2019 Emergency & Critical Care Conference

Proceedings

TEXAS A&M UNIVERSITY
Veterinary Medicine & Biomedical Sciences
Office of Veterinary Continuing Education

2019 EMERGENCY AND CRITICAL CARE CONFERENCE: Presenters Biographies

Dr.Gareth Buckley, DVM - Dr. Buckley went to veterinary school at the University of Cambridge, UK followed by a rotating internship at the Royal Veterinary College, London. He then moved to Massachusetts where he completed a specialty internship followed by residency in Emergency & Critical Care. He became a Diplomate of the American College of Veterinary Emergency & Critical Care in 2010. He joined the faculty at University of Florida in the Summer of 2010. Dr Buckley was also made a Diplomate of the European College of Emergency & Critical Care in 2015. His research interests include CPR, respiratory disease and patient safety. He is currently a Clinical Associate Professor of Emergency & Critical Care and Chief Medical Officer of the Small Animal Hospital at University of Florida in Gainesville, FL.

Dr. Alison B. Diesel, DVM, DACVD – Clinical Associate Professor. Dr. Alison Diesel is a Clinical Asssociate Professor in Texas A&M University. She graduated from Kansas State University College of Veterinary Medicine in 2005 then completed a rotating internship in small animal medicine and surgery at the Veterinary Referral and Emergency Center in Norwalk, Connecticut She worked as an emergency clinician for one year prior to beginning a three-year residency in dermatology at the University of Wisconsin-Madison School of Veterinary Medicine; she became board certified (ACVD) in 2010. She joined the faculty in the fall of 2010 to continue to expand the growing dermatology service and to help guide veterinary students in the management of skin disease in companion animals. Her main research interests lie in feline dermatoses, expanding knowledge of the cutaneous microbiome in companion animals, and methicillin-resistant Staphylococcal skin infections.

Dr. Justin Heinz, DVM - Clinical Assistant Professor. VSCS. Dr. Heinz graduated from Purdue University and went on to complete a small animal rotating internship at Louisiana State University, followed by his residency in emergency and critical care at Texas A&M University. He also is a diplomate of the American College of Veterinary Emergency and Critical Care. He currently works in emergency and critical care in the Department of Veterinary Small Animal Clinical Sciences.

Dr. Jonathan Lidbury, BVMS, MRCVS, PhD, DACVIM, DECVIM-CA- Assistant Professor. VSCS. Dr. Lidbury graduated from University of Glasgow, Scotland. He worked for several years in general and referral practices in the United Kingdom before completing an internship in small animal medicine and surgery at the California Animal Hospital, Los Angeles, California. He then joined the Texas A&M University GI Lab as a PhD student and he started his residency in small animal internal medicine and received his board certification in 2011. His areas of interest include small animal gastroenterology and is working to develop new non-invasive tests for liver disease in dogs infections with a particular emphasis on feline *Tritrichomonas foetus* infection. A list of her peer-reviewed publications and grants can be viewed at: orcid.org/0000-0001-8725-9530:

Dr. Christine Rutter, DVM- Clinical Assistant Professor. **VSCS.** Dr. Rutter graduated from Mississippi State University, completed an internship in Louisville, Kentucky and a residency at Tufts University. She was in private specialty practice for 6 years. Her interests include coagulation, immune mediated blood dyscrasias, transfusion medicine, trauma management, post-operative patient care, and respiratory disease.

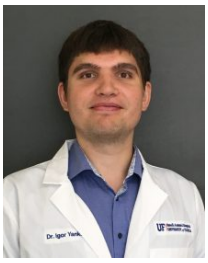
Dr. Amy Savarino, PharmD, DICVP, FSVHP - Pharmacist. VMTH. Dr. Amy Savarino is the Chief Pharmacist at the Texas A&M University Veterinary Medical Teaching Hospital. She was previously the Small Animal Pharmacist for 9 years. Amy received the W. Terry Stiles award which

formally recognizes hospital staff who demonstrate outstanding service in support of the VMTH through exceptional skills in many or all of the following areas: clinical service, patient service, client service, and service to profession..

Dr. Lucien Vallone, DVM- Clinical Assistant Professor. VSCS. Dr. Vallone graduated from Mississippi State University. He is a Diplomate of American College of Veterinary Ophthalmologists. His areas of interest include diseases and surgery of the ocular surface.

Dr. Emma Warry, DVM - Clinical Associate Professor. Dr. Emma Warry joined the Texas A&M College of Veterinary Medicine & Biomedical Sciences, Department of Small Animal Clinical Sciences as a clinical associate professor of medical oncology in 2018. Dr. Warry obtained her veterinary degree from University of Sydney and went on to complete a residency in medical oncology at Colorado State University, as well as a fellowship in bone marrow transplantation at North Carolina State University. In 2013, she joined the faculty at Ohio State University as a clinical assistant professor and will complete her master's degree in clinical trial design at OSU in 2019.

Dr. Sonya Wesselowski, MS, DVM, DACVIM (Cardiology*)* - Assistant Professor. VSCS. Dr. Wesselowski graduated from Kansas State University with her MS and DVM. She then went on to complete an internship at Vet Care Animal Hospital & Referral Center and her residency in cardiology at Virginia Tech University, where she also graduated with another MS degree. Her areas of interest include degenerative mitral valve disease, echocardiographic imaging, and congestive heart failure.



Dr. Igor Yankin, DVM - Dr. Igor Yankin has joined VSCS as a Clinical Assistant Professor in Emergency & Critical Care. Dr. Yankin completed his DVM at Kuban State Agrarian University in Krasnodar, Russia. He then completed a rotating internship at Oregon State University, and a residency in Small Animal Emergency & Critical Care at the University of Florida.

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Smartphone Ophthalmology: Simple and Powerful Strategies to Use Your Phone as a Medical Device

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Ophthalmology is a medical discipline that rewards those who have a strong foundation in pattern-based disease recognition. This skill is reinforced quickly when the examiner is able to consistently visualize and photograph ophthalmic lesions. Obtaining magnified, high-resolution images of the anterior segment and ocular fundus allows the examiner to study the disease at hand more intensively. Further, photographs provide the general practitioner with the opportunity to consult with colleagues and/or ophthalmologists, to bolster the medical record with an accurate representation of disease, and to engage with clients more effectively. The concepts in these notes emphasize smartphone photography, but lean on fundamental concepts of ophthalmic photography in general that are described thoroughly elsewhere.[1]

Using a smartphone as an ophthalmic examination tool has 3 major benefits:

1) Magnification: a) Without accessory equipment, ~ 2-4x magnification can be achieved with very simple techniques. b) With inexpensive accessory equipment, ~ 4-10x magnification can be achieved!
2) Fundoscopy: Many veterinarians are surprised to find that their smart phone allows for an easy method of performing a) direct fundoscopy and b) indirect fundoscopy.
3) Photographs and videos: The ability to easily capture and save high-quality images of the eye is an invaluable benefit to the medical record, client education, and our own continuing education.

1a) Using the smartphone to achieve magnification. Method I – No accessory equipment:

- Android and iPhone smartphones differ slightly, however the concepts are similar between the two platforms.
- Move the phone to the closest point (about 2.5 inches or 6-7cm) at which the eye is still in focus and take an image or video.
- Select the most representative image(s).
- Zoom and crop the image. This can be accomplished using native camera software (iPhone or Android devices) or by merely taking a screen shot (press the home key and power key simultaneously) after zooming into the original photo. Zoom and crop once for ~2x magnification or twice for ~4x magnification (this is the limit at which the image typically becomes pixelated).

1b) Using the smartphone to achieve magnification. Method II – Utilizing accessory equipment:

- There are MANY commercial macroscopic lens attachments marketed for iPhone and Android smartphones.

- The cost of macrolens accessory products range from $5 to $400. I routinely use a macrolens that is sold for $10 and have been very happy with the results.
- The magnification ranges between 4x and 10x for most macrolenses, however 25x to 100x microscope attachments are also commercially available. I use a 4x macrolens and manipulate images as outlined above to double the magnification and emphasize specific ocular lesions.
- Most macrolens attachments are not compatible with the native smartphone LED light source and an external light source is needed to illuminate the eye. An assistant holding a Finoff transilluminator can provide appropriate illumination to the eye in a dim room to achieve high quality images. Alternatively, overhead surgical lamps can be employed.
- The focal length is MUCH shorter when using macrolens attachments and the phone needs to be moved to within ¼ to ½ of an inch, or 6 to 13mm.
- Due to the motion of the patient, I choose to capture a slow motion video when using a macrolens. The high frame rate of the video allows me to pause, slide to the best-focused image(s), take a screenshot, and then save or modify the selected image.
- Using these techniques, I have been able to demonstrate MANY common ocular lesions including subtle distichia, faint corneal neovascularization, corneal dystrophies and degenerations, lens instability, and many more similar pathologies. The results have been very rewarding.

2) Smartphone fundoscopy:

Smart phones can be utilized as a direct or indirect ophthalmoscope for the purpose of fundoscopy with or without the aid of commercial accessory products. The following section outlines fundoscopy *without* the use of commercial accessory products. The baseline requirement to perform this technique relies on a continuously running LED during camera mode settings. This feature became available in the iPhone with the release of the iPhone 4 and was seen more commonly in smartphones running on the Android platform around the same time (2010).

2a) Fundoscopy with the direct technique using the iPhone:

- This is ideally performed in a dark room environment, in patients without senile nuclear sclerosis (i.e. less than ~7 years of age) or significant lens opacity, and with a dilated pupil, though this is not required.
- Dampen the LED light by placing a strip of white porous medical tape over the LED light source. This reduces glare from the tapetum and is preferred for the patient's safety and comfort.
- Set the camera-mode to slow-motion video and set the LED to run continuously.
- The phone is directed towards the patient and placed within 1-4cm of the eye until the image is focused on the display. Note, that the focal point will vary depending on the species examined.
- Tapping the display brings objects of interest into better focus and adjusts the camera's ISO (light sensitivity) during the exam, providing a crisper image.
- Images are selected and saved as outlined above.

- Advantages of this technique: The direct technique is easy to learn and technically simple to perform.
- Disadvantages of this technique: The direct technique can be more challenging with phones that have a wider distance between the camera lens and the LED light. For instance, the iPhone 6 plus creates shadowing of the fundus unless the pupil is extremely dilated. Additionally, many smartphones have difficulty avoiding autofocus of lens opacities where they are present. Senile nuclear sclerosis, and subtle cataracts will often interfere with a clear view of the fundus with the direct technique.

2b) Fundoscopy with the indirect technique using the iPhone:

- The room environment and phone are set up as outlined above
- A condensing lens (20D, 2.2D, or 28D) is used in a similar manner to traditional indirect ophthalmoscopy: Briefly, after a fundic reflex is generated by holding a light source at arm's length away from the patient, the condensing lens is positioned over the eye.
- Unlike traditional indirect ophthalmoscopy, in which the light source stays aligned with the examiners dominant eye, the smartphone is moved between the condensing lens and the examiner so that an image is viewed on the smartphone display.
- Tapping the display brings objects of interest into better focus and adjusts the camera's ISO (light sensitivity) during the exam, providing a crisper image
- Images are selected and saved as outlined above.
- Advantages of this technique: Wider field of view. Ability to view the fundus clearly despite lens opacities or a small pupil. This technique is less dependent on a coaxial light source. For instance, the iPhone 6 plus does not cast a shadow on the fundus with this technique (in contrast to the direct technique).
- Disadvantages of this technique: Technically more challenging. Adequate restraint and manipulation of the eyelids is essential.

3) Photographs and videos using smartphones:

The benefits of photography are outlined below:

- Examiner has the ability to review and study the abnormality more intensively.
- The medical record is enhanced with photographic documentation of change over time, adding objectivity to an otherwise subjective examination.
- The general practitioner can engage more effectively with the client by displaying subtle lesions in real time. Owners are more likely to accept treatment recommendations and more likely to be compliant with these recommendations if they can actually see the abnormality.
- Collaboration with colleagues and/or consultation with specialists is improved.

References

1) RJ. McMullen, Jr., NJ. Millichamp, and CG. Pirie DJ., Veterinary Ophthalmology, 5th ed., John Wiley & Sons, Inc. Ames, IA, 2013. pp. 729-789.

Canine Glaucoma: Examination and Treatment Strategies

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The topic of canine glaucoma is complex and our understanding of diagnostic and treatment strategies are constantly evolving. Educational resources (online journals, review articles, text books, etc.) have a tendency to broadly review this subject and the general practitioner may have a difficult time discerning what topics to apply in practice. These notes provide the opposite approach, taking an intentionally minimalist strategy to emphasize the most recognizable examination features of three broad and simplified categories of glaucoma. Then, the most basic treatment strategies for inherited primary canine glaucoma are discussed. The following outline will be followed:

1) Definition, simplified classification, and examination features of canine glaucoma
2) Discussing treatment objectives and prognosis with the owner
3) Treatment for acute inherited primary glaucoma in the visual eye
4) Maintenance therapy for the inherited primary glaucoma (affected eye and prophylaxis for the unaffected eye)
5) Surgical therapies for the blind and chronically uncomfortable eye

1) Definition, simplified classification, and examination features of canine glaucoma:

Definition: Glaucoma in the dog is currently defined as a pressure >25mmHg associated with progressive vision loss.

- Both components of this definition are required to establish a diagnosis. Therefore, the examiner needs to feel comfortable assessing vision (see examination features). Along the same lines, the diagnosis requires tonometry. If a tonometer is not available, any patient with a red eye should be referred to a practice that has a tonometer.
- It is quite common to obtain erroneously high pressures in patients who are fractious, brachycephalic, or painful (squinting), due to unintended iatrogenic pressure on the globe or neck. Trust your lowest reading.

Simplified classification: Examination features of each will be described, however, these 3 categories lead to the most extreme variations in examination features, diagnostic work-ups, and treatment.

- Primary inherited glaucoma
- Inherited anterior lens luxation-induced glaucoma
- Glaucoma secondary to uveitis, hyphema, or neoplasia

Examination features that are non-specific to the three forms of glaucoma: It is important to make a special note of acute vs. chronic clinical signs as this distinction guides therapeutic recommendations.

• Pain – Acute is more likely to show blepharospasm (squinting), epiphora (tearing), head shyness, vocalization, or even aggression. Chronic pain is more subtle but is remarkably consistent between animals. Owners will often concede that their dog is more lethargic, playing less, and sleeping more often.

• Episcleral injection – Seen in acute or chronic presentations. Robust, occasionally tortuous subconjunctival vessels that move toward the limbus.

• Corneal edema – Seen in acute or chronic presentations. Diffuse corneal edema has been reported to occur when the IOP exceeds 40mmHg.

• Optic nerve cupping and retinal degeneration – These features can be directly visualized within 5 days of the onset of glaucoma, however, they are become more prominent in chronic presentations. These features may be difficult to visualize for many practitioners. Assessment of vision loss is equally important!

• Vision loss – Can occur within 24 hours of disease onset. Confirmation of vision loss is often overlooked in unilateral presentations of glaucoma. The examiner needs to feel confident determining if the affected eye has a direct pupillary light reflex (PLR) and if this reflex is delivered to the unaffected eye (i.e. consensual PLR). Proper technique for testing the menace response (no noise, no airflow to the face) is critical. Finally, the examiner should assess the dazzle reflex with the brightest light source available.

• Buphthalmia – Chronic clinical sign. Globe enlargement takes several days to occur in most breeds. Puppies and Shar-Peis are notable exceptions in which globe enlargement can occur rapidly.

• Haab's stria, aka, Descemet's streaks – Chronic clinical sign. These represent fractures of Descemet's membrane.

• Lens sub/luxation - Chronic clinical sign. Occurs following stretch of the globe and detachment of ciliary zonules from the equator of the lens.

Examination features more commonly found in inherited primary glaucoma:

• Consistent breed – Basset Hounds and Cocker Spaniels are the two most commonly affected breeds. Labrador Retrievers, Boston Terriers, Chow Chows, Samoyeds, and Flat-Coated Retrievers have also been shown to have inherited, primary glaucoma.

• Consistent age – Young/middle aged dogs (4-8 years) are most commonly affected.

• Mydriasis – Acute or chronic lesion. Damage to the optic nerve and mechanical pressure exerted on the iris stroma lead to pupil dilation. This is an especially helpful distinction when contrasted with secondary, inflammatory causes of glaucoma.

• Absence of intraocular inflammation – Inflammation may be present, however, this is not the most prominent feature.

Examination features more commonly found in inherited anterior lens luxation-induced glaucoma:

• Consistent breed – Numerous small terrier breeds (Jack Russell Terrier, Yorkshire Terrier, Miniature Bull Terrier, etc. etc.) have a genetic predisposition to develop lens instability.

