostic imaging should be considered for any patient who exhibits symptoms of urinary tract obstruction; the passage of a urinary catheter does not exclude the presence of a foreign body as seen in the present case. Furthermore, after the first obstruction event, the cat never experienced episodes of stranguria or hematuria. Finally, to ensure that no catheter fragments are left inside the bladder, it would be crucial to monitor the integrity of every catheter during any disostruction.

E-mail: matilde.vernaccini@phd.unipi.it

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

POSTOPERATIVE INTERMITTENT ROPIVACAINE ADMINISTRATION VIA QUADRATUS LUMBORUM PLANE CATHETERS IN A DOG AFTER UNILATERAL LAPAROTOMIC ADRENALECTOMY

M.D. Degani[1], G.B. Bolen[1], S.L. Larchevêque [1], S.N. Noël [1], L.D. uriez[1], K.G. Gommeren[1], A.B. Briganti[2], C.S. Sandersen[1]

[1] Veterinary Teaching Hospital, University of Liege, Liege, Belgium
[2] Veterinary Teaching Hospital, University of Pisa, Pisa, Italy

Background:

The quadratus lumborum (QL) block is an ultrasound (US)-guided locoregional anesthesia technique that consists of injecting the local anesthetic in the thoracolumbar fascia surrounding the QL muscle, providing abdominal somatic and visceral analgesia[1]. Catheter placement in the QL plane for local anesthetic administration is reported in humans for postoperative analgesia after abdominal surgery[2].

Case presentation:

A 6-year-old, 32 kg, female Siberian Husky underwent laparotomic right-sided adrenalectomy for removal of a cortical cortisol-secreting adenocarcinoma. Premedication consisted of methadone 0.2 mg/kg and dexmedetomidine 1 µg/kg intravenously (IV), general anesthesia was induced with propofol IV and maintained with isoflurane in an oxygen/air mixture. A bilateral QL block was performed with ropivacaine 0.5% (0.3 ml/kg per side). Surgery lasted 300 minutes, due to adhesions between the mass and caudal vena cava, but concluded uneventfully. Consequently, a catheter for epidural administration was placed under US guidance in each hemiabdomen, between the QL muscle and the transverse process of the first lumbar vertebra (L1)[2]. Shortly, a Tuohy needle was introduced in plane in a ventrolateral-to-mediodorsal direction and advanced towards the ventral aspect of the transverse process of L1. Located through the thoracolumbar fascia and near the transverse process, a 0.5 mL saline injection produced inter-fascial plane hydrodissection. A catheter was advanced approximately 5 cm through the needle into the plane, and then fixed to the skin. Correct placement was confirmed by computed tomography, injecting 0.5 mL of iopromide contrast medium. Ropivacaine was instilled before recovery and every 6 hours thereafter for 48 hours. The dog was capable of walking without assistance within 2 hours after extubation. Postoperative therapy consisted of prednisolone 0,5 mg/kg IV BID and trazodone 4 mg/kg per os TID. Glasgow Composite Measure Pain Scale (GCMPS) was assessed every 4 hours as of 1-hour post-surgery (T1). GCMPS was 7/24 only at T1 when methadone 0.2 mg/kg IV was administered. After 48 hours of an uneventful postoperative period, the dog was discharged after catheter removal.

New/Unique information:

Based on this case report, ropivacaine administration through catheters in the QL plane may represent a valid strategy for postoperative analgesia after laparotomy in dogs.

E-mail: mdegani@uliege.be

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

SPONTANEOUS LUNG LOBE TORSION RESULTING IN A SADDLE THROMBUS IN A 2-YEAR-OLD SIGHTHOUND

E. Tinsman[1]

[1] Bluepearl Veterinary Partners, Portland, United States

Background:

Spontaneous and secondary lung lobe torsions have been well documented in pugs and sighthounds. Thromboembolic complications involving the pulmonary artery and azygos have also been reported; however, to the author's knowledge, this is the first documented case of spontaneous lung lobe torsion with subsequent development of a saddle thrombus in a dog.