• Consistent age – Young/middle aged dogs (4-8 years) are most commonly affected.

• Anterior lens luxation – This can be challenging to diagnosis. Pay particular attention to the pupil margin. Can you see the entire pupil margin crisply/clearly? If not, then light refraction from an acute anterior lens luxation may be responsible. Purkinje-Sanson images reflected from

the smallest circle of light produced by the direct ophthalmoscope can also be used to detect depth of ocular opacities and lesions within the anterior chamber.

Examination features more commonly found in glaucoma secondary to uveitis, hyphema, or neoplasia:

- Miosis – Inflammatory pupil constriction is mediated by prostaglandins, a markedly miotic pupil is more suggestive of secondary glaucoma than primary glaucoma.
- Iris bombe – 360 degree posterior synechia (iris attachment to the lens) that results in an anterior bulging of the iris. Pathognomonic for uveitis.
- Dyscoria – An abnormally shaped pupil is often associated with posterior synechia and is consistent with uveitis.
- Deep corneal vessels – Termed ciliary flush when present 360 degrees around the limbus. These vessels look like a hedgerow, or crown of thorns and suggest intraocular inflammation. Pathognomonic for uveitis.
- Hyphema – Apparent as a red, dependent fluid line within the anterior chamber or a uniformly red eye when complete.
- Hypopyon – Apparent as a white, dependent fluid line within the anterior chamber.
- Keratic precipitates – Stippled cellular adhesions to the ventral aspect of the corneal endothelium. Pathognomonic for uveitis.
- Intraocular mass – The most common primary neoplasm is melanocytoma and often causes marked dyscoria and expansion of the iris/ciliary body. Primary tumors do not often cause marked intraocular inflammation, but are often responsible for causing glaucoma. Lymphoma is the most common metastatic tumor found in the eye. Metastatic tumors are known for causing severe and intractable intraocular inflammation.

2) Discussing treatment objectives and prognosis with the owner:

Three treatment objectives for glaucoma:

- Vision – poor prognosis
- Comfort – good prognosis with medical and/or surgical intervention
- Cosmesis – variable prognosis depending on the surgical procedure elected

Prognosis:

- Inherited primary glaucoma: Set the expectations at the first evaluation! This disease unfortunately causes bilateral blindness and pain in the majority of affected patients. Unilateral presentations are most often blind on presentation and owners should be made aware that glaucoma is likely to occur within 6-12 months (8 months on average) in the unaffected eye without prophylactic therapy. With prophylactic therapy, glaucoma onset can be delayed to approximately 3 years. Note that emergent therapy for salvaging vision in a buphthalmic eyes is typically not required, as these eyes have a grave prognosis for vision. Options should instead be presented for managing chronic discomfort.
- Inherited anterior-lens luxation glaucoma: Grave for vision with medical management alone. Guarded for long-term vision with surgical therapy as approximately 50% of dogs experience blindness within 2 years following either surgical extraction or manual reduction of an anteriorly luxated lens.

- Glaucoma secondary to uveitis, hyphema, or neoplasia: The conversation with the owner needs to deviate away from the eye-specific objectives and instead focus on the dog's systemic health. Enucleation is often discussed as a simultaneously diagnostic and therapeutic option for these cases.

3) Treatment for acute inherited primary glaucoma in the visual eye:

Following a thorough assessment of vision where the PLR, and/or, menace response, and/or dazzle reflex are intact the following protocol can be utilized:

- 0.005% Latanoprost and 2% Dorzolamide: Instill 1 drop of each medication every 15 minutes for 1 hour. Reassess the intraocular pressure. If controlled transition to maintenance therapy. If uncontrolled, transition to mannitol and consider referral.
- Intravenous mannitol: 1g/kg over 20 minutes through a filter. Caution in heart failure, renal failure, and diabetic patients.

4) Maintenance therapy for inherited primary glaucoma (affected eye and prophylaxis)*:

- Affected eye: 0.005% Latanoprost q12 hours (most effective therapy for primary glaucoma in dogs) combined with 2% Dorzolamide q8 hours.
- Unaffected eye: 2% Dorzolamide q12h (this is an area that needs more research).
- *This is an example of a minimalistic approach that this author often utilizes, especially in circumstances where owner compliance is uncertain. It should be noted that additional therapies are often advocated, including anti-inflammatory therapies, neuroprotective therapies, and oral anti-hypertensive therapies for both the affected and unaffected eye.

5) Surgical therapies for the blind and chronically uncomfortable eye:

- Enucleation – fewest complications, poor cosmetic outcome.
- Evisceration an intrascleral prosthesis – cosmetic option in which the cornea and sclera are retained.
- Chemical cycloablation – intravitreal injection with gentamicin or cidofovir.

References available from the author

Cardiopulmonary Resuscitation

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Cardiopulmonary arrest is often an unexpected event however there are a few steps that can be taken to try to prevent it from happening. Careful and frequent monitoring of patients will help in identifying those at risk of arrest. It is also important to record and communicate any clinical patient changes in the record and to the rest of the staff including technicians. Having clearly written orders for when a staff member should alert a doctor is key.

Being prepared is an important part of achieving a successful CPR. The first step is talking to owners about their wishes. In an emergency clinic it is a good habit to ask any owner what their CPR wishes are (do not resuscitate, closed chest or open chest CPR). Having a dedicated crash cart in an area with room for the animal, multiple people and access to oxygen is the next step. A crash cart should include drugs (see chart at the end), endotracheal tubes and stylets, laryngoscope, long polypropylene catheters, tracheostomy tubes, a surgical instrument pack, sterile gloves, the sharpest scissors in the hospital, blades, thoracocentesis set up, and fluids with administration set and pressure bag. IVC supplies should be housed somewhere nearby. A defibrillator and suction set up should also be stored with the crash cart. There should be a way to supply 100% oxygen and positive pressure ventilation as well (anesthetic machine or oxygen line with ambu bag).

Recognizing arrest is the next step. The patient may have a respiratory arrest alone or in combination with cardiac arrest. Assessment of the ABCs (airway breathing and circulation) must be performed rapidly. The patient will not be breathing, have no heart beat or a severe bradycardia and will be unconscious. Do not spend time palpating for a pulse in an unresponsive apneic pet. Once an arrest is confirmed the next step is to call a code so that there are enough people to help.

<u>The CPR team:</u>

- One leader who is giving clear and directed instructions to each individual
- Responders are people receiving and completing the tasks the leader has given them
- A recorder to document what was done, dosages and timing

<u>Performing CPR:</u>

The only things we do during CPR that have proven to make a difference are chest compressions, ventilation and defibrillation. As such these should be the most important focus of CPR.

Basic Life Support (BLS) comprises compressions and ventilation. An airway should be secured with an endotracheal tube that is inflated and ventilations begun at 6-10 bpm. Avoid hyperventilation as it leads to further cerebral and coronary ischemia. A breath is given to see a slight visible chest rise (around 10 ml/kg tidal volume) at a maximum pressure of about 20 cmH_2O with an inspiratory time of 1 second. 100% oxygen should be used if available. If an endotracheal or tracheostomy tube cannot be placed provide mouth to snout breaths.

Compressions should be begun in lateral recumbency while the endotracheal tube is being placed. (barrel chested dogs may be put in dorsal recumbency). The goal is to achieve 100-120 bpm with 30-50% compression of the thorax. The chest wall must be allowed to fully recoil before the next compression. There are 2 ways to provide compressions:

Mechanism 1- The Cardiac Pump: This mechanism relies on the heart being directly compressed to form an artificial systole. Hands are placed directly over the heart and compress it, this works for patients < 15 kg or those with very compliant chest walls such as cats.

Mechanism 2- Thoracic Pump: This relies on changes in intrathoracic pressure and the arrangement of one way valves in the heart and great vessels providing forward blood flow. Hands are placed on the widest point of the chest in lateral recumbency, to provide the largest amount of chest wall movement. This is the primary mechanism in animals > 15 kg.

A 2 minute cycle of compressions should be performed with the person performing compressions changed after each cycle to avoid fatigue. Any interruptions in compressions should be kept to a minimum to avoid loss of all the foreward flow you have worked so hard to generate. There should be no interruptions between changing compressors (new person places hands over current compressor's and then the current compressor slides their hands out as the new person takes over).

If you are by yourself a compression to ventilation ratio of 30:2 should be performed and then repeated.

AFTER compressions and ventilation are started catheters can be placed and monitors connected. Use ECG gel and not alcohol for lead placement.

Advanced Life Support (ALS) comprises everything else we do and is not started until after BLS.

ECG:

- Necessary to assess rhythm and direct needed therapy
- 4 rhythms associated with CPA
    - Pulseless electrical activity
    - Asystole
    - Ventricular fibrillation
    - Pulseless ventricular tachycardia

Rhythm Analysis in CPA:

There are four recognized arrest rhythms in CPA (see Figures 1-4): asystole, pulseless electrical activity (PEA), ventricular fibrillation (VF), and pulseless ventricular tachycardia (pVT).

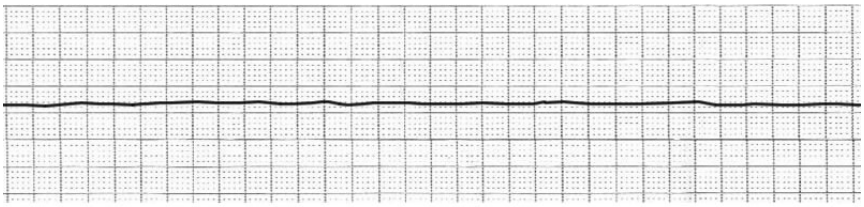

Figure 1. Asystole.

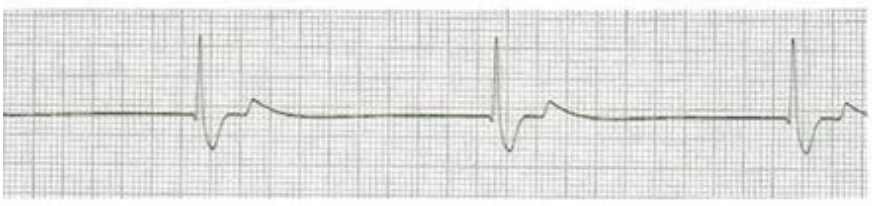

Figure 2. Pulseless electrical activity.

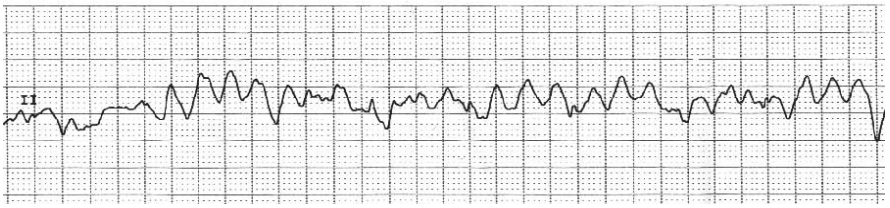

Figure 3. Ventricular fibrillation.

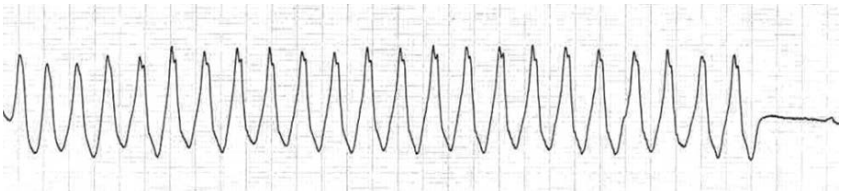

Figure 4. Pulseless ventricular tachycardia.

Drugs:

- First reverse any previously given analgesics/sedatives if possible and turn off anesthesia
  - Opioids – naloxone
  - Benzodiazepines – flumazenil
  - Alpha 2 agonists - atipamezole
- Vasoconstriction (to increase MAP/SAP) is attempted with epinephrine or vasopressin.
  - Epinephrine
    - Non-specific adrenergic agonist used for alpha effects in CPR
    - Beta1 effects can be detrimental due to inotropic and chronotropic effects leading to increase myocardial oxygen consumption and pro arrhythmogenic effects. High dose epinephrine is more likely to cause these negative side effects so should only be used if multiple low doses have not worked.
    - Repeat 0.01mg/kg dose every 3-5 min or every other 2 minute cycle of compression. Recent research suggests that early administration of epinephrine may be of benefit.
  - Vasopressin
    - Used for V1 receptor mediated effects in CPR
    - No detrimental Beta effects
    - Now very expensive and generally not used as little benefit over epinephrine
- Atropine is used to reduce parasympathetic tone. It should be used whenever suspicion of a vagally mediated arrest occurs – this can include animals with severe respiratory distress, severe gastrointestinal disease (especially vomiting, GI distention) or animals undergoing mechanical ventilation. It is the first line treatment for sinus bradycardia. Dose is 0.04mg/kg.
- Antiarrhythmics
  - Ventricular tachycardia can be treated with lidocaine or amiodarone.
  - Bradycardia treated with atropine
- Calcium gluconate used if hyperkalemic, hypocalcemic or calcium channel blocker toxicity
- Sodium bicarbonate is only used if CPR is prolonged (>15 min) or if blood gas analysis demonstrates a severe metabolic acidosis.
- All drugs should be given IV or IO.
- Intratracheal administration is ok for naloxone, atropine, vasopressin, lidocaine, epinephrine. These drugs should be diluted in sterile water or saline and given down a long catheter placed down the endotracheal tube followed by a breath. Doses are usually doubled. Epinephrine has been given up to 10 x the IV dose.
- Intracardiac injections are contraindicated.

Fluids:

- Use small IV fluid boluses (6-10mls) to help push administered drugs into central circulation
- Large IV boluses of fluids should not be given unless the patient was already hypovolemic prior to the arrest as this will only increase right atrial pressure and therefore decrease coronary perfusion.

Defibrillation:

- Being able to recognize common arrest rhythms is key.
- Defibrillation is the only treatment for ventricular fibrillation and can also be used for pulseless ventricular tachycardia
- If the arrest has been longer than 4 minutes a 2 min cycle of CPR should be performed prior to defibrillation.
- Monophasic current is in one direction. 4-6 J/kg
- Biphasic current is found on more modern defibrillators and flows in both directions, may be more effective and allows for lower doses. 2-4 J/kg
- Make sure there is no alcohol on the patient, place conductive gel on the paddles (make sure not on yourself) and place a paddle on each side of the chest over the heart at the level of the costrochondral junctions. Tell everyone to stand back and avoid touching the patient or table. Check that everyone is safely away, shout "CLEAR" and discharge the device. Immediately start compressions for 2 min before assessing the ECG.
- If unsuccessful after the 2 min CPR cycle the dose can be increased by 50%.
- As the dose increases so does the risk of myocardial ischemia
- A single shock followed by immediate resumption of chest compressions allows for minimal interruption in forward blood flow.
- If defibrillation is not effective you may try amiodarone.
- ONLY if no defibrillator is available, a precordial thump may be attempted. The heel of the hand is used to strike the patient directly over the heart. This is unlikely to be effective and may be harmful.

ETCO2:

- Circulation of CO_2 through the body to the lungs is used to assess adequacy of CPR and ROSC
- In ROSC $ETCO_2$ will increase greatly
- Goal of 15 mmHg in dogs and 20 mmHg in cats during CPR. If this is not being met change how compressions are being performed or compressor.

Others

- Blood gas to check electrolytes and glucose – venous.
- Interposed abdominal compressions: the abdomen is compressed during the relaxation phase of cardiac compressions. The goal of this is to increase venous return from the abdomen and improve cardiac output.
- Open chest compressions may be considered in these instances
    - Pleural space disease
    - Thoracic wall disease
    - Pericardial effusion
    - Diaphragmatic hernia
    - Unwitnessed arrest
    - Closed chest CPR > 15 minutes.

ROSC: return of spontaneous circulation is identified when there is return of a perfusing heart rhythm and a rapid increase in $_{ETCO2}$ as pulmonary blood flow increases.

After ROSC

- Check electrolytes as often become hyperkalemic and hypocalcemic
- Provide ventilator support as needed
    - Maintain normal (not hyper…) oxygenation
    - Avoid hypo- or hypercapnia so as to limit changes in cerebral blood flow and perfusion
- Provide pressor support as needed, often placed on a dopamine CRI
- Mild induced hypothermia (34-37˚C, 93-98.6˚F)- only if have advanced critical care capabilities as multiple detrimental event can occur
    - Only for patients that remain comatose
    - Start ASAP and maintain for 24-48 hr
    - If a patient with ROSC has naturally induced mild hypothermia it is reasonable to not rapidly rewarm them (0.25-0.5˚C/hr)
- Consider hyperosmotic therapy to reduce cerebral edema based on individual case

Prognosis after CPR is variable dependent on the cause of the cardiac arrest. Survival for sick patients in the ICU is less than 5%, however some circumstances such as anesthesia related arrest may carry a much better prognosis.

Approach to Severe Trauma

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Treatment of badly traumatized animals is amongst the most challenging aspect of emergency medicine. These animals are very often critically ill and have multiple serious injuries demanding your attention. Unlike most groups of critically ill patients, young animals are overrepresented and owners will often go to great lengths to try to save them. The aim of this lecture is to review common traumatic injuries, resuscitation strategies and decision making in animals with severe trauma.

Triage and initial assessment

All animals with severe trauma should be triaged back to a treatment area. Assessment is the same for any critically ill animal, as a guideline follow the "major body systems" rule: cardiovascular, breathing and neurological function.

Cardiovascular assessment should include heart rate, rhythm and auscultation, peripheral pulse quality, mucous membrane color and capillary refill time. Evidence of hypovolemia means that you need to go looking for possible sites of bleeding. Arrhythmia can be from one of two sources – thoracic trauma will commonly lead to myocardial contusion and secondary arrhythmia, severe hypovolemia can lead to myocardial hypoxia.

Assessment of breathing is more challenging, as successful treatment requires you to localize the source of the dyspnea. Causes of dyspnea in trauma include lung injury (pulmonary contusions), pleural space disease (pneumothorax, diaphragmatic hernia, hemothorax) or chest wall injury (rib fractures, flail chest). As a rule, pulmonary contusions will cause an increase in lung sounds and sometimes crackles on auscultation, pleural space disease will cause a decrease in lung sounds. The problem is that commonly these conditions occur together, so severe pulmonary contusions made quiet by a significant pneumothorax may mean that the patient auscults normally, in this situation asymmetric lung sounds may be your best clue that something is wrong. One trick is to consider a diagnostic thoracocentesis, in dogs especially, this is a fairly benign procedure that can quickly get you answers.

Neurological function should be rapidly assessed; at this stage we are concerned with brain function and obvious evidence of spinal fractures. An assessment of mentation is often all that is required at this stage – an animal that is clearly awake and responsive is unlikely to have significant brain injury. If the animal is dull, obtunded or comatose then a more in depth examination of cranial nerves is warranted. Clues that the spinal cord may be damaged include forelimb extensor rigidity, pain or irregularities on palpation of the spine, loss of deep pain or anal tone.

Laboratory testing

Ideally all traumatized animals should have a PCV and TS, lactate, blood glucose and electrolytes. The PCV/TS can give you a lot of information on the cardiovascular status of your

patient; a surprisingly high PCV (>50%) especially when coupled with a low TS (<6.5g/dL) is a good indicator that your patient has significant blood loss, the high PCV is due to splenic contraction in response to hypovolemia. Coagulation testing (PT/PTT, platelet count) can also be extremely helpful in guiding your resuscitation strategies. Where available, blood typing should be performed if there is a likelihood of the patient receiving blood tranfusion products.

Common injuries in trauma

In this section, we will discuss different types of trauma and the common injuries that are associated with each.

High velocity trauma usually involves the animal being hit by a motor vehicle. Common injuries seen (separated by body system) include:
Head: severe head trauma, jaw fractures, skull fractures, ocular injuries
Thoracic: rib fractures, pulmonary contusions, pneumothorax
Abdominal: hemoabdomen commonly from splenic fracture, liver lobe fracture or mesenteric vessel rupture
Orthopedic: long bone fracture, pelvic fractures, spinal fracture, traumatic disk prolapse
Integument: shearing and limb degloving wounds

Crush injuries are usually seen in pets when the wheels of a vehicle go over the thorax or abdomen. A common scenario is an older dog sleeping behind the car which gets reversed over. Injuries particularly seen in this kind of trauma include:
Thoracic: diaphragmatic hernia
Abdominal: urinary bladder rupture
Orthopedic: severe comminuted pelvic fractures
Integument: body wall hernia, degloving wounds

High-rise injuries are seen most commonly in cats that live in apartment blocks. Specific typical injuries include:
Head: severe head trauma, mandibular fractures, hard palate fracture, ocular injuries
Thoracic: pulmonary contusions and pneumothorax
Orthopedic: spinal fracture

Bite wounds, severe trauma from bite wounds is typically seen in cats and small dogs attacked by larger dogs or wild animals. Commonly seen injuries include:
Head: depressed skull fractures, ocular trauma
Thoracic: penetrating wounds, lung lobe contusion and laceration, pneumothorax, hemothorax, rib fractures, flail chest
Abdomen: penetrating wounds, internal organ damage
Orthopedic: open fractures
Integument: body wall hernia

Resuscitation strategies in trauma

Fluid choices in trauma are the subject of much debate, there is little clinical research for veterinary situations, but there is much experience in human trauma, especially in the military. When thinking of resuscitation strategies it is essential to think of severe trauma not as just

mechanical damage but to think of it in the same way that we think of other life threatening conditions such as sepsis – in other words we have the underlying injury that we can (hopefully) fix, but we also have to deal with the body's response to that injury and the physiologic derangements that come with it. Thinking of severe trauma as a "sepsis like syndrome" can be helpful to formulating a battle-plan to deal with the severely traumatized patient.

One major component of severe trauma, especially trauma involving major blood loss is development of coagulopathy. This comes from a combination of blood loss (consumptive coagulopathy), fluid resuscitation (dilutional coagulopathy) and intrinsic development of coagulopathy secondary to the trauma itself (mechanism not well understood). If you are dealing with major blood loss it is essential to monitor the patient for hemostatic dysfunction.

There are several strategies for resuscitation in trauma, when using any of them have in the back of your mind that you want to resuscitate just enough but not too much – over resuscitation can be as damaging as under resuscitation.

Conventional resuscitation usually involves the use of isotonic crystalloid solutions. Traditionally these are administered at doses equal to three times the estimated blood loss. The advantages of this strategy are that the fluids are widely available, inexpensive, and easy to administer in an emergency. The significant downside is that these fluids have no oxygen carrying capacity and carry no plasma proteins. Use of large volumes of crystalloid solutions is associated with significant worsening of dilutional coagulopathy. From a practical point of view, the way to approach this technique is to infuse increments of 20-30ml/kg usually over 5-15 minutes, then re-assess the patient using normal end points of heart rate, lactate, mucous membrane color, mental status etc. This can be repeated several times, but remember at some point you are going to need to give blood products if the patient does not rapidly stabilize.

Low volume resuscitation is practiced widely in human and veterinary medicine. This involves using hypertonic solutions, often in combination with artificial colloids. The advantage of this approach is that the low volumes can be delivered quickly, even if venous access is limited. The fluids used are widely available although somewhat more expensive than balanced electrolyte solutions, and use of this technique may not have such a negative impact on coagulation status as conventional resuscitation. Hypertonic saline is the most common solution used, at a dose of 2-5ml/kg over 5 minutes. Slow administration usually avoids any significant side effects due to transient hypernatremia. This dose can be repeated 2—3 times to effect. It can also be combined with administration of an artificial colloids solution at a dose of 3-5ml/kg; this will prolong the plasma expansion effects of the hypertonic saline. This strategy is particularly appropriate for patients with significant ongoing bleeding (i.e. hemoabdomen) or for head injury where large volumes of crystalloids are likely to be deleterious. It is important to remember to follow up with some isotonic crystalloids to prevent dehydration.

Hemostatic resuscitation is a relatively new concept that has been developed by the military for use in severely traumatized soldiers. The aim of this strategy is to prevent the coagulopathy associated with major trauma and fluid resuscitation as discussed above. Interest in this area grew significantly after the observation that people with coagulopathy at presentation had significantly worse outcome than those without coagulopathy. Use of this approach in human

trauma victims has significantly improved outcome. In practice, this approach is often underused due to the costs involved in using blood products, however for clients who can afford it, it is an effective strategy. It takes a little time to prepare blood products for transfusion, so conventional or low volume resuscitation is usually used in the first few minutes of the resuscitation.

Suggested practical approach to hemostatic resuscitation for a large breed (30-40kg) dog:
- 500-1000mls balanced electrolyte solution as a bolus – if cardiovascular parameters stabilize then stop. If not then continue…
- 8-10ml/kg packed red blood cells (PRBCs)
- Infuse one unit of fresh frozen plasma (FFP) with the first 2 units of PRBCs
- If transfusion beyond 2 units of PRBCs is required then transfuse PRBCs and FFP at a ratio of 1:1

In the example above, blood component therapy (PRBCs and FFP) is used, this carries one major problem – there are few if any viable platelets in stored blood products and so by using this strategy you are not addressing any thrombocytopenia mediated coagulopathy. If available, fresh whole blood (FWB) will provide platelets as well as red cells and clotting factors and so is the best choice for hemostatic resuscitation. Again, military experience has shown significantly improved outcomes with the use of fresh whole blood in victims of severe trauma.

Delayed resuscitation is a strategy only used when the patient has a known surgical lesion causing bleeding. The thought behind it is that fluid therapy is withheld until the cause of the bleeding has been surgically repaired and then the patient is aggressively resuscitated. It relies on rapid surgical intervention with an aim of time to presentation to the ER to surgical intervention being less than 15 minutes. Because of the time pressure and the lack of evidence of its usefulness in veterinary patients it is rarely used, there are other safer strategies available.

Imaging
Radiographs can be very helpful in working up the trauma patient, having said that remember that when working up cardiovasculary unstable or dyspneic patients, much relies on the physical exam and bedside tests. Radiology is the worst place that a critically ill patient can be. If you manage to stabilize your patient that is the time to take radiographs, if you cannot stabilize your patient without more information then get radiographs but do it quickly and carefully.

Thoracic radiographs are often the most useful in the trauma patient it is possible to identify contusions, pneumothorax and sometimes evidence of penetrating injuries. Abdominal radiographs are most useful to look for evidence of free gas (usually from penetrating wounds or GI tract rupture) or body wall hernia. These do not need to be perfect radiographs – they are survey films.

If there is suspicion of a spinal fracture then take good quality radiographs, start with lateral views but if these look normal and there is still suspicion of a fracture then you must take the orthogonal views – it is remarkable easy to miss a clinically significant unstable spinal fracture on single lateral views. When manipulating the animal be careful, sedation or anesthesia for

radiographs reduces muscle tone and increases the risk of a fracture displacing. Use of a radiolucent backboard is an excellent adjunct to safe spinal films.

Well described ultrasound techniques (tFAST and aFAST) are extremely useful for identifying the presence of free fluid in the abdomen and chest. This is an easy bedside test that requires minimal movement of the patient and can help guide further testing and treatment.

Orthopedic radiographs – these are rarely indicated in the emergency stage of working up a patient with severe trauma. Fractures are often straightforward to identify on physical examination. The time and restraint needed to get good quality orthopedic radiographs is time better spent treating more life threatening injuries.

Advanced imaging such as MRI, CT scanning or myelography can be very useful in work up of neurotrauma. The timing of these interventions is going to be determined by the neuro status of the patient and would likely require referral to specialist emergency centers with advanced imaging and surgical capabilities.

Positive contrast urethro-cystograms are very helpful studies for working up possible urinary tract rupture, as it is normally possible to stabilize these animals they are rarely emergent studies to complete.

Treatment of specific conditions

Severe head trauma is an immediately life threatening condition, treatment aims at reducing intracranial pressure and supporting cerebral blood flow. Fluid choices have already been discussed but hypertonic saline with or without a colloid added is an excellent choice for head trauma, it provides not only volume expansion but also reduces cerebral edema. Mannitol at a dose of 1g/kg can also be used to reduce cerebral edema. The resultant diuresis, however can cause hypovolemia if adequate follow up fluids are not administered. The use of other diuretics such as furosemide is not indicated in the management of head trauma. In patients with compromised ventilation from head trauma or in comatose patients, endotracheal intubation is usually indicated. Ventilation should be provided as needed to maintain a normal $ETCO_2$. Assessment using the modified glasgow coma scale can be helpful in providing prognostic information, advanced imaging techniques (CT or MRI scan) can be helpful in determining the extent of head trauma and the likelihood of recovery.

Traumatic hemoabdomen should be treated by hemodynamic stabilization. To avoid worsening of ongoing bleeding, consider using low volume or hemostatic resuscitation strategies. Pressure wrapping of the abdomen is unlikely to be helpful and may contribute to worsening of dyspnea. If a pressure wrap is used, it should be gradually removed to prevent re-bleeding. Surgical explore is occasionally, but rarely required. If a patient with traumatic hemoabdomen is failing to stabilize despite adequate resuscitation, usually including blood products then surgical exploration is warranted. As a guideline, fewer than 10% of cases of traumatic hemoabdomen require surgical intervention if cautious fluid therapy is employed.

Adequate treatment of dyspnea requires you to be able to localize the source of the dyspnea based on physical examination or testing. Pulmonary contusions will tend to worsen over the

first 12-24 hours and will then begin to improve. Avoiding over fluid resuscitating animals with pulmonary contusions is important, as excessive fluid will cause increased alveolar bleeding. Oxygen supplementation by facemask, nasal cannulae, oxygen cage or intubation are all effective methods of treatment of hypoxia due to pulmonary contusions. Some severely affected animals may require 24-48 hours of mechanical ventilation.

Treatment of pneumothorax is by thoracocentesis. A needle or over the needle catheter is introduced into the pleural space usually at the level of the 7^{th} or 8^{th} intercostal space (for large dogs a 1.5 inch needle is required), the needle is slowly advanced whilst applying suction from a syringe attached to an extension set and 3 way tap, once air is obtained the chest is evacuated manually. In the event that an end point is not reached or the animal requires 3 or more thoracocentesis procedures due to continuing build up of air, thoracostomy tubes are indicated. For this the animal must be anesthetized and intubated. In severe cases, continuous suction units or a one way Heimlich valve can be used to ensure continuous evacuation of the pleural space.

Arrhythmias in traumatized animals should be treated as in any other situation – correct underlying problems e.g. hypovolemia, anemia, pain. If the arrhythmia does not resolve then the animal likely has myocardial contusions. Treatment of ventricular arrhythmia with lidocaine (2mg/kg iv) will often be effective. These animals will often require follow up treatment with either a lidocaine constant rate infusion (50-75mcg/kg/min) or an oral anti-arrhythmic drug such as sotalol (1.5-2mg/kg orally twice daily). Longer acting anti-arrhythmic drugs such as sotalol should not be started until the animal is adequately fluid resuscitated as they can lower cardiac output.

Analgesia should be provided where indicated. There is a preference toward using pure agonist opioids such as hydromorphone, morphine, methadone or fentanyl as these have rapid onset of action and can be titrated and reversed should the need arise. The use of opioids as a constant rate infusion following an initial bolus is particularly helpful in these patients. If further analgesia is required, other drugs such as ketamine or lidocaine (dogs only) can be added to the infusion.

Decision making – when to go surgery

Sometimes the decision as to when to take an animal into the operating room is a challenging one. Some animals have clear indications for surgery, but due to the nature of severe trauma, they are often cardiovasculary or respiratory compromised and therefore poor anesthetic candidates. Indications for early surgery/anesthesia include:

- wounds penetrating thoracic or abdominal cavities
- evidence of damage to the GI tract
- ongoing bleeding which fails to rapidly stabilize with medical management
- spinal fracture
- neuroimaging and surgery (e.g. for disk prolapse) in an animal which has lost deep pain sensation
- diaphragmatic hernia with liver, intestines or stomach in the chest (ideally all animals with any kind of traumatic DH should be explored as soon as possible)
- body wall hernia with GI or bladder entrapment

Indications for surgery/anesthesia once the pet is cardiovasculary stable include:

- open fractures
- body wall hernia without evidence of organ malposition
- significant skin wounds e.g. degloving injuries
- urinary tract rupture (after contrast imaging)
- neuroimaging and surgery (e.g. for disk prolapse) in an animal with intact deep pain but absent motor function

Indications for surgery/anesthesia when convenient:

- closed fractures
- reconstructive procedures (skin flaps, grafts etc)
- neuroimaging and surgery (e.g. for disk prolapse) in an animal with intact motor function

Temporary stabilization techniques for surgical disease

Sometimes early surgery for certain conditions is not possible because of the status of the animal, facilities or personnel available. In this circumstance, there are a variety of tricks you can use to stabilize the pet temporarily. Remember that if a dyspneic animal requires emergency procedures, intubation and ventilation during the procedure is mandatory – this way you have control of the airway and are less likely to run into a crisis situation.

Large skin wounds/deficits can be treated very quickly by copious flushing and application of a wet to dry dressing. Remember that these dressings do not have to be perfect – do them quickly; you can always redo them once the pet is more stable. Open fractures can often be treated in a similar way.

Uroabdomen can often be stabilized by placement of an indwelling urinary catheter to keep the bladder empty. The urine can be drained from the abdomen using a standard needle or over the needle catheter attached to a collection system. If available, temporary peritoneal dialysis catheters are extremely effective at providing drainage of the abdominal cavity.