Case presentation:

A 2-year-old female spayed Silken Windhound presented as a referral for cardiac evaluation and thoracic CT for a suspected lung lobe torsion. The patient had a one-week history of recurrent pleural effusion of waxing and waning volume treated with repeated thoracocentesis. Fluid analysis confirmed the presence of a modified transudate. Thoracic radiographs performed prior to transfer showed cranial deviation of the right cranial bronchus. Sonographic images obtained by a third party before transfer were suggestive of a thrombus in the left ventricle, and complete echocardiogram was recommended prior to anesthesia. On presentation the patient had mild respiratory effort but was otherwise hemodynamically stable. She was hospitalized overnight on intravenous fluids, supplemental oxygen and analgesic medications. Within hours of admittance the patient became acutely paraplegic with clinical signs suggestive of an arterial thromboembolism at the level of the aortic trifurcation (saddle thrombus). An echocardiogram confirmed normal cardiac structures and absence of turbulent flow. The previously documented soft tissue opacity was no longer present. The owners elected to euthanize due to the patient’s non-ambulatory state and uncertain prognosis. The patient received, with owner consent, approximately 3 mls/kg of iodinated intravenous contrast media as part of the euthanasia protocol. Post-mortem full-body radiographs and computed tomography confirmed the presence of a right cranial lung lobe torsion as well as a saddle thrombus with occlusion of both the external and internal iliac arteries. The authors posit that the saddle thrombus observed in the left ventricle originated from the pulmonary vein, moving into the heart following a partial or complete de-rotation of the affected lung lobe.

New/Unique information:

Cardiac evaluation prior to thoracotomy and discussion surrounding perioperative thrombolytics may be warranted in certain cases of lung lobe torsion. Development of a saddle thrombus worsens prognosis.

E-mail: atinsman@me.com

ERECTOR SPINAE PLANE BLOCK IN A DOG WITH ACUTE PANCREATITIS

F. Perini[1], C. Di Franco[1], A. Briganti[1]

[1] University of Pisa, Pisa, Italy

Background:

The erector spinae plane (ESP) block is an ultrasound-guided interfascial block. The local anesthetic is infiltrated between the erector spinal muscle and the vertebral transverse processes. Local anesthetic spreads over 3-6 vertebral spaces in a cranio-caudal direction which results in blockage of multiple nerve roots. It provides analgesia for spinal surgeries, thoracotomies, and laparotomies.

Case presentation:

A miniature schnauzer, sterilized female, 11 years old, 8.5 kg of weight dog was admitted to the intensive care unit. The patient presented to the emergency room for hematochezia and abdominal pain. Blood gas analysis, blood exams, and abdominal ultrasound were performed and acute pancreatitis was diagnosed. After one intramuscular bolus of methadone 0.2 mg/kg pain was assessed with the 4AVet scale and resulted in a score of 8, thus analgesia was switched to intravenous infusion of fentanyl at 3 µg/kg/h and lidocaine at 1 mg/kg/h. After 2 hours pain score decreased to 6 but the dog appeared very depressed: at this point a bilateral ESP block was performed at T9 level with 0.3 mL/kg of ropivacaine 0.5% per injection point. Pain was re-assessed 1, 4, and 7 hours after the block. One hour after the block the pain score was 2 so fentanyl infusion was decreased to 2 µg/kg/h while lidocaine was stopped. Then pain score was 1 and fentanyl infusion was stopped and methadone 0.2 mg/kg was administered if the pain score was raised over 4.

New/Unique information:

Pancreatitis is an inflammatory reaction within the pancreas which can result in abdominal pain, anorexia, and vomiting. To the author's knowledge, few information is present regarding the use of ESP block to treat abdominal pain in dogs. ESP block targets sympathetic nerve fibers and it desensitizes both ventral and dorsal rami causing both somatic and visceral analgesia. Complications are rare since the site of injection is far from the pleura, blood vessels, and the bone marrow. In this case, ESP block seemed to be effective to provide abdominal analgesia in case of pancreatitis and it allowed to reduce systemic analgesic drugs leading to a reduction in side effects.

E-mail: francesca90p@gmail.com

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

HAND GRANADE BLAST INJURIES IN A DOG DURING ACTIVE SERVICE IN A WAR ZONE

S.K. Klainbart[1], E.K. Kelmer[1], I.L. Lenchner[2], Y.F. Fishelson[2], R.B. Ben-Amotz[3], S.E. Efrati[4]

[1] Department of Small Animal Emergency and Critical Care, Koret School of Veterinary Medicine, Rehovot, Israel
[2] Oketz Unit, Israeli Defense Force, Tel Aviv, Israel
[3] Department of Small Animal Surgery, Koret School of Veterinary Medicine, The Rob, Rehovot, Israel
[4] The Sagol Center for Hyperbaric Medicine and Research at Shamir Medical Center, Be'er Ya'akov, Israel

Background:

Although canines have been used extensively in various military operations, there are no detailed reports describing the features and treatment of hand-grenade injuries in dogs.