The clinical signs associated with diaphragmatic hernia can sometimes be alleviated by holding the patient upright, allowing the abdominal organs to fall back through the diaphragm into the abdomen, this is useful for a few minutes in order to get these patients into surgery, it is not a long term fix.

A flail chest can sometimes be stabilized by laying the animal with the flail side downwards; this prevents some of the aberrant movement of the flail segment.

Distal limb fractures or instability can be managed by standard splinting techniques, temporary casting or a modified Robert Jones bandage. A humeral fracture or elbow luxation can be stabilized with a spica splint. Fractures of the femur are not amenable to external coaption so sedation and good analgesic techniques should be employed until definitive repair is possible.

Antimicrobial treatment

Animals with wounds which penetrate body cavities or those with extensive skin wounds benefit from early antibiotics. For wounds affecting skin only a cephalosporin is often appropriate such as cefazolin. For animals with any kind of bite wounds or those with penetration into body cavities, especially if gastrointestinal compromise is suspected, then broader spectrum antimicrobials such as potentiated pencillins would be recommended.

Conclusions

When you are faced with an animal with severe trauma, remember that their status can change rapidly. You need to think about how to get them through the next 30 minutes as well as what treatments are in their long and medium term future to give owners realistic ideas about outcome and cost. The major considerations when faced with the emergent trauma patient include:

- How to resuscitate from shock?
- Prioritize the injuries – which are immediately life threatening, which can wait?
- Does the pet need emergent surgical intervention?
- What can be done to temporarily stabilize the patient before definitive interventions are possible?

Bedside Workup of the Dyspneic Cat

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Respiratory distress is a common reason for cats to present to a veterinarian particularly in the emergency setting. Cats with moderate to severe respiratory distress are often challenging to handle as they will rarely tolerate significant restraint and stressing them by performing extensive diagnostics, or sometimes even a full physical examination can significantly worsen their condition.

A range of diseases can cause respiratory distress in cats and an understanding of what they are and how they commonly present helps the clinician to narrow down the range of possible diagnoses and to start appropriate empiric therapy often prior to a definitive diagnosis.

As in other species, respiratory distress in cats can result from disease in a number of anatomic compartments:

1) The upper airway (particularly the larynx)
2) The lower airways
3) The lung parenchyma
4) The pleural space
5) The heart
6) Extra-thoracic causes

Common causes of respiratory distress by anatomic compartment:

Upper Airway:

- Laryngeal neoplasia (lymphoma, squamous cell carcinoma)
- Inflammatory laryngeal disease
- Laryngeal paralysis (usually secondary to another disease in cats)
- Trauma
- Nasopharyngeal polyp (young cats)
- Foreign body

Lower Airway:

- Feline Lower Airway Disease (asthma)

Lung Parenchyma:

- Pulmonary edema
    - Cardiogenic, non-cardiogenic
- Neoplasia
- Lungworm
- Contusions
- Inflammatory disease
- Infectious
    - Mycoplasma, typical bacterial pneumonia (rare in cats)

Pleural Space:

- Pleural effusion
    - CHF, fluid overload, chylothorax, pyothorax, hemothorax, FIP, neoplasia
- Pneumothorax
    - Traumatic, spontaneous (in cats often due to end-stage FLAD)
- Diaphragmatic hernia

Heart:

- CHF leading to pleural effusion or cardiogenic edema
- DCM or end-stage "burnt-out" HCM leading to poor cardiac output
- Severe arrhythmia
- PPDH
- Pericardial effusion

Extra-thoracic causes (rare):

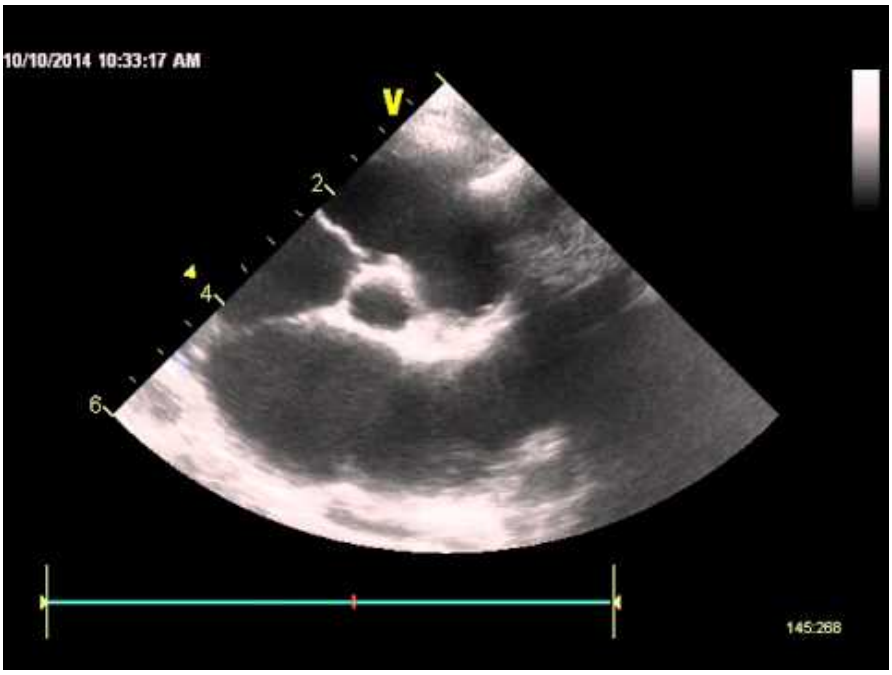
- Uncontrolled hyperthyroidism
- Pain

One of the challenges of managing respiratory distress in cats is that they are inherently unstable - excessive handling, for example for detailed physical examination, radiographs or catheter placement can result in significant clinical deterioration and even death in severely affected patients. This is made worse if the cat is fractious or unused to being handled. In situations where the cat is stable with only mild clinical signs then full diagnostic workup can be performed prior to treatment, in some situations, however, limited diagnostics must be performed and empiric treatment started prior to definitive diagnosis.

Emergency diagnostics (in a cat with moderate to severe respiratory distress):

Physical examination: Observe the cat, note the breathing pattern – is the dyspnea primarily on expiration, indicative of asthma or inspiration (everything else). Is a cough present? In a young cat, presence of cough is highly suggestive of asthma, in an older cat other differentials such as neoplasia must be considered. Is there marked upper airway noise or stridor during inspiration? Any signs of external trauma – scuffed nails, lacerations or abrasions? Auscult the chest – is a cardiac murmur or arrhythmia present? Although presence of a cardiac murmur is not specific for congestive heart failure, in a cat with respiratory distress you would likely be tempted to treat with a diuretic prior to further diagnostics. Do you hear crackles or wheezes? Crackles are suggestive of parenchymal disease or asthma, wheezes typically accompany asthma. Absence of lung sounds is highly suggestive of pleural space disease such as pleural effusion. Rectal temperature can be an important clue from your physical examination, cats with CHF almost always have a slightly low rectal temperature, cats with a normal to high rectal temperature are unlikely to have CHF – this is important as it can help you to rule in or out one of your most common differentials.

Bedside Tests: Ultrasound is very helpful for checking for presence or absence of pleural effusion, pericardial effusion. If pleural effusion is present on ultrasound or highly suspected based on physical examination then thoracocentesis can be both diagnostic and rapidly therapeutic. In more experienced hands, ultrasound can also be used to assess left atrial size and myocardial function to investigate the possibility of CHF. A cross sectional "four chamber view" showing large LA compared to aorta (>2:1) is very suspicious of significant volume overload.



NT-pro-BNP is emerging as a very useful bedside test in cats. If it is normal it is extremely unlikely to that CHF is the cause of the respiratory distress. If it is abnormal then CHF is much more likely. Note that extreme azotemia can occasionally cause false positives so if the cat does not respond appropriately to therapy then other differentials should be considered.

Examples of positive NT-proBNP test:

Thoracocentesis

Thoracocentesis can be both diagnostic and therapeutic in the event of a significant pleural effusion. Some basic testing of the fluid (protein content, glucose level, triglycerides and cytology) can be very helpful in determining a diagnosis and therefore appropriate next steps.

Pleural effusion can come about by one of three pathophysiologic mechanisms; increased hydrostatic pressure, decreased lymphatic drainage or trauma. Typically, we divide the disease into 3 based on the type of effusion present: transudate, modified transudate or exudate (including haemorrhage). This narrows down the differentials considerably and so step one in working up of pleural effusion is always to get a sample and figure out what kind of fluid it is.

Pure transudate: unusual finding. Usually seen in animals with hypoproteinemia

Modified transudate: common, differentials include congestive heart failure, neoplasia, chylothorax and lung lobe torsion.

Exudate: differentials include neoplasia, haemorrhage and infection

Once the type of fluid is established then further testing – cytology, cultures are needed to narrow down the differential list.

Specific diseases:

Congestive heart failure (MT): In cats, pleural effusion can occur secondary to hypertrophic, restrictive or dilated cardiomyopathy. Treatment involves draining of the pleural effusion followed and treatment of the underlying condition (diuretics, other cardiac medications).

Neoplasia (MT/Ex): In cats, neoplastic pleural effusion is most commonly secondary to a anterior mediastinal mass often lymphoma – this is especially common in cats positive for FeLV. Systemic chemo can be used for lymphoma, sometimes intracavitatory chemo can be effective for control of the effusion associated with mesothelioma, carcinoma. Small volumes of malignant effusion can sometimes be resolved with pleurodesis.

Chylothorax (MT): can be due to neoplasia, heart failure, trauma or be idiopathic. Neoplasia and heart failure causing chylothorax must be managed according to the underlying disease. Sometimes traumatic chylothorax (due to damage to the thoracic duct) will resolve given time and intermittent thoracocentesis is sufficient to control the clinical signs. Idiopathic chylothorax can be challenging to manage. Surgery is usually recommended – usually a combination of thoracic duct ligation or ablation and pericardectomy. The success rate of surgery is around 60%. Other options include intermittent thoracocentesis (but require regular taps, often every couple of days and can get severe cachexia – these patients tend not to survive beyond a few months if surgery does not help. A low fat diet can reduce the rate of build up of chyle. Diagnosis of chylothorax is confirmed by testing the triglyceride level of the effusion.

Lung lobe torsion (MT) – can be confirmed with radiographs "bubbly" appearance to lobe or computed tomography scanning. Surgery is often curative although some animals continue to effuse.

FIP (Ex): grave prognosis, often supportive care only – intermittent thoracocentesis.

Pyothorax (Ex): Cats can often be managed medically with appropriate antibiotic cover, placement of thoracostomy tubes and flushing of the chest. Those cats who do not respond quickly to medical management likely require thoracotomy. Some clinicians advocate early thoracotomy for pyothorax in cats and feel it improves time to resolution of disease.

Hemothorax (Ex – blood): rare in cats, usually due to trauma or neoplasia. Cats rarely eat anti-coagulant rodenticide although that would be a differential in rare cases.

Full diagnostics (in a cat with mild respiratory distress or following initial stabilization):

Almost always start with a thoracic radiograph. The rest of your workup is going to depend on this finding. The lung pattern, cardiac size and presence or absence of pleural effusion will dictate

where you go next. Routine bloodwork (CBC, Chemistry profile are usually performed at the same time)

Upper airway workup:

- Sedated oral/laryngeal exam exam (be prepared for a crisis!)
- Retroflexed endoscopy
- Advanced imaging (CT)

Cardiac workup:

- EKG
- Echocardiogram
- Pleural fluid analysis

Lower airway disease:

- Albuterol trial
- Heartworm test (in endemic areas)
- Fecal testing
- BAL (optional)

Pleural effusion – see above for thoracocentesis

- Fluid analysis
    - Inflammatory/not inflammatory
    - Protein and cell count
        - Exudate (high cell count/high protein) think pyothorax or cancer
        - Modified transudate think CHF or cancer
        - Transudate (very rare) – low albumin? Idiopathic
- Differentiate between cardiac and non-cardiac
- If neoplastic effusion consider staging

Emergency management of respiratory distress:

Oxygen supplementation is crucial in the dyspnoic cat – consider oxygen cage as this will allow administration of oxygen the need for handling or restraint. Otherwise providing oxygen by mask can also be helpful but some cats will not tolerate it.

If there is reasonable suspicion of cardiac disease then consider an empirical dose of furosemide – it is rare that this will cause a problem and it may cause enough improvement to allow for further diagnostics.

Try and assess for pleural effusion early – thoracocentesis will rapidly relieve respiratory distress in these cats and allow you to proceed to further diagnostics. Treating a cat with pleural effusion from CHF with furosemide will rarely help in the emergency setting.

If suspicious of asthma then treatment with an albuterol inhaler or terbutaline injectable can allow for stabilization of the patient for further diagnostics.

Longer term treatment of the condition is going to rely on definitive diagnosis of the disease.

INHALATION THERAPY: SOUNDS COOL, BUT IS IT FOR MY PATIENTS

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We've all used nebulizers on patients, but what are we really doing? Can we put medications in that noisy little machine? If so, what would we use it for? Believe it or not, you use inhalation therapy all the time (Oxygen! Isoflurane! Sevoflurane!), but we can probably do more. It turns out there are some interesting nontraditional applications for inhaled medications- and some that you might want to employ in your practice!

Inhaled Drug Delivery (Size Matters)

For the discussion of aerosolized medications, particle size is everything. Larger particles (>10 μm) are deposited by impaction in the oropharynx where they may be absorbed or swallowed. Particles 1-5 μm are deposited by sedimentation in the large airways or near an airway bifurcation. Depending on their bioavailability, these particles may either be absorbed through the walls of the airways and into the bronchiolar blood supply or they are carried up the muciloary tree and swallowed. Smaller particles (< 1μm) can diffuse out into the periphery of the lung by diffusion and random adhesion. Particles that make it to the alveolus can act locally, can be absorbed through the thin alveolar membrane into the pulmonary blood supply, or they can be removed by macrophages and end up in the lymphatics.

The moral of the story is that aerosolized drug is deposited all along the respiratory tree, not just the peripheral lung. Since our patients require the use of a mask or spacer to receive inhaled medications, contamination of the face and eyes with drug also occurs. Many medications in their liquid/injectable form are irritating to the oral mucosa and respiratory tract either because of their low pH or high osmolarity. In order to avoid patient discomfort, irritation, bronchospasm, and cough, clinicians should be mindful of drug characteristics and deposition while choosing a treatment plan. Hyperosmolar, acidic, or irritating medications should be significantly diluted or avoided.

Aerosol Technologies

There are three types of devices used to deliver aerosol medications to patients: metered dose inhalers (MDI), dry powder inhalers (DPI), and nebulizers[1]. An albuterol inhaler is a good example of an MDI. MDIs administer a prescribed dose of medication from a pressurized cannister through a valve. The vapor pressure and velocity of the propellant and the valve orifice size determines the droplet size created and can be adjusted depending on the density of the drug. These are reliable, predictable devices, but their "blast" delivery method is off-putting to veterinary patients, requiring the use of pediatric or veterinary spacers. Active inhalation during activation (cannister compression) of an MDI can deliver up to 50% of a drug into the lung, but veterinary patients with discordant breathing and using a spacer achieve roughly 5% delivery to the oropharynx and less than 5% deposition in the lung[2]. Common spacers used in veterinary patients are the AeroKat or the AeroChamber with a neonatal mask. This doesn't mean that MDIs aren't

valuable (we use them all the time), but it does explain why a cat needs two puffs of an albuterol inhaler, just like a person does.

Dry powder inhalers use a powdered lactose molecule to disperse dry drug into the respiratory tract on inspiration. The most common DPIs are the "disk" inhalers for long-term corticosteroids. Unfortunately, the use of DPIs requires that a patient actively inhale through a device for use. Since our patients won't inhale on command, DPIs don't currently have a role in veterinary medicine.

Nebulizers come in two varieties: the well-known atomizer jet (compressor) nebulizer, and the ultrasonic (mesh) nebulizer. Most clinicians have used a nebulizer, though most haven't put medications in them. When giving medications, all nebulizers require the use of a tight-fitting facemask to prevent environmental drug loss and maximize drug inhalation. Studies in children find that even small facemask leaks drastically drop nebulizer efficacy,[1] and that is likely true in our patients as well. Atomizer jet nebulizers force compressed air (8 -10 L/min) through a nozzle into the reservoir to aerosolize the saline or drug solution. Jet/compressor nebulizers are the most commonly used nebulizers in veterinary medicine. They allow versatility of the drug and dose prescribed, though the time required to administer a prescribed dose of medication can be considerable. Newer ultrasonic nebulizers use a vibrating fenestrated plate to produce droplets as the drug passes through the mesh. Ultrasonic nebulizers may offer faster administration and quieter operation compared to atomizer jet nebulizers, but studies in veterinary patients are currently lacking. Overall, nebulizers likely allow the greatest deposition of drug to the canine lung, compared to other aerosol technologies, but that percentage is still less than 5%[2]. Don't forget that nebulizers deposit as much as 50% of aerosolized drug into the oropharynx and GI tract of veterinary patients, which may actually be beneficial if that's your target.