Case presentation:

A 9-year-old spayed female Malinois military explosives dog was presented after a hand grenade exploded next to her during combat in a war zone. Her soldier applied combat field dressings on the wounds, and placed an arterial tourniquet on her front right leg due to a bleeding open fracture. She was treated with IV crystalloids in the field and was evacuated back to the border where the tourniquet was removed. She was then treated with IV crystalloids and colloids, tranexamic acid, antibiotics, and butorphanol before being admitted within 4.5 hours of the trauma. On presentation, the dog was non-ambulatory and semi-comatose with multiple shrapnel injuries to the head and trunk, and a modified Glasgow coma scale score of 11. The dog suffered from head trauma, loss of vision, and multiple open fractures of the right metacarpal bones. A head and cranial body CT-scan revealed shrapnel scattered around the head and cranial body, multiple skull fractures, and 2 metal shreds in the forebrain alongside an opaque area in the left frontal lobe suspected to be a hematoma. Blood work revealed anemia, hypoproteinemia, and hypoalbuminemia. Treatment included IV crystalloids, tranexamic-acid, antibiotics, mannitol, packed-cells, fresh frozen plasma, fentanyl, and Levetiracetam. During her long hospitalization, the dog was ventilated intermittently, suffered aspiration-pneumonia, underwent placement of a PEG-tube, bilateral enucleations, placement of a bi-valve cast and endured intensive physical therapy. After 2 weeks of hospitalization she was systemically stable; however still non-ambulatory and minimally responsive. The dog underwent 30 daily sessions of hyperbaric oxygen therapy during which her status improved; she became ambulatory and responded to smell and sensational stimuli (being both blind and deaf). The dog was discharged after 50 days to be adopted by the Soldier's family.

New/Unique Information:

This is the first report of a successful treatment of a dog that suffered severe hand grenade blast injuries with multiple treatment modalities, including hyperbaric oxygen therapy.

E-mail: klainbart@gmail.com

NONCARDIOGENIC ARTERIAL THROMBOEMBOLISM IN A CAT FOLLOWING AORTIC AIRGUN PELLET INJURY

S. Muthmann[1], J. Ortlieb[1], S. Schäfer[1], F. Blunschi[1], S. Körber[2], J. Müller[2], D. Farke[1], E. Haßdenteufel[1]

[1] Small Animal Clinic, Justus-Liebig University Giessen, Gießen, Germany
[2] Institute of Veterinary Pathology, Giessen, Germany

Background:

Arterial thromboembolism (ATE) of cardiac origin is a common complication in cats with cardiomyopathies. Occasionally, thromboembolism of non-cardiogenic origin occurs, most commonly due to pulmonary neoplasia or hyperthyroidism. Two case reports exist describing aortic thromboembolism in cats from migrating airgun pellets, creating a mechanical obstruction inside the vascular system. This report describes the first case of gunshot injury with an aortic lesion causing ATE, resulting in paraparesis in a cat.

Case presentation:

An 8-month-old neutered male domestic shorthair cat was presented with signs of ATE, showing paraparesis, disorientation, and vestibular dysfunction. Thoracic radiographs and echocardiography showed no signs of an underlying cardiac cause. However, radiographs revealed an airgun pellet above the cardiac silhouette. The owners reported that the cat had received treatment for a presumed bite wound on its left shoulder the past week. Additional diagnostic imaging was performed to determine the precise location of the pellet and the extent of the present thrombi. Computer tomography revealed that the left scapula and fifth rib were shattered, and the pellet was lodged between the main bronchi and left pulmonary artery. Two focal thromboemboli were observed: one in the descending aorta at the level of the base of the heart and the other extending from the renal arteries to the aortic bifurcation. A therapeutic attempt was made with enoxaparin constant rate infusion (3mg/kg/d). After three days, a faint pulsation was detected in the femoral arteries. The left hind limb had palpable pulse after six days. Unfortunately, sufficient perfusion could not be regained in the right hind limb, which became necrotic after eight days. The owners elected for amputation, however, the attempt proved to be futile intraoperatively, and the cat was euthanized. Postmortem examination confirmed the location of the airgun pellet between the tracheal bifurcation and the pulmonary artery. A 2 mm aortic lesion was identified in the aortic arch posterior to the left subclavian artery branch. The abdominal aortic thromboembolism measured 4 cm and extended into the right common iliac artery.

New/Unique Information:

This is the first case of an aortic lesion from a gunshot injury with secondary noncardiogenic arterial thromboembolism.

E-mail: sofie.muthmann@vetmed.uni-giessen.de

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

PRESENTATION OF HYPOALDOSTERONISM IN A DOG

M. F. H. Fumero-Hernández[1 2], P.B. Bou[1], M.R. Ribas[1], L.B. Bosch[2], C.T. Torrente[2]

[1] Fundació Hospital Clínic Veterinari UAB, Barcelona, Spain
[2] Departament de Medicina i Cirugia Animals, Facultat de Veterinaria UAB, Barcelona, Spain

Background:

Primary hyperaldosteronism is an endocrine pathology, rarely reported in the canine species. It is characterized by excessive aldosterone production at the adrenocortical level, often associated with functional tumors or hyperplasia of the adrenal glands. This condition typically manifests with hypokalemia, hypernatremia, muscle weakness, and hypertension. In this case report, we describe the characteristic analytical findings and an unusual clinical presentation of this disorder in a dog.

Case presentation:

An 11-year-old mixed-breed dog presented to the clinic with a history of polidipsia-polyuria, generalized weakness, ataxia, and marked ventroflexion of the neck. The general physical examination revealed severe dehydration (8%) but not hypertension or other relevant clinical findings. Serum biochemistry indicated severe ionized hypomagnesemia (0.3 mmol/L. Range: 1.8 y 2.5) and hypokalemia (2.79 mmol/L. Range: 4.0–5.4). Subsequent hormonal measurements revealed hyporeninemia (0.26 ng/ml/hour. Range: 0.4-1.9) and hyperaldosteronemia (400.72 pg/ml. Range: 15-102). An ACTH stimulation test rule out hypoadrenocorticism, showing a basal cortisol of 2.6 µg/dL (Range: 0.5-5.5) and post-ACTH of 6.8 µg/dL. As part of the initial therapy to stabilize the patient, we administered potassium chloride (0.1 mEq/kg/h) and magnesium sulfate (5 mg/kg/h) via constant rate infusion. In the hours following admission, there was a remission of the initial symptoms of generalized weakness and ventroflexion of the neck. Abdominal ultrasound revealed a mass at the caudal pole of the left adrenal gland. Despite a presumptive diagnosis of primary hypoaldosteronism associated with an adenoma/carcinoma, advanced diagnostic imaging techniques (CT scan) and subsequent adrenalectomy with histopathological analysis were proposed. However, financial constraints led us to opt for medical treatment with spironolactone and oral potassium supplementation after correcting the electrolyte and fluid balance. Subsequent follow-ups confirmed a favorable outcome, with correction of the initial analytical abnormalities.

New/Unique information:

This case illustrates an unusual presentation of primary hyperaldosteronism in a dog, characterized by ventroflexion of the neck and normotension. It emphasizes the crucial role of appropriate fluid therapy in

critically ill patients to correct severe electrolyte imbalances, enabling stabilization for further diagnostic efforts.

E-mail: marcos.vet@outlook.es

DIABETIC KETOALKALOSIS IN A DOG: A CASE REPORT

M.N. Saada Mussuto[1], P. Bou [1], M. Ribas[1], L. Bosch[1 2], C. Torrente[1 2]

[1] Fundació Hospital Clínic Veterinari UAB, Bellaterra, Barcelona, Spain
[2] Departament de Medicina i Cirurgia Animals, Facultat de Veterinària de la UAB, Bellaterra, Barcelona, Spain

Background:

Diabetic Ketoacidosis (DKA) in small animals is marked by hyperglycemia, glucosuria, ketonemia/ketonuria, and metabolic acidosis. Ketone buildup usually leads to acidosis, though other conditions may alter blood pH, complicating diagnosis. In human medicine, there's a theory of hypochloremic alkalosis in DKA patients. In the absence of gastrointestinal signs, mechanical-functional ileus or relevant drug therapy, such findings may stem from an intracellular ion shift, transferring chloride to red blood cells or interstitial fluids to counter bicarbonate loss. However, the exact mechanism remains unclear, warranting further research. Notably, diabetic ketoalkalosis hasn't been documented in canines, to the author's knowledge.