Known Medication Options

β_2 agonists: Albuterol inhalers are probably the non-anesthetic inhaled medication veterinarians are most familiar with. β_2 agonists are used to relax bronchospasm in patients with small airway occlusion or induce intracellular potassium shift in hyperkalemic patients. It's important to note that patients with severe small airway disease are intolerant of facemasks and likely have poor airflow in the distal lung, making inhaled therapy inferior to systemic β_2 agonist administration. Albuterol inhalers (90 μg, 2 puffs into spacer q12h) are often prescribed to cats for rescue treatment of asthma.

Long-acting corticosteroids: Beclomethasone (QVAR 80 μg – 2 puffs into spacer q12h) and fluticasone (Flovent® 220 μg- 2 puffs into spacer q12h) are options for the treatment of lower airway disease in both dogs and cats. Unlike cats, dogs don't typically have traditional "asthmatic" dyspnea with air trapping. They display lower airway disease in the form of a chronic cough and increased airway sensitivity. Daily use of an inhaled long-acting corticosteroid may decrease systemic steroid side effects when compared to an oral corticosteroid. Some veterinary patients with lower airway disease can be managed solely with inhaled medications. Alternative therapies for lower airway disease (e.g. leukotriene inhibitors) are limited by the popularity of dry powder inhalers, which handicap them in the veterinary market.

Mucolytics: Saline is a very effective mucodilutant, and it is blissfully easy and inexpensive. A 10 to 15-minute nebulization of saline will humidify the entire respiratory tree, dilute secretions, and

allow improved mucociliary clearance. Human physicians will sometimes use nebulized 3% hypertonic saline in pediatric patients to help reduce airway swelling and break up secretions, but this has not been adequately evaluated in dogs or cats. The use of aerosolized N-acetylcysteine (Mucomyst®) has largely fallen out of favor in veterinary medicine due to the ongoing debate over its lack of improved outcomes and possible contribution to airway inflammation and bronchoconstriction[3].

Antibiotics: The use of inhaled antibiotics goes in and out of vogue, and while we all have opinions, nobody 100% knows the right answer. There is evidence that antibiotic aerosols may have a role in chronic, antibiotic resistant, and *Bordetella bronchiseptica* pneumonias. The main problem with using aerosol monotherapy is the difficulty determining how much drug is actually delivered to the airways. With the low deposition rates described above, it's currently impossible to predict the correct dose and administration interval that will provide adequate antimicrobial control without risking resistance in uncomplicated pneumonias. *B. bronchiseptica* is found in high concentrations in respiratory secretions and directly inhibits the action of the mucociliary elevator. There are a few abstracts and publications that show that aerosolized gentamicin may reduce the duration of illness, decrease clinical dyspnea, and may reduce bacterial load in airway secretions. You won't find inhaled antibiotics in the recently released antimicrobial use guidelines for treatment of respiratory tract disease in dogs and cats[4], and the author agrees that this is an adjunctive therapy at best.

Lesser Known Options:

Opioids: Opioids are very effectively administered by the aerosol route, but (like other inhaled medications) effective dose delivered can be quite variable. A lot of the drug administered can be lost to the environment or within masks/spacers, so high doses may be required to achieve adequate analgesia. It is hypothesized that inhaled opioids may act directly on mu receptors in the lung to diminish the need for systemic analgesia in the treatment of dyspnea. Medications such as fentanyl, hydromorphone, and morphine have been evaluated for systemic pain management and the relief of dyspnea and found that the evidence is either mixed or strong for the use of these medications[5]. Given the scarcity of opioid medications and the uncertain efficacy of this route, this is an interesting, but not proven method to provide analgesia. I would recommend this for the treatment of dyspnea or in hospice situations only.

Furosemide: Furosemide is also successfully used as an aerosol for the relief of dyspnea, but not because of its diuretic properties. Aerosolized furosemide may reduce bronchoconstriction, inhibit the cough reflex, and may act on small airway receptors to diminish the sensation of dyspnea without diminishing respiratory drive or altering the breathing pattern. It's thought that this effect may be mediated by the drug's inhibition of the Na-K-2Cl cotransporter and the local accumulation of sodium within the lung[6]. The dose of drug that should be nebulized and inhaled is not known, but it stands to reason that doses in excess of therapeutic intravenous doses would be appropriate. The author has used this therapy in conjunction with injectable opioids with some success in the alleviation of suffering of dyspneic patients where advanced respiratory intervention was not an option.

Lidocaine: While there is no safety data on the use of nebulized lidocaine in veterinary patients, it has been used as a steroid-sparing option for the treatment of human asthma. One study found that cats with experimentally induced asthma that received 2% lidocaine (2mg/kg) rather than placebo had improved airway flow but unchanged levels of airway inflammation[7]. Considering its potential for toxicity in cats and lack of superiority to traditional therapy, it's hard to recommend the routine use of lidocaine in asthmatic patients, but it is an interesting option to consider.

Epinephrine: You may remember that epinephrine is one of the drugs that can be administered in high doses via the airway during CPR. It is also used in pediatric medicine as a local vasoconstrictor to decrease edema in both the large and small airways. There is a case report that used both phenylephrine and epinephrine to reduce airway swelling of a patient with upper airway occlusion after surgical correction of brachycephalic obstructive airway syndrome[8]. It is thought that epinephrine induces vasoconstriction by α adrenoreceptor activation and induces bronchiole relaxation through β stimulation. Of note, Primatene Mist™ (aerosol epinephrine) has recently come back on the market and is available without a prescription for the management of human asthma. In humans, this medication is used to alleviate the clinical signs of asthma (bronchospasm), but it should not be used as a substitute for β agonists and (inhaled/oral) corticosteroid therapy. It's role in the management of veterinary lower airway disease is unknown, but your clients may ask about it.

Heparin: Aerosolized heparin can prevent bronchoconstriction and may play a role in smoke inhalation induced acute lung injury. It's unknown if heparin (a glycosaminoglycan) could decrease pulmonary inflammation or if it decreases fibrin formation and subsequent airway obstruction. Studies in human patients receiving nebulized heparin show improvement in mortality and lung injury scores without increased risk of hemorrhage. No dosing recommendations have been made in veterinary medicine, but a recent study was unable to document systemic anticoagulation (prolongation of aPTT) with as much as 200,000IU of nebulized racemic heparin administered to dogs weighing 20-30kg[9].

Does inhalation therapy have a place in your practice? It probably already does, but there are few resources for clinicians outside of asthma management. What guidance I can provide is that the risk of causing harm with aerosolized medication is probably low so long as the medication you are administering is isotonic and has a neutral pH. A small percentage of the dose you prescribe will actually make it to the lower airways and lungs, and the vast majority will end up on the face, in the oropharynx, and GI tract. It is not possible to predict efficacy of inhaled drugs, but they may provide a valuable adjunct to more established therapeutic options.

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Cardiopulmonary Resuscitation (CPR)

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CPR in Veterinary Medicine

Studies evaluating the efficacy of CPR in veterinary medicine are few and mostly retrospective in nature. The 2012 RECOVER guidelines incorporated these data with human studies to create recommendations aimed at improving survival in our patients. **Currently, survival to discharge following cardiopulmonary arrest (CPA) in veterinary species is <6% if not associated with anesthesia; survival for anesthetic-related CPA approaches 50%.** Even in the human field, survival is reported at approximately 20% for these cases.

One of the largest reasons for the discrepancy in the underlying cause for CPA. In humans, the leading cause of death is ventricular fibrillation secondary to a myocardial infarction. In veterinary patients, multi-organ dysfunction typically results in high vagal tone leading to arrest.

Pathophysiology of Arrest

Given the dismal survival rates, patient at risk for arrest should be monitored closely and intervention should occur before CPA. Hypoxemia is one of the most common etiologies resulting in arrest, accounting for 38% of cases in one study. Other causes including anemia, arrhythmias, cerebral trauma, and anaphylaxis. The common signs associated with arrest were agonal breathing and apnea (38% and 32%, respectively), following by collapse in 21% of patients.

Arrest can occur in several different ways. Respiratory arrest carries a better prognosis (28% survival in dogs and 58% in cats), as the heart continues beating for short period of time following to development of apnea. Patient require intubation and ventilation while the underlying cause for the arrest is investigated and corrected. Cardiopulmonary arrest is more common encountered and involves cessation of cardiac contractions.

Electrical activity of the heart varies in CPA. **The most commonly encountered rhythm is asystole (~70% patients)**, where there is no electrical or mechanical activity of the heart. **Pulseless electrical activity (PEA) occurs when there is still electrical activity in the heart, but these impulses do not stimulate contractions**. Complexes generated can appear as sinus beats or escape rhythms. This is seen in approximately 20% of cases. **Ventricular fibrillation occurs rarely in veterinary medicine(<10% patients) and is usually seen in association with CPR, cardiac disease, or anesthesia.**

Within 20 seconds of arrest, electrical activity within the brain is compromised due to lack of oxygen for high energy metabolism. Five minutes after CPA, there is complete depletion of adenosine triphosphate stores within the body. Finally, after 30 minutes of arrest, irreversible histologic changes within cells are identified.

The stages of ventricular fibrillation are well defined in humans and give important information to determine what intervention is necessary. The electrical phase (phase I) occurs within the first 4 minutes. There is little to no ischemic damage to myocardial cells at this time. Consequently, this phase is when defibrillation is most successful. The circulatory phase (phase 2) occurs for the next 6 minutes. During this time, reversible ischemic damage occurs within the cardiac myocytes. **CPR is recommended during this phase to provide oxygen prior to defibrillation**. Patients that are defibrillated during this phase without a round of CPR suffer

increased ischemic damage and are likely to fibrillate again, even if cardioversion is successful initially. The metabolic phase (phase 3) describes fibrillation that has occurred for greater than 10 minutes. There is irreversible ischemic damage to the myocardium and defibrillation is typically unsuccessful.

Basic Life Support

The fundamentals of CPR focus on improving perfusion to the heart and brain, while identifying and addressing the underlying cause of the arrest. A large support staff is required to accomplish this goal: **leader, compressors, ventilator, drug administrator, recorder/timer, and a someone to obtain a history (for out-of-hospital arrests).**

While many pieces of equipment are available in the veterinary clinic, an **electrocardiogram (ECG) and end-tidal carbon dioxide (EtCO2) monitor** are to two that provide the most valuable information during an arrest and should be attached to the patients immediately. The ECG is essential for rhythm diagnosis between compressors and helps determine the next therapeutic course of action. **For non-shockable rhythms (asystole and PEA) epinephrine and compressions are indicated; for shockable rhythms, defibrillation and compressions are indicated**. The EtCO2 monitor can be used to estimate the efficacy of resuscitation, as a value of **15 mmHg is considered to correlate to effective compressions**. If an EtCO2 of 15 mmHg is not achieved, efficiency of compressions should be evaluated: proper rate, proper depth, proper compression point, proper posture, etc. **While not all patients will achieve an EtCO2 of >15 mmHg during CPR (which conveys a poor prognosis for recovery), every attempt should be made to ensure that this low value is not from inadequate compressions**. Additionally, patients that achieve return of spontaneous circulation (ROSC) will have a drastic increase in the EtCO2 produced indicating successful resuscitation.

Compression should be performed at a **rate of 100-120 compressions per minute**. The chest should be compressed **1/3 to 1/2 of the total width, allowing for complete recoil**. Incomplete compressions or rapid compressions will decrease ventricular filling and decrease cardiac output. Studies have documented that efficacy of compressions decrease **after 2 minutes regardless of perceived fatigue**. It is recommended that compressors be rotated every 2 minutes (1 round), regardless. Optimal compressions generate approximately 20% of normal cardiac output when performed appropriately.

In small dogs and cats, compressions are performed directly over the heart, in line with the cardiac pump theory. Using this method, the ventricles of the heart are compressed mimicking a contraction. For **larger breed dogs**, direct compression of the heart is more difficult. In these patients, **compressions are performed at the highest point of the chest**. This increases intrathoracic pressure and causes the great vessels (vena cava and aorta) to collapse. Recoil causes these vessels to expand again and triggers the movement of blood. This is known as the thoracic pump theory and the heart acts as a conduit for blood instead of the mechanism of movement. While most dogs and cats should be placed in a lateral recumbency**, barrel-chested dogs (ie: bulldogs) should be placed in dorsal recumbency for compressions**. In general, any dog that can lay flat on his back should have compressions performed in dorsal recumbency.

Veterinary patients can have various upper airway disorders that can results in arrest (ie: elongated soft palate in bulldogs, laryngeal paralysis in Labrador retrievers). A quick upper airway examination may give evidence for the underlying reason for arrest. Once intubated, the patient should receive **one breath every 10 seconds with an inspiratory time of 1 second and a pressure of 10-15 cmH2O**. Hyperventilation will maintain positive intrathoracic pressures

which will compress the major vessel and impede venous return. Hypoventilation will allow for the accumulation of carbon dioxide, resulting in cerebral vasodilation, increased intracranial pressure, and decreased cerebral perfusion.

Advanced Life Support

Advanced life support incorporates the administration of medications to improve resuscitation. The foundation medications include epinephrine, atropine, and vasopressin.

Epinephrine is a non-selective sympathomimetic, resulting in alpha and beta adrenergic stimulation. This results in increased systemic vascular resistance, heart rate and force of contraction. Myocardial oxygen demand will be increased with increasing doses. **Despite the effects on the heart, the primary purpose of administering epinephrine is to increase systemic vascular resistance, shunting blood back to the core organs.** Lose dose epinephrine (0.01 mg/kg or 0.1mL/10kg of 1:1000) is recommended for the first two administrations every other cycle. High dose epinephrine (0.1 mg/kg or 1mL/10kg of 1:1000) is recommended after low dose has failed. **Higher doses will achieve a higher ROSC, but is also associated with higher rates of neurologic impairment and mortality. In cases of tachyarrhythmias requiring defibrillation (pulseless ventricular tachycardia and ventricular fibrillation) administration of epinephrine is not immediately recommended, except in cases of prolonged arrest (CPR > 10 minutes).**

Vasopressin acts on V1 receptors in peripheral vasculature to increase systemic vascular resistance without increasing myocardial oxygen demand. Several studies in human and veterinary species have found this medication non-superior to epinephrine. The dose is 0.8 U/kg with or in place of epinephrine. Vasopressin is extremely expensive (~$140/vial), limiting its usage.

Atropine is a parasympatholytic that decreases vagal tone. Both human and veterinary studies have demonstrated no clear benefit or detriment at the recommended dose of 0.04 mg/kg. This medication is administered every other cycle, opposite of epinephrine/vasopressin (ie: first 2 minutes, a patient receives a low dose of epinephrine, then the next two minutes the patient receives a dose of atropine). Higher doses (0.4 mg/kg) are associated with a decreased ROSC.

In out-of-hospital arrests, obtaining venous access can be difficult, but necessary for success. While a catheter is being placed, patients can receive epinephrine, atropine, vasopressin, lidocaine, and naloxone via the endotracheal tube. The doses administered should be doubled and given through a red rubber catheter down the endotracheal tube to get as close to the carina as possible and improve absorption.

Defibrillation is indicated for ventricular fibrillation and pulseless ventricular tachycardia only; asystole and pulseless electrical activity do not respond to defibrillation. This is due to the fact that defibrillation attempts to depolarize all the cardiac myocytes simultaneously, allowing for the functional syncytium to return. The initial dose is 2-4 J/kg (20J/10kg) and administered following a round of compressions in an unwitnessed arrest (since it is unknown how long the fibrillation has occurred for). If the initial defibrillation is unsuccessful, another round of CPR is performed and the dose is increased by 50%. In the event that ventricular fibrillation occurs during CPR, compressions should be continued until the defibrillator is charged and deliver during the next subsequent switch between compressors. The patient should be rotated into dorsal recumbency (if compressions are not already being performed in this position). Paddles should be covered in electrode transducer gel (ultrasound gel will not have the same degree of conductance) and placed on adjacent, lateral thoracic walls

directly over the heart. **No one should be in direct contact with the table or patient after the paddles are in place;** the patient in maintained in dorsal recumbency by the operator holding the paddles against the chest wall. The operator should call "CLEAR!" and all participants should answer with "CLEAR!" Following discharge of the defibrillator another round of compressions is performed and rhythm analysis is performed at the next at the following compressor change. **In cases of shockable rhythms, epinephrine is typically not administered; instead defibrillation is performed every cycle that a shockable rhythm is diagnosed until the patient converts to a perfusing or non-shockable rhythm.**

Additional therapies have been discussed for CPR, but none have demonstrated any efficacy, but may carry side effects. Anti-arrhythmics are recommended only in cases of prolonged arrhythmias; however, some of these medications make the patient refractory to defibrillation. Corticosteroid are not recommended unless specifically indicated for the underlying disease process (ie: hypoadrenocorticism) due to high rates of side effects. Bicarbonate is recommended only in cases of prolonged CPR. IV fluids should be administered only to hypovolemic patients, as they will increase right atrial pressure in normo- and hypervolemic patients, decreasing venous return.