Case presentation:

A 9-year-old female poodle, previously diagnosed with diabetes mellitus but without insulin treatment, presented to the emergency department of a referral veterinary hospital with a 48-hour history of only anorexia, weakness, and dehydration. The diagnostic protocol included a CBC, serum biochemistry, blood ketone measurement, urinalysis, and abdominal-thoracic POCUS exam. Key findings in the diagnostic tests performed included severe hyperglycemia (47.9 mmol/L), ketonemia (7.1 mmol/L), hyperphosphatemia (2.32 mmol/L), hyponatremia (sodium corrected for glucose: 131.7 mmol/L), and hypochloremia (87.9 mmol/L). Blood gas results revealed severe alkalemia (pH 7.62), normal pCO2: 38 mmHg (45-50), severely increased HCO3-: 41 mmol/L (18-26) and SBE: 19.6 mmol/L (-4/+4). Complementary exams detected subclinical bacteriuria and hyperechogenicity of the pancreas suggestive of moderate pancreatitis. Traditional acid-base analysis detected primary metabolic alkalosis with respiratory compensation. Semiquantitative analysis revealed a more complex imbalance: all components except chloride exerted an acidifying effect, with chloride having a positive and notably alkalizing effect (+34.2 mmol/L) over the final SBE (19.6 mmol/L). Despite the lack of an identified cause, the detection of hypochloremia as a possible main factor in alkalemia allowed us to adjust the therapeutic plan, focusing on an alternative fluid therapy (0.9%NaCl). Complementary symptomatic and regular treatments for DKA were started, and the patient was finally discharged 4 days post-admission.

New/Unique information:

This work presents a unique case of diabetic ketoalkalosis in a dog. Comparing acid-base findings using traditional and semiquantitative (Fencl-Stewart) methods could not only clarify chloride's crucial role in this rare complex imbalance but also enhance the management of such endocrine emergency.

E-mail: martinasaada1@gmail.com

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

ACUTE COLLAPSE AND ARDS SECONDARY TO A MULTIPLE AUTOIMMUNE SYNDROME IN A DOG DIAGNOSED WITH ATYPICAL HYPOADRENOCORTICISM, HYPOTHYROIDISM AND POLYMYOSITIS

S. Hefer[1], E. Kelmer[1], I. Green[1], M. Mazaki Tovi[2], K. Aharoni[3], S. Klainbart[1]

[1] Small Animal Emergency and Critical Care, Koret School of Veterinary Medicine, Jerusalem, Rehovot, Israel
[2] Small Animal Internal Medicine, Koret School of Veterinary Medicine, Jerusalem, Rehovot, Israel
[3] Koret Veterinary Teaching Hospital, Jerusalem, Rehovot, Israel

Background and objectives:

Autoimmune diseases are a common pathology in dogs. Three or more autoimmune pathologies occurring simultaneously are known in human medicine as multiple autoimmune syndrome, affecting up to 25% of cases. However, in veterinary medicine, this condition is considered rare. This case report describes a multiple autoimmune syndrome in a dog, encompassing atypical hypoadrenocorticism, hypothyroidism and polymyositis syndrome.

Case presentation:

A 6.5-year-old spayed female dog was presented with acute collapse and dyspnea. A few weeks before presentation, the dog was suspected to suffer from polymyositis by the referring veterinarian. On arrival, the dog was cyanotic and dyspneic and was immediately intubated and ventilated. Physical examination revealed extreme bradycardia (<40 BPM), myotic pupils, general muscle twitching and spasm, ptyalism and diarrhea. Point-of-care ultrasound and thoracic radiographs revealed non-cardiogenic pulmonary edema. Complete blood work showed elevated muscle enzymes activity and mild azotemia. The next day, the dog was still unconscious. Abdominal ultrasound revealed thin adrenal glands. Basal cortisol and ACTH stimulation test were consistent with hypoadrenocorticism. T4 concentration was low, TSH levels were high and thyroglobulin autoantibody result was positive. Muscle biopsies revealed mild lymphocytic myositis. The dog was treated with broad-spectrum antibiotics due to bacterial pneumonia, prednisone and hydrocortisone, thyroxin, and supportive care; weaned off mechanical ventilation, and has slowly recovered. She was discharged with the same treatment and physiotherapy. The dog was admitted 6 days later with a fever, which quickly deteriorated, and was euthanized at the owner's request. Necropsy results revealed multifocal gastroesophageal ulcers with perforation of the esophageal wall. The thyroid and adrenal glands appeared small and atrophic, consistent with clinicopathological diagnosis, and the skeletal muscles showed diffused atrophy at different degrees of severity. Polymyositis was confirmed by histopathology.