Open Chest CPR

External compressions rely on normal thoracic cavity anatomy; any disease involving the chest can ultimately blunt the efficacy of external compressions for this reason. Open chest CPR is indicated in cases of pleural space or pericardial disease, thoracic trauma, giant breed dogs, concurrent surgery, and prolong CPR (>10 minutes). Internal compressions are associated with an increased ROSC and decreased neurologic impairments; however, there is significant morbidity and cost following this procedure with no change in survival to discharge.

The patient is placed in lateral recumbency (preferably left, but this is not always possible during CPR). The 6th intercostal space is quickly clipped and prepped while external compressions continue. Sterile gloves should be worn. Ventilation is transiently stopped and an incision is made along the cranial aspect of the rib. Once the chest cavity is entered, ventilation resumes and internal massage is initiated. In cases of pericardial effusion, the pericardium should be torn to allow the fluid to evacuate. Rib retractors can be placed after cardiac massage as started. Massage should be performed with the flats of the hands from apex to base of the heart. Internal massagers should be rotated every 2 minutes with each new person using new sterile gloves.

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Respiratory Disease in Puppies and Kittens

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Puppies and kittens often experience different respiratory disease to that seen in their adult counterparts. Additionally, their small size can present challenges with diagnostic imaging, certain treatments including establishing vascular access and even limitations on the physical examination.

Young animals may be more prone to infectious disease, especially around stressful times such as the period around being newly adopted into a family home or being shipped long distances either in a rescue or commercial setting. Additionally, much of the time the breeding conditions may be unknown or less than ideal, this can further increase the risk of infectious disease especially if the dam's vaccination status is unknown.

Additionally, because of their small size puppies and kittens are prone to trauma. This can involve head trauma, crush injury and falls all of which can have adverse impacts on the respiratory system.

Not withstanding their propensity to experience infectious disease or trauma, it is often during the transition from liquid to solid foods immediately prior to and during the adoption phase that congenital abnormalities such as PRAA or congenital megaesophagus can become clinically apparent.

Common Infectious Diseases of Puppies and Kittens: affecting the respiratory system:

Kittens:

The wet form of Feline Infectious Peritonitis (FIP) is relatively common in kittens and can result in pleural or pericardial effusions. Diagnosis is based on clinical signs, signalment and thoracocentesis yielding a thick, yellow protinaceous exudate. Supporting diagnostics can include bloodwork – often a high globulin level is seen in affected kittens. PCR to detect the modified coronavirus in the fluid can also be a helpful supportive test. Coronavirus titers on blood are non-specific and generally unreliable. Newer tests including IFA testing on tissue or fluid samples are on the horizon.

Infectious pneumonia in kittens is extremely rare - occasionally, viral infections which commonly cause upper respiratory tract signs such as herpes virus and calici virus can cause a viral pneumonia in kittens, although the extremely pathogenic calcici virus pneumonia is actually more common in adult cats. Kittens with pneumonia will almost always have concurrent upper respiratory signs consistent with URI. Diagnosis is by radiography and respiratory PCR swab.

B. bronchiseptica has been reported as a cause of pneumonia in kittens. While rare, it typically causes severe cough, fever and a marked broncho-intersitial pattern on radiographs. Diagnosis Is by PCR or culture of the organism from a broncho-alveolar lavage or endo-tracheal wash.

Lungworm caused by A abstrusus or C aerophila is a rare but present cause of lung disease in young cats. Radiographic appearance can range from a bronchial pattern through to severe alveolar pattern depending on the severity of disease. Diagnosis is by direct airway sampling or Baermann fecal testing.

Puppies:

The most common lung disease seen in puppies is bacterial pneumonia. This is often an extension of the "kennel-cough complex" which can include primary infection with B bronchiseptica. It will often begin as an upper respiratory infection, however, unlike adult dogs, puppies will almost invariably contract pneumonia. Other infectious agents can include pasteurella, streptococcus and pseudomonas. Additionally, bacterial pneumonia can occasionally be caused in this age group from aspiration – this is especially true if the the neonate is being artificially fed by bottle or tube or if they have a congenital disorder such as megaesophagus, PRRA or other condition causing an increase in risk of regurgitation. Diagnosis of bacterial pneumonia is typically by thoracic radiography and clinical signs. Confirmation or further investigation of specific etiological agent including culture and sensitivity can be achieved through endo-tracheal wash, broncho-alveolar lavage and subsequent cytology and culture.

Common viral infections include adenovirus, parainfluenza and distemper. These may affect multiple puppies within a litter and are best diagnosed by PCR testing. Some of these viruses such as distemper have other classic clinical signs associated with them such as tremors and hyperplasia and hardening of the paw pads.

Common Traumatic Injuries of Puppies and Kittens affecting the respiratory system:

Puppies and kittens are often dropped, accidentally stepped on or trapped in furniture such as recliners or folding chairs. This can cause direct injury to the thorax resulting in pulmonary contusions, pneumothorax and occasionally even diaphragmatic hernia. These are typically diagnosed based on history, exam and radiographic imaging. Treatment is usually supportive.

Head trauma and certain other insults such as seizures, electric shock etc can affect the respiratory system by causing the development of non cardiogenic edema. This is a peracute onset edema typically initially localized to the dorso-caudal lung field, although in severe cases it can spread throughout given time. Treatment is usually supportive and many animals will recover within 2-3 days if they do not develop secondary lung injury. Treatment with diuretics has been reported but is controversial.

Common Congenital Disorders affecting the respiratory system:

Some diseases such as persistent right aortic arch or congenital megaesophagus affect the respiratory system due to their increasing the likelihood of regurgitation and subsequent aspiration pneumonia. These are usually diagnosed based on clinical signs and radiographs.

Ciliary dyskinesia can present as severe or recurrent pneumonia without an obvious underlying cause. This disease causes absent or inco-ordinated ciliary clearance of mucous. Diagnosis can

be by examination of the sperm of intact male dogs, or demonstration of several sites showing ultrastructural lesions in the cilia on biopsy. This is a rare disease.

Congenital diaphragmatic hernia can usually be diagnosed on radiographs, it can be difficult to differentiate historical traumatic events from true congenital disease, however surgical correction of both is warranted.

Cardiac congenital abnormalities can lead to congestive heart failure and pulmonary infiltrates. Treatment with furosemide to stabilize the heart failure followed by echocardiography and definitive treatment of the defect if possible gives the animal the best chance at long term survival.

Workup and Diagnostic Testing:

Thoracic Radiographs:

Bacterial Pneumonia typically has the appearance of an alveolar pattern which is distributed in the cranial and ventral regions. Air bronchograms are present.

Non cardiogenic pulmonary edema is usually a focal soft tissue opacity/alveolar pattern present in the dorso-caudal aspect of the lung field on the lateral view, the infiltrates can be seen usually bilaterally in the caudal lung field on the VD view.

Trauma/contusions demonstrates a patchy infiltrate present throughout lung fields, often worse on one side than the other depending on side of impact. Other signs of trauma such as external injury or rib fractures may be visible on the images. Other injuries such as diaphragmatic hernia may be present.

Congestive Heart Failure in dogs, is often accompanied by cardiomegaly, peri-hilar interstitial or alveolar infiltrate and enlargement of the pulmonary vasculature. In cats, it may appear as an interstitial to alveolar patchy infiltrate with a more widespread distribution.

Cardiomegally and signs of CHF may be seen in diseases such as PDA. PRRA can appear radiographically as a dilated esophagus which acutely tapers around the heart base – confirmation and differentiation from congenital megaesophagus can be with contrast study or endoscopy prior to surgical correction.

Infectious disease testing:

Most bacteria can be grown with conventional culture and sensitivity techniques. Remember that some pneumonias can have an anaerobic component so testing both aerobic and anerobic cultures can be helpful. Mycoplasma spp often require specialized culture medium.

PCR testing can be extremely helpful in testing for viral etiologies of respiratory disease – PCR testing is extremely sensitive and accuracy is influenced greatly by the quality of the assay. Reputable veterinary laboratories should be utilized to avoid false positives and negatives.

Treatment Plans:

Antimicrobials are a powerful part of the armory when used appropriately. It is critical to think of the answers to a few questions before reaching for an antibiotic. As we have discussed it is often challenging to differentiate those diseases which are purely bacterial from others such as viral disease.

A series of questions to ask when considering using an antibiotic:

- How suspicious are you of bacterial infection?
- Where is the most likely location for the infection?
- What are the likely pathogens in that site?
- History of antibiotic use?
- Community versus hospital acquired infection?
- How SICK is this patient?

The mainstay of therapy for infectious bacterial pneumonia is APPROPRIATE and EARLY antibiotic therapy. The choice of antibiotic depends on what kind of infection is to be treated, what is the likely pathogen, is a resistant organism likely and how sick is the patient that you are treating. When treating critically ill patients with sepsis, it is just as important to think "How well does this plan fail" as "How likely is this plan to work?" It is important to have a good sense of what to choose for empiric antimicrobial therapy as cultures take 3 – 5 days to come back – by this time your critically ill patient will likely be already recovered or beyond saving.

Risk Factors for Resistance:

- Prior antibiotic use (within the last 90 days)
- Hospital acquired infection
- Long standing infection
- Surgical implants

Example – different presentations of puppy pneumonia **these are just options not a definitive list**

- Healthy, outpatient* these are just options not a definitive list
    - Likely pathogen *Bordetella bronchiseptica*
    - Most things will work = penicillins, doxycycline etc
- Sick, outpatient
    - Likely mixed infection with *Bordetella* as a component
    - Potentiated penicillin
- Sick, inpatient
    - Likely mixed infection with *Bordetella* as a component
    - Potentiated penicillin +- fluroquinolone
- Sick, inpatient, previous txt with antibiotics
    - Likely mixed population with high risk of resistance
    - Penicillins or potentiated penicillins for Gram +ve combined with aminoglycoside for Gram –ve cover and for its synergistic effects

- Chloramphenicol

It is especially challenging to decide when to use antimicrobials in cats with signs of upper respiratory infection. While there are no clear answers, the current guidelines on anti-microbial use recommend avoiding antimicrobials when the cat has clear or serous discharge and minimal systemic signs especially if it has been less than 10 days duration. Consider antimicrobial use in cases where the signs last longer than 10 days, the discharge is mucopurulent or the cat is showing other systemic signs such as extreme lethargy or anorexia.

In all cases of respiratory disease, supportive care is important. Oxygen supplementation by oxygen cage or nasal cannula can be life saving if hypoxemia is present.

Coupage and nebulization can be helpful in cases of infectious pneumonia, it should be avoided in cases of trauma to avoid worsening contusions and causing further discomfort.

Fluids can be both helpful and dangerous in lung diseases. As a rule, puppies and kittens should be kept adequately but not over-hydrated. This allows for secretions to be moistened allowing for expectoration in cases of pneumonia. Giving fluids also allows for supplementation of dextrose which can be critical in puppies and kittens who are anorexic to prevent hypoglycemia. It is important though, to know that fluids can worsen some lung disease such as cardiogenic and non cardiogenic edema. Fluid therapy is absolutely contraindicated in cases of cardiogenic edema and should be extremely conservative (or avoided) in cases of non cardiogenic edema. For these reasons, empiric fluid therapy should be avoided before a diagnosis is made and when it is performed, should be performed conservatively and with care. Note that normal fluid requirements of puppies and kittens are slightly higher than adult dogs and cats being around 3-3.5ml/kg/hr.

Prognosis:

- Most conditions are potentially reversible, even relatively severe pneumonia or pulmonary contusions.

- Congenital conditions may require ongoing medical management or surgical correction and generally carry a more guarded prognosis, these can include PRRA or congenital megaesophagus. Exceptions to this rule include some surgically or interventionally treated cardiac abnormalities such as persistent ductus arteriosis which can have a good prognosis if appropriately managed before severe congestive heart failure develops.

- Brachycephalic puppies with pneumonia actually have a similar outcome to non brachycephalic animals, however their risk of recurrence can be higher and they may have co-morbidities.

- Animals requiring mechanical ventilation have a much more guarded prognosis than animals who can be managed with medication and supplemental oxygen alone.

Fluid Therapy in Cardiopulmonary Disease

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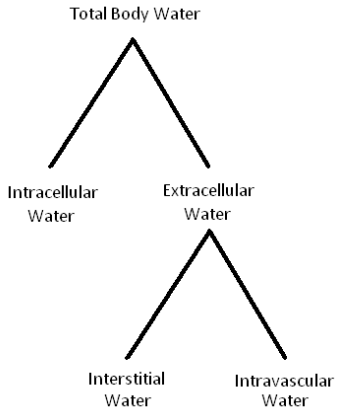
Fluid therapy is often the mainstay of treatment for many diseases encountered in veterinary medicine. Most animals who are admitted to the hospital for non-routine procedures or admissions will be put in intravenous fluids, and many may receive subcutaneous fluids on an outpatient basis. This lecture aims to review the times when fluid therapy can potentially be harmful and to discuss ways that we can mitigate the harmful effects of fluids while still treating our underlying disease.

The body is made, in large part, of water; and it is the proper distribution of that water that in part allows for normal cellular functions. Total body water (TBW) is approximately 60% of body mass. This water is compartmentalized into the intracellular (IC) and extracellular (EC) fluid compartments. The EC fluid is further subdivided into the intravascular (IV) and interstitial (IS) compartments (Figure 1). Of the TBW, roughly two-thirds is contained within the IC space. Of the EC fluid, about a quarter of it is maintained in the vascular space as plasma, with the remainder making up the IS space. How much water is "held" within each compartment is determined by the protein and electrolyte content of each space. Cell membranes are freely permeable to water, but the movement of solutes are tightly regulated.



Water loss from the various compartments will manifest differently. In general, we do not have "access" to the water content of the IC space – we cannot measure or estimate it directly, except with inferences based on laboratory tests (see later). However, we can gain insight into the water content of the IS and IV spaces. Loss of water from the IS space we call dehydration; loss of water from the IV space we call hypovolemia. Dehydration does not necessitate hypovolemia, and vice versa. That is Dehydration ≠ Hypovolemia. A patient may be dehydrated without a significant loss of blood volume and a patient may have lost IV volume without being dehydrated. However, they may be present concurrently. A dog that is hit by a car and lacerates a large blood vessel will be hypovolemic, but (unless it was unlucky enough to be sick prior to the trauma) is unlikely to be dehydrated. Likewise, if a cat is deprived of water for many days, it will become dehydrated, but, because the body preserves blood volume (and therefore blood pressure) at the expense of interstitial water, that cat will not be hypovolemic until the dehydration becomes very severe – generally around 8-10% or greater.

This means that we are generally giving fluids for either HYPOVOLEMIA (such as a trauma patient) or DEHYDRATION (as seen in a vomiting animal). This impacts how quickly we need to restore normal balance – ideally, dehydration is corrected with 12-24 hours. Hypovolemia can be life threatening and should be corrected as quickly as possible**.**

Fluid Types:

Crystalloids – Crystalloids can be generally defined as aqueous solutions (usually of mineral salts) that can freely pass through a semipermeable membrane. Crystalloids can be classified in a variety of ways – by their tonicity, whether they are balanced or unbalanced, or whether they are replacement or maintenance crystalloids.

Fluid tonicity refers to the effective osmolality of the fluid. An effective osmole is a solute that cannot freely pass through a semipermeable membrane and as such, it acts to "hold" water on one side of the membrane. Examples include sodium and chloride. Ineffective osmoles can freely pass across a semipermeable membrane (e.g., blood urea nitrogen).

Fluids may be isotonic (having a similar tonicity as extracellular fluid), hypotonic, or hypertonic. Normal fluid tonicity in dogs and cats is around 300 mOsm/kg and most isotonic fluids fall between about 270 mOsm/kg and 350 mOsm/kg.

Hypertonic fluids are sometimes utilized for their ability to hold or even draw water from the IS space and into the IV space. Hypertonic saline (usually between 5-7%) causes IV volume expansion well beyond the actual volume delivered, although it will rapidly redistribute. It should not be used in patients who are dehydrated or who have severe hyper- or hyponatremia.

Hypotonic fluids are sometimes used (especially as maintenance fluids – see below), but should never be bolused or given at high flow rates and generally should have a tonicity above ~100

mOsm/kg. Sterile water (with a tonicity of 0 mOsm/kg) should never be given intravenously. It will cause a rapid shifting of water intracellularly, causing red blood cell swelling and death.

Whether a fluid is considered balanced or unbalanced refers to the electrolyte composition of the fluid and whether it approximates EC fluid. The most important determinant of this is the chloride concentration, which is generally lower in balanced solutions.

Maintenance fluids are used to replace the daily obligate losses of water and solutes in patients that are not eating and drinking. Most of this fluid loss occurs via the kidneys (sensible fluid loss) and is quite low is sodium, but relatively higher in potassium (when compared with extracellular fluid). Additional losses (the insensible losses) occur via water and solute lost from feces, respiration, and sweat. This is generally about 1/3 of the maintenance water lost and is also relatively low in electrolytes. For this reason, maintenance fluids contain low sodium concentrations (~40 mEq/L) and relatively higher potassium concentrations. Because sodium is the largest contributor to tonicity, maintenance fluids are often hypotonic. Some commercially available fluids have dextrose added to them to increase tonicity (the dextrose is not a significant source of calories – it is a misconception that dextrose is added for any nutritional value). Because they are low in sodium and frequently hypotonic, maintenance fluids should not be bolused and generally should not be given at more than twice maintenance rates

Replacement fluids are used to replace a fluid deficit from the IV or IS spaces, as with hypovolemia or dehydration, respectively. This is why the composition of replacement fluids is similar to EC fluid – that is, high in sodium and chloride, and low in potassium. Replacement fluids may be given at maintenance rates (as with dehydration) or bolused (as with shock or hypovolemia). Often in practice, clinician will use a replacement fluid for maintenance fluid therapy. For most patients, this does not have much clinical significance, as the kidneys will simply excrete the extra sodium. In patients with renal or cardiac disease, however, the extra salt load may have serious consequences, therefore it is important to have at least one maintenance fluid available for cases in which the higher sodium content of replacement fluids is contraindicated.

Colloids – Colloids are fluids that contain large, insoluble molecules that do not freely cross a semipermeable membrane. Colloids act to "hold" water within the vascular space for a longer duration than crystalloids. For this reason they have been suggested for use in resuscitation, although this is controversial. Colloids generally are considered natural or synthetic. The natural colloids include plasma, fresh whole blood, and – to a lesser extent – packed red blood cells. The natural colloids are generally not used for their colloidal properties, but rather for their coagulation factors (as with plasma) or the hemoglobin content (as with whole blood or packed red cells).

The synthetic colloids usually have a much higher colloid osmotic pressure (COP) than plasma, which is around 20 mmHg. They have been shown to maintain intravascular volume for longer than crystalloids (although to a lesser magnitude) and they can increase COP in patients with hypoalbuminemia. There are advantages and disadvantages to using colloids in place of

crystalloids for resuscitation, although they have not been proven superior in prospective, randomized trials in people. Additionally, many of them have potential adverse side effects that must be considered when choosing whether to use a colloid, which colloid to choose, and what dose to use.

In general for this topic, we will say if there is any concern over cardiovascular or lung function then COLLOIDS should be avoided at all costs, and CRYSTALLOIDS used instead.

As a general guide fluid therapy in animals with cardiac or lung disease should be CONSERVATIVE and goal directed. It is most important in these patients to know when to stop so frequent patient re-assessment is needed. Once the patient is normovolemic and drinking on its own, discontinuation of IV fluids may be the best options. Subcutaneous fluids carry the same if not higher risk of causing volume overload in at risk patients as do IV fluids. In general, a low rate of IV fluids in patients where fluid therapy is considered risky is better than a bolus of SQ fluids.

Common scenarios include animals with heart disease who appear to require fluids. How well an animal will tolerate fluids will depend on the type of cardiac disease that is present. In an animal who is known to be in congestive heart failure, use of any kind of fluid at any dose is contraindicated, even if the animal appears interstitially dehydrated. While this is often an easy call, there are many others which are more borderline.

Dogs with a mitral murmur who have not been in heart failure before are most likely to tolerate fluid therapy. In general they are hyperdynamic with respect to cardiac output and so can cope with some extra pre-load. Again, start with a conservative rate of fluids and lower or discontinue the fluids as soon as the patient appears normally hydrated again.

Dogs who have historically been in heart failure but are markedly dehydrated, often because of furosemide use or because of development of vomiting, diarrhoea during treatment are at higher risk of developing heart failure when fluids are started. Again, dogs with mitral valve disease are more likely to be tolerant of a low rate of IV fluids than dogs with myocardial disease such as DCM. In these cases, starting at a maintenance rate to judge their response to a small amount of fluids may be prudent. Again, IV fluids should be discontinued as soon as possible. Another approach to these patients as long as they are not vomiting would be to place a naso-gastric or naso-esophageal tube and feed them water to rehydrate them. Discontinuation of furosemide is recommended during this time as giving both fluids and a diuretic is counter-productive. Some animals with mild-moderate dehydration in this category who are still showing some interest in food or water may respond well to simply skipping a couple of furosemide doses and allowing them to correct themselves by drinking. In general, cats tolerate fluids more poorly than dogs. A cat who has historically been in heart failure will likely go very quickly back into heart failure when fluids are administered – if IV fluids are definitely required, then they should be started at between half and full maintenance and discontinued as soon as possible.

Some clinicians advocate using hypotonic fluids such as 0.45% saline, believing that the lower sodium load is less likely to push them into failure, while this makes intrinsic sense, no evidence

to support this approach exists and it is certainly reasonable to use a isotonic balanced electrolyte solution such as LRS provided it is used cautiously.

Animals with lung disease can also pose a fluid challenge. In general, avoid fluids in the dyspnoic cat – very few diseases that cause dyspnea in cats will respond favourably to fluids and undiagnosed congestive heart failure is very common. The exceptions to this rule are very dehydrated cats with upper respiratory infections and cats with pyothorax who will need aggressive fluid therapy as part of the management of their sepsis.

Dogs with pneumonia generally need fluids and in some cases if they are severely septic may even present with hypovolemic and require fluid resuscitation. In severe cases of pneumonia, overly aggressive fluid resuscitation can result in increases in lung water and subsequent worsening of respiratory function. In these animals, titration of fluids to normal end points is critical – you want the animal to be normovolemic and normally hydrated. In these cases, we are not concerned about the effect of fluid therapy if we get them back to a normal state, we are concerned about avoiding OVERHYDRATION. Puppies have a higher resting water requirement and so puppies with pneumonia may require higher rates of IV fluids. Cats arrely get pneumonia and those who do require very conservative, careful fluid therapy.

Animals with pulmonary contusions from trauma can pose another challenge. These animals often have polytrauma and may be bleeding and suffering from hypovolemia, in these cases the hypovolemia is life threatening and must be treated. Low volume resuscitation with hypertonic saline (2-4ml/kg) is often employed initially in these cases. This is another scenario where “just enough” fluid resuscitation is the key. Overly aggressive resuscitation to above-normal end points can result in worsening bleeding and worsening pulmonary contusions and lung function. In the absence of brain injury, aiming for an initial systolic blood pressure between 90-100mmHg is probably adequate.

Animals with non-cardiogenic pulmonary edema can be easily worsened by fluid therapy. In general, no fluid is best. If the animal is young and needs dextrose supplementation then consider either oral supplementation or if this is not possible due to the degree of dyspnea then as small a volume of crystalloids as possible to dilute the dextrose should be used. These are usually cases which are acute in onset and typically start to resolve within 2-3 days so dehydration is minimal. Some clinicians will treat these animals with diuretics, however unless aggressive fluid therapy has been performed and the animal is overloaded, this is unlikely to be helpful.

Finally, dogs with interstitial lung disease such as pulmonary fibrosis can be worsened by fluid therapy. IV fluids should be given only if absolutely necessary. If their respiratory rate begins to worsen with fluids then discontinue and give 2-3mg/kg furosemide to see if that has an effect. Interstitial accumulation of fluid can result in marked respiratory impairment in these already very compromised dogs.

MANAGEMENT OF HEPATIC ENCEPHALOPATHY

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Definition

Hepatic encephalopathy was first described over 100 years ago in dogs with surgically created portocoaval shunts and was originally called “meat encephalopathy”. In humans, hepatic encephalopathy is defined as “the spectrum of neuropsychiatric abnormalities seen in patients with liver dysfunction after exclusion of other known brain disease.” Gow suggested a more relevant definition for use in companion animals: “neurological dysfunction as a result of hepatic disease and/or portosystemic shunting”.

Clinical significance

Although the true prevalence of HE in dogs is not known it is certainly is not an uncommon diagnosis. In a recent retrospective study, 68% of dogs undergoing surgical attenuation of a single congenital portosystemic shunt (CPSS) had preoperative neurological abnormalities. Hepatic encephalopathy is an important cause of morbidity in dogs and clinical signs can range in severity from mild manifestations, such as apathy and mental obtundation, to seizures, coma, and even death.

Clinical findings

The signs of HE are often initially subtle and intermittent but can progress in both frequency and intensity. Signs may become worse in the postprandial state. In a retrospective study of 118 dogs with HE the most frequently recorded historical clinical signs were lethargy (27%), altered behavior (26%), obtundation (25%), ataxia (24%), seizures (22%), head pressing (19%), ptyalism (19%), vomiting (18%), blindness (17%), circling (13%), shaking or twitching (12%), and anorexia or hyporexia (11%). At the time of hospital admission, abnormal neurologic findings were recorded for (47%) dogs, and the most frequently recorded clinical signs were obtundation (25%), ataxia (19%), paresis (8%), conscious proprioceptive deficits (7%), seizures (5%), stupor or coma (4%), circling (3%), abnormally delayed menace response (3%), and tremors (3%), as well as blindness, abnormally decreased pupillary light response, head pressing, ptyalism, head tilt, and anisocoria (2% each). However, it is important to note that some of these signs may have been due to concurrent disease.

Diagnosis

The diagnosis of HE in dogs is made based on the presence of consistent clinical signs, exclusion of other causes of encephalopathy, laboratory testing, and response to treatment. The signs of HE are discussed above. Other metabolic causes of encephalopathy such as hypoglycemia or uremia can easily be ruled out be performing a serum chemistry panel. Serum biochemistry can also be useful to determine if hepatic disease of CPSS is likely. Dogs with CPSS can have other changes such a decreased or low normal serum albumin and cholesterol concentrations. Dogs with chronic hepatitis often have increased liver enzyme activities.

Measurement of plasma ammonia concentration is sometimes performed to aid diagnosing HE. While ammonia concentration is correlated with the severity of HE in dogs it is a poor predictor of the severity of HE in individual patients. It is also possible for dogs with HE to have plasma ammonia concentrations within the reference interval. Proper sample handling is critical to avoid

falsely elevated ammonia measurements; samples must be collected on ice and analyzed within 30 minutes. This means ammonia must be measured in-house. Therefore, clinicians should not overly rely on ammonia measurement to diagnose this disease. Interestingly in humans HE is a diagnosis of exclusion and ammonia measurement is not considered to be necessary for diagnosis.
Documenting the presence of congenital or acquires portosystemic shunts can also be helpful to diagnose HE. Measurement of serum bile acids or ammonia can both be useful for this purpose but also have limitations. Diagnostic imaging can also be very helpful for this purpose. Computed tomography angiography has superseded abdominal ultrasound and portal scintigraphy as it allows excellent characterization of shunt anatomy.

Management

Whenever possible treat the underlying cause of HE, for example by attenuating a CPSS. Successful shunt attenuation results in an improvement of HE. However, persistent shunting can be associated with the persistent or recrudescence of HE as can the development of portal hypertension and subsequent formation of acquired portosystemic collateral vessels. Although dogs with CPSS can be successfully medically managed for prolonged periods of time their median survival is longer after surgery.
It is important to identify and address any potential precipitating factors such as hypokalemia or alkalosis (Figure 1). Intravenous fluid therapy can also be very helpful to correct any fluid deficits and prevent dehydration. Warm water enemas remove fecal contents and gastrointestinal hemorrhage and are given at a dose of 10 mL/kg every 4–6 hours until signs improve. Patients that are stuporous or comatosed may require intubation to protect their airway. If the patient has clinical signs consistent with cerebral edema (worsening forebrain deficits, and increased systemic blood pressure possibly with reflex bradycardia) mannitol may be indicated at a dose of 0.5-1 g/kg *IV*. Dogs suspected to have gastrointestinal ulceration should be treated with omeprazole and sucralfate.

Figure 1: potential precipitating factors for hepatic encephalopathy

Precipitating factor	Proposed mechanism of action
Sepsis	Inflammatory mediators have a synergistic effect with ammonia, increase blood brain barrier permeability, and lead to altered neurotransmission
Gastrointestinal hemorrhage	Increased protein load and ammoniagenesis
Constipation	Dehydration, electrolyte abnormalities, small intestinal dysbiosis, and bacterial translocation
Excess dietary protein	Increased ammoniagenesis
Dehydration	Electrolyte changes, increased renal ammoniagenesis
Drugs	Sedative agents (benzodiazepines, opioids) cause depression of cerebral function Diuretics (cause electrolyte imbalances, alkalosis and dehydration)
Hypokalemia	Leads to movement of intracellular potassium into the extracellular space, extracellular alkalosis, and trapping of ammonium ions within cells
Hyponatremia	Enhanced astrocyte swelling

Alkalosis	Increased access of ammonia to neurons (due to a shift in in the equilibrium from ammonium ions to ammonia, which can pass freely through cell membranes)
Uremia	Increased renal ammoniagenesis
Superimposed hepatic injury	Decreased hepatic conversion of ammonia to urea

When formulating a nutritional plan for any patient it is important to start be assessing their calorie needs and to ensure that these are met. Some dogs with HE first present when they are still growing and this also needs to be considered. Severe protein restriction is no longer recommended for dogs with HE as it can lead to protein malnutrition which can lead to a number of deleterious effects including loss of skeletal muscle mass and worsening of HE. Traditionally dogs with HE were protein restricted fed renal diets (12-15% protein on a dry matter basis). More recently diets marketed for use in dogs with hepatic disease have become available. These typically contain 14-18% protein on a dry matter basis. This still may not be sufficient protein for optimal long-term feeding, especially for growing animals. Non-meat protein-based sources, such as soy are sometimes recommended for dogs with HE as they are believed to be less encephalogenic than meat protein. It is reasonable to initially feed a dog with HE a commercial "hepatic" diet but once the signs of HE are controlled, it is recommended to add non-meat protein (e.g. tofu or cottage cheese) to the patient's diet to help prevent protein malnutrition. Multiple small meals should be fed in an attempt to reduce the postprandial ammonia load. Consultation with a veterinary nutritionist can be helpful.

Lactulose is a non-absorbable disaccharide that is believed to: trap ammonium ions within the colon leading to decreased absorption of ammonia into the portal circulation; (ii) inhibit ammonia production by colonic bacteria; (iii) stimulation of incorporation of ammonia within bacterial proteins; (iv) reduce intestinal transit times leading to decreased bacterial ammonia release; and (v) increase fecal excretion of nitrogenous compounds. Lactulose can be given orally to patients with chronic HE. It is usually started at a dose of 1 to 3 mL per *PO* per 10 kg of body weight every 6 to 8 hours. The dose is then adjusted until the patient passes three to four soft stools per day. Lactuolse can also be given as a retention enema in which case cleansing water enemas are given first. The dose above can then be used diluted to 30% administered and retained for 30 minutes.

In patients that fail to respond to nutritional management and lactulose or those that present acutely with severe HE, antibiotic treatment is indicated. Because of the potential of long-term antibiotic use to induce bacterial resistance ideally once the patient is stabilized these drugs should be discontinued. Neomycin is a poorly absorbed aminoglycoside antibiotic that is sometimes used to treat HE in dogs. The gastrointestinal absorption of neomycin is usually very low, but can be increased in some patients. Substantial systemic absorption can cause ototoxicity or nephrotoxicity. For this reason, I no longer use this drug to treat HE in dogs. Metronidazole is another antimicrobial that is sometimes used for the treatment of HE in dogs. Metronidazole is metabolized by the liver and can cause neurological signs. Therefore, it is usually given at a reduced dose of 7.5 mg/kg *PO* q8−12 hours in dogs with HE. Metronidazole is the antimicrobial I most commonly use to treat HE. Ampicillin, amoxicillin, or amoxicillin clavulanate have all been used to treat HE and aside from occasionally causing gastrointestinal side effects they are usually well tolerated.