New/Unique information:

This case report describes a complex and unique presentation and diagnosis of a dog suffering a multiple autoimmune syndrome. The identification of a single endocrinopathy should prompt a veterinary professional to consider the potential development of a poly-endocrinopathy in the patient. Additionally, practitioners need to recognize the likelihood of additional autoimmune involvement in non-endocrine organs, leading to diverse disease manifestations and clinical presentations.

E-mail: sarahhefer@gmail.com

EVECC
CONGRESS 30 MAY - 1 JUNE 2024
GOTHENBURG | SWEDEN

WILDLIFE-INDUCED POLYTRAUMA IN DOGS: A CASE SERIES OF NINE WILD BOAR ENCOUNTERS

F. Paul[1], T. Niedenführ[1]

[1] Veterinary Faculty, University of Leipzig, Leipzig, Germany

Background:

Hunting dogs are exposed to an increased risk of wildlife-induced injuries, from wild boars or other animals. In Germany, wild boars in urban areas are on the upswing, extending the risk of wild boar encounters beyond hunting dogs. Despite this development, literature documenting injuries caused by wild boars in dogs is scarce. This case report illuminates nine cases presented at a veterinary teaching hospital between 2019 and 2023.

Case Presentation:

Among the examined cases, four dogs sustained injuries during hunting activities. The most prevalent injury was perforating thoracic trauma, closely followed by large lacerating wounds and perforating abdominal trauma. All wounds exhibited significant contamination, with substantial amounts of soil and wild boar fur frequently present. Cases with perforating abdominal trauma displayed internal organ injuries, such as urethral tear-off and spleen injury. Additionally, two dogs presented with fractures—one with a tibia-fibula fracture and another with a severe mandibular fracture necessitating amputation. Upon initial presentation, all dogs were in hemodynamic shock, necessitating intensive fluid resuscitation and, in some instances, transfusion. Comprehensive surgical interventions were imperative for wound debridement, cleaning, and irrigation, especially given the extensive affected skin areas. Intensive analgesic and antibiotic treatments were administered, underscoring the severity of the injuries. While all nine dogs survived to discharge, the treatment demanded prolonged in-hospital care, resulting in substantial financial costs.

New/Unique Information:

Polytrauma arising from encounters with wild boars in dogs is notably severe, affecting critical structures and sharing similarities with injuries documented in humans attacked by wild boars. Timely presentation at the emergency department is imperative due to the high level of hemodynamic compromise and pain. The significant contamination in injuries from wild boar encounters carries a heightened risk of sepsis, emphasizing the urgency of prompt and thorough wound management.

E-mail: frauke.paul@kleintierklinik.uni-leipzig.de

NEUROLOGICAL DYSFUNCTION AND SEVERE HYPOKALIEMIA IN A CAT AFTER INGESTION OF RHODODENDRON SPP

G. Ceccherini[1], F. Romoli[1], G.B. Cherubini[2]

[1] BLUVET SpA, Lucca, Italy
[2] University of Pisa, Department of Veterinary Science, Pisa, Italy

Background:

Grayanotoxins present in Rhododendron (Azalea) plants disrupt sodium channels in cell membranes, leading to disturbances in skeletal muscles, cardiac muscles, and nerves.

Case presentation:

A 4-year-old spayed female European domestic cat was admitted to the emergency room exhibiting acute vomiting, ataxia, and open-mouth breathing. The cat showed mild responsiveness, ataxia, tachypnea, sporadic open-mouth breathing, and vocalization. Arterial blood pressure and mucous membranes were normal. Rectal temperature was 36.5°C, and heart rate was 120 bpm. Cranial nerves, menace response, pupillary response, and palpebral reflex were normal. Abdominal and thoracic focused assessment with sonography for trauma were negative. Owners denied toxic ingestion. Venous blood gas, complete blood count (CBC), and chemical panel were conducted. The cat was hospitalized, receiving intravenous Ringer lactate at 4 ml/kg/h pending blood work results. Within minutes, the cat's condition deteriorated, presenting in lateral recumbency, obtunded, vocalizing, tachypneic, with severe bradycardia (80 bpm) confirmed by electrocardiography. Simultaneously, blood gas analysis revealed severe hypokalemia (2.8 mmol/L; reference range 3.6-4.5 mmol/L) and metabolic acidosis (pH 7.34; BE-ecf -10.8 mmol/L, reference range -2 to +2 mmol/L; PCO2 22.7 mmHg, reference range 35-45 mmHg; HCO3- 15 mmol/L, reference range 21-25 mmol/L). Complete blood count and serum biochemistry were within normal limits. Potassium chloride was supplemented in a second IV line as continuous infusion titrated not to exceed 0.5 mEq/kg/h. Oxygen was also administered. Continuous electrocardiographic monitoring was conducted. N-acetyl cysteine at 50 mg/kg iv, ascorbic acid 500 mg iv, and maropitant 1 mg/kg/h iv were also administered. Owners later mentioned the potential ingestion of Rhododendron leaves by the cat. Twelve hours later, the cat showed improvement, with resolved neurological symptoms. On discharge, potassium levels were 3.5 mmol/L, and the cat was alert, had an appetite, and a heart rate of 170 bpm.

New/Unique information:

This is the first reported case of Rhododendron intoxication in a cat. Symptoms were attributed to an inability to inactivate neural sodium ion channels, resulting in continuous increased vagal tone.

Comprehensive management, including fluid therapy, potassium supplementation, and supportive care, led to successful recovery. This case underscores the importance of considering plant toxicity in feline emergencies, necessitating prompt diagnosis and intervention.

E-mail: gianilaceccherini@virgilio.it

CHYLOABDOMEN IN A DOG WITH ACUTE PANCREATITIS

G. Ceccherini[1], F. Romoli[1], C. Puccinelli[2]

[1] BLUVET SpA, Lucca, Italy
[2] University of Pisa, Pisa, Italy

Background:

Scientific reports of chylous ascites in companion animals have been limited.

Case Presentation:

A 4-year-old male Epagneul Breton presented to the emergency department with a history of acute abdominal pain, vomiting, and diarrhea persisting for 72 hours. The dog exhibited obtundation, abdominal distension, severe pain, red mucosal membranes, and a capillary refilling time of less than 1 second. Rectal temperature was 38.3°C and mean arterial pressure (MAP) was 120 mmHg. Abdominal ultrasound revealed abundant effusion, peritoneal reactivity, pancreatitis, and gastroenterocolitis. Abdominocentesis revealed milky abdominal fluid with elevated triglycerides (260 mg/dl vs. 60 mg/dl in serum). Cytologic analysis showed 1000 erythrocytes/µL and 1500 leukocytes/µL, with no bacteria or neoplastic cells visualized, suggesting chylous ascites. Blood counts, biochemistry, and echocardiography were unremarkable. CT scan confirmed acute pancreatitis and suspected multifocal peritonitis. Moderate lymphadenopathy and mild bilateral pleural effusion were also observed. The dog was hospitalized and received intravenous Ringer lactate solution at 4 ml/kg/h, fentanyl constant rate infusion (CRI) at 2 µg/kg/h, lidocaine CRI at 1 mg/kg/h, maropitant at 1 mg/kg, and ranitidine at 2 mg/kg. The dog continued hospitalization with fluid therapy, a modulated analgesic plan, and nutritional management via a nasogastric tube for 5 days. Upon discharge, abdominal ultrasound showed significant improvement in pancreatitis, with no detectable abdominal fluid.

New/Unique Information:

The positive outcome achieved without the need for exploratory laparotomy highlights the effectiveness of medical treatment, pain management, gastrointestinal symptom control, continuous monitoring, and nutritional management. This stands in contrast to other reports where the outcome was predominantly unfavorable or necessitated exploratory surgery.

E-mail: gianilaceccherini@virgilio.it