DERMERGENCIES

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In most situations, when a dermatology case presents on emergency it is more of a convenience factor for the client (e.g. not able to get into see primary care veterinarian during normal business hours). However, there are occasions when a case of skin disease presents as a "derm-urgency", or even a true "dermergency". This presentation aims to discuss the process of identifying a dermatological emergency, how to effectively triage these cases, and considerations for specific disease entities that fall under this umbrella.

FACTORS THAT POINT TOWARDS A "DERMERGENCY"

There are a few key features that should alert the clinician that they may be dealing with a rather urgent or emergent dermatological condition. This includes:

- Acute development of moderate to severe pruritus or pain
- Rapid development of skin lesions
- Concurrent systemic abnormalities (fever, lethargy, gastrointestinal signs)
- Presence of large cutaneous defects

APPROACH TO THE DERMATOLOGICAL URGENCY/EMERGENCY

As with working up most patients with skin disease, the most important "tests" are a complete and thorough history and physical examination. This information will guide practitioners which differential diagnoses may be most likely to consider and will help shape a therapeutic plan. Special attention should be paid towards previous and current medications or therapeutics (including vaccinations, preventatives, supplements, and diet) as well as time frame of administration since adverse drug reactions can pose some of the more serious cutaneous presentations. Establishing the presence or absence of pruritus (or pain) should additionally be part of this history gathering exercise. Development and progression of clinical signs, including the appearance of skin lesions, is imperative information to obtain. Even though time is frequently limited on receiving in the ER, this step in the triage of dermatological emergencies is paramount towards likelihood of a successful diagnosis and therapeutic plan; even before examining an animal, I have a good idea what I may be dealing with strictly from the history provided.

Following acquisition of a complete history, a thorough physical examination is needed. Identification of concurrent systemic signs should be part of this, however much emphasis should be placed on the dermatological examination. Recognition of which lesions are present on the patient (papules, pustules, crusts, erosions, ulcers, etc.) as well as their location (Haired skin only? Non-haired skin? Mucous membranes?) and pattern of distribution (Symmetrical? Asymmetrical? Distal extremities?) will be the framework for crafting a list of differential diagnoses to pursue.

In many cases of true "dermergencies", biopsy is indicated to confirm a diagnosis. Ideally, samples should be taken prior to initiation of therapy, especially in the case of corticosteroids since this can influence the disease process and hence the ability of the pathologist to confirm etiology. Early lesions should take precedence over more chronic or traumatized lesions as these are typically most diagnostic. Collecting samples from various stages of development however can additionally be beneficial as the condition progresses. Submission to a dermatopathologist is recommended especially for less commonly identified disease entities (see additional information below). When infectious conditions are part of the differential diagnoses (i.e. deep pyoderma, systemic mycoses – these both tend to be a bit more chronic and progressive, however may have concurrent systemic manifestations lending to an emergency visit), tissue culture (e.g. from a biopsy sample) should be submitted as well as histopathology samples.

"DERM-URGENCIES"

Although the owner might perceive it as an emergency, more frequently a "derm-urgency" will present on ER. These are conditions that if left unchecked for periods of time can turn into more of an emergent situation, or can become much more severe. However, they are typically not life threatening to the patient. Some conditions that fall into this category include:

- **Urticaria and angioedema** – Often due to an acute hypersensitivity reaction. This may be triggered by vaccines, other drugs reactions, biting/stinging insects, or other unknown factors. Food on occasion can also be an underlying trigger. Urticaria and angioedema tends to toe the line between a "derm-urgency" and a "dermergency" as should clinical signs progress, a more anaphylactic presentation can occur (think: shock involvement – GI tract in dogs, respiratory system in cats). Y'all know what to do with these cases 😉 (antihistamines, steroids, supportive care as needed).
- **Acute otitis externa** – It's often the behavior of the animal that turns it into an "emergency" for the clients (constant head shaking/scratching that wakes them up in the middle of the night). Topical therapy with an otic medicant is typically needed to address secondary infections which are often present, however don't forget the benefit of systemic anti-inflammatory corticosteroids in these patients. NSAIDs, Apoquel, and Cytopoint generally not beneficial for otitis.
- **Pyotraumatic dermatitis** – AKA "acute moist dermatitis", or "hot spot". These surface infections if left unchecked can become deep pyoderma in a relatively short period of time (even within a few hours). Initially though, they only involve the surface layers of the skin. Party line for a "hot spot": clip it, clean it, dry it out. Added to that though, is identification of possible inflammatory triggers leading to "hot spot" development. The most common locations for these lesions to occur include the rump (trigger = flea allergy dermatitis) and along the neck/behind the ears (trigger = otitis externa; make sure to do ear cytology and treat any infections in these cases). As with acute otitis externa, the benefits of a short course of systemic corticosteroids cannot be further underscored.
- **Eosinophilic folliculitis and furunculosis of the face** – Lesions including erythema, papules, nodules, erosions and ulcerations are typically noted on the muzzle and face of the dog. Severe pruritus and/or pain is generally observed. This is typically triggered by envenomation from biting or stinging arthropods or snakes. Large numbers of eosinophils are evident on cytology. Secondary bacterial infection is not uncommon. Once again, corticosteroids are a best friend for this condition.
- **"Sick" pemphigus foliaceus (PF)** – Considered to be the most common autoimmune skin disease seen in companion animals, the lesion of PF is a pustule caused by separation of keratinocytes from each other due to an attack on adhesion molecules in the skin. These pustules are typically quite transient/fragile and more frequently patients will present with thick, mounded crusts. Both haired and non-haired skin are typically affected, however the oral cavity is spared. Some of these patients will present systemically ill (lethargy, fever, weight loss) lending to more of the "emergent" situation. Biopsy is indicated to diagnose this condition (collection of an intact pustule is ideal) with subsequent immunosuppressive therapy. Acantholytic keratinocytes, often in small groups or "rafts", are frequently noted on cytology with large numbers or neutrophils (and occasionally eosinophils) in the absence of infectious organisms (e.g. bacteria).

"DERMERGENCIES"

A handful of dermatological conditions present more of a true emergent situation. In these cases, severe cutaneous signs are often present that may also have concurrent severe systemic consequences. These cases often require additional support to combat development of sepsis, protein, and hydration losses that occur. Some specific conditions that fall into this category include:

- **Erythema multiforme (EM)** – Although the exact pathogenesis of the condition is unknown, this is a cutaneous reaction pattern of multifactorial etiology (e.g. drug administration, neoplasia, infections, connective tissue disease) in which a cellular immune response is directed against various keratinocyte-associated antigens. The disease has historically been subcategorized (based on a confusing and somewhat controversial system adopted from human medicine) dependent on percentage of skin affected. As the name implies, multiple forms of erythema may be observed clinically. Cases will present with an acute onset of erythematous, annular macules, elevated circular plaques, and papules in partially symmetric patterns. With progression, erosions and ulcers are additionally seen. Oral cavity and other mucous membranes will generally be affected by this disease. Severely affected animals may be febrile, depressed, anorectic, and painful. Biopsy will show individual cell apoptosis with lymphocyte satellitosis in multiple layers of the epidermis. Immunosuppression is indicated unless a neoplastic trigger is identified.
- **Toxic epidermal necrolysis (TEN)** – This is a rare condition in which a cell-mediated induction of keratinocyte death results in full thickness coagulative necrosis of the epidermis. There is controversy whether this is a more severe form of EM or whether these are separate conditions. Clinically, widespread macular erythema progresses to confluent erythema followed by severe ulceration over large portions of the body. Underlying triggers may be associated with drug administration or neoplasia. In this condition, much of the therapy revolves around supportive care for what the skin typically accomplishes (hydration, sepsis, theromoregulation). Prognosis is considered to be very poor.
- **Cutaneous vasculitis** – This is an inflammatory disease of blood vessels, typically secondary to immune complex deposition (type III hypersensitivity reaction) within vessel walls. This may be a primary (autoimmune) problem or secondary to an underlying trigger (various infectious etiologies, environmental triggers, drug reactions etc.). Lesions include purpura/ecchymoses, necrosis, and punctate ulcers which can increase in size which most commonly affect distal extremities. As lesions progress, crusts and scarring alopecia. Acute flares are best managed with aggressive anti-inflammatory/immunosuppressive therapy (provided an obvious infectious etiology is not present).
- **Cutaneous drug reactions** – Clinical signs can be myriad as drug reactions can mimic many other dermatopathies. Lesions may range from erythema, urticaria, angioedema, to papules and pustules, to erosions and ulcerations. History of ALL medications, therapeutics, supplements and diet is imperative when considering a cutaneous adverse drug reaction. Essentially, ANY medication or therapeutic can cause a cutaneous drug reaction, however antibiotics and NSAIDs have been implicated most commonly.
- **Wells-like Syndrome and Sweets-like Syndrome** – With both conditions, the etiology is unknown in companion animals. Underlying triggers may be possible, however are often uncertain. Lesions are similar with both conditions to include an acute onset of erythematous macules that progress and coalesce to form arciform or serpiginous plaques or wheals. Pitting lesions and even nodules may additionally be apparent. Abdomen, thorax, and face may be more commonly affected. In both cases, concurrent (or just before/after development of skin lesions) gastrointestinal signs (vomiting, diarrhea) are observed. On examination, erythematous mesions

typically do not blanche when pressure is applied. Biopsy shows superficial and deep perivascular dermatitis with marked dermal edema and vascular dilation. Differentiation between the two conditions depends on which inflammatory cell is primarily involved in the dermatitis; with Wells-like syndrome eosinophils will predominate whereas neutrophils will be most prominent with Sweets-like syndrome.

ADDITIONAL READING: Kersey KM, Rosales M, Roberts BK. "Dermatologic Emergencies: Identification and Treatment." *Compendium* 2013, E1-9.

Anemia in the ER

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Anemia is one of the most common clinical findings encountered in veterinary medicine; unfortunately, the identification of this problem typically raises more questions than it answers. This is because anemia is a symptom and not a diagnosis. Several different classification schemes of anemia exist: response by the body (regenerative versus non-regenerative) and etiology (loss, destruction/hemolysis, or lack of production). Rapid identification and classification of anemia can be immensely useful to the emergency clinician, allowing for quick therapeutic intervention and appropriate discussion with owners. This can be accomplished with five simple tests available in the emergency department: packed cell volume and total solids, serum color evaluation, saline agglutination test, blood smear, and a reticulocyte count.

Packed Cell Volume/Total Solids

Packed cell volume (PCV) refers to the percentage of blood composed of red blood cells RBCs (i.e.: in a patient with a PCV of 45%, if 1mL of blood is removed, 0.45mL will consist of RBCs). The PCV can be artifactually increased by insufficient spinning of a sample or decreased by dilution with other compounds, most commonly EDTA. While often used interchangeably, hematocrit (HCT) is a calculated value determined on most hematology analyzers. Agglutination can artificially lower HCT on analyzers.

PCV is the most commonly used means of diagnosing anemia, but only provides half of the story. Total solids (TS) can provide additional insight into the nature of the anemia, helping the clinician the differentiate between blood loss and lack of production or destruction. This is because TS is a more sensitive indicator for hemorrhage than PCV. The spleen acts as a large reservoir for RBCs, with contraction occurring in response to circulating catecholamines. Blood proteins are not stored within the spleen. As a result, hemorrhaging patients may have a low normal PCV, despite having lost a significant amount of blood volume. A patient in shock with a normal PCV and low TS should prompt the clinician to investigate locations of significant hemorrhage.

There are few locations within the body that can contain enough blood following loss to result in clinical signs of hemorrhagic shock. This include the thoracic cavity, abdominal cavity, the gastrointestinal tract, and the subcutaneous/interfascial space – and, of course, outside the body. Patients bleeding into their lungs will display signs of respiratory distress due to decreased oxygen exchange prior to shock from hemorrhage. Patients with pericardial effusion will show signs of shock from decreased venous return before signs of decreased oxygen delivery from anemia. The four internal sites and one external site for massive hemorrhage should be evaluated in any patient suspected of bleeding AND re-evaluated during the course of resuscitation for continued loss.

Table 1. Common Patterns for PCV/TS in Diseases

Decreased PCV/ Normal TS	**Increased PCV/ Normal TS**	**Normal PCV/ Decreased TS**	**Normal PCV/ Increased TS**	**Increased PCV/Increased TS**	**Decreased PCV/Decreased TS**

Hemolytic anemia	Polycythemia Vera	PLE	Multiple myeloma	Hemoconcentration	Chronic blood loss
Aplastic Anemia	Hyperthyroidism	PLN	FIP		Subacute blood loss
RBC Aplasia	Cushings	Liver failure	Chronic globulin production		
Anemia of Chronic Disease	Acute Hemorrhagic Diarrheal Syndrome	Acute blood loss (with splenic contraction)	Severe dehydration + anemia		
	EPO-secreting tumors	Third spacing	Lipemic serum		

Serum Color

Serum color is a simple, but oft overlooked diagnostic that can provide valuable information regarding the nature of the anemia. Large scale hemolysis, as seen with immune-mediated hemolytic anemia, will typically result in pigment changes. Serum color will change based on the location of the hemolysis.

Intravascular is predominantly complement mediated and occurs within the vessel, though physical damage (from vasculitis, splenic torsion, neoplasia, garlic/onion, zinc, etc.) can result in direct lysis as well. As a result, free hemoglobin is released into systemic circulation, coloring the serum red. This form of hemolysis has a rapid onset and is typically slow to respond to immunosuppressive medications, unless an underlying cause can be identified. In contrast, extravascular hemolysis will typically result in increased levels of bilirubin within serum, producing a yellow color. This is the result of antibody-mediated phagocytosis of the RBCs and occurs within the reticuloendothelial system of the spleen and liver. As the hemoglobin is broken down, it overwhelms the metabolic capacity of the liver, allowing unconjugated bilirubin into circulation. Spun microhematocrit serum should be evaluated against a plain white backdrop to allow for better visualization prior to determining total solids. Severely anemic patients with a hemolytic disorder are unlikely to have clear serum.

Saline Agglutination

Agglutination of RBCs is a common feature of immune-mediate hemolytic anemia (IMHA) and occurs as a result of antibody mediated aggregation of cells. It should be noted that agglutination is not the only feature, as several confounders of this test exist. Rouleaux can occur within samples, mimicking auto-agglutination. Patients who received a blood transfusion 5-7 days prior may have positive saline agglutination tests, without true IMHA. This may signify a delayed transfusion reaction, where the patient is mounting a response against donor cells, not their own.

A saline agglutination test is easy to perform: add one drop of 0.9% saline to 1 drop of EDTA whole blood. This volume of diluent is typically enough to disperse rouleaux, but not in all cases. If RBCs still aggregate macro- or microscopically, the cells should be washed in saline and the test repeated. Slides without macroscopic evidence for agglutination can also be examined for microscopic agglutination. Findings of a positive saline agglutination test should be taken in light

of other available diagnostics (i.e.: PCV/TS, serum color, etc.) as this test is not definitive for IMHA due to error from rouleaux.

Blood Smears

Hematology analyzer provide valuable information regarding cell counts, but blood smears are essential to aid in the diagnosis of etiologies for anemia and to complement automated read outs. RBC morphology, infectious disease agents, and platelet counts can all be determined from blood smear evaluation.

RBC morphology is an essential portion of blood film and can help provide support for diagnosis, especially in cases of anemia. Spherocytosis is a loss of central pallor within the RBC that occurs following incomplete antibody-mediated phagocytosis by monocytes. The presence of spherocytes is one of the diagnostic criteria for IMHA. They may also be identified in patients that have received a transfusion, as spherocytes will develop during blood storage. Schistocytes are another important morphology of RBCs. These are irregularly shaped cells that have lost the typical round shape of a healthy RBC. They occur as a result of vasculitis, fragmentation, and neoplasia (particularly hemangiosarcoma).

Certain blood-borne parasites can be identified on blood smear, most notably mycoplasma. Practitioners should take note to prepare fresh blood smears when evaluating for these organisms, as their attachment to the cell surface is mediated by calcium. Anticoagulants, such as EDTA, chelate calcium and may result in decreased organism adhesion to the membrane making cytologic diagnosis more difficult.

Platelets should always be evaluated in conjunction with RBCs on blood films. This is accomplished by counting the number of platelets per high powered field (100x magnification), determining the average, and multiplying this number 15,000. In patients with severely low platelet counts (<20,000), the thrombocytopenia may be the result of immune-mediated destruction. Any anemia accompanying these findings may be related to blood loss or, less likely, Evan's syndrome (IMHA with immune-mediated thrombocytopenia). The PCV/TS pattern can help to distinguish between IMHA and blood loss anemia. Platelet counts >60,000 are likely a result of consumption and may indicate bleeding.

Reticulocyte Count

As previously mentioned, anemia can also be classified as regenerative or non-regenerative based on reticulocyte counts. Reticulocytes are immature RBCs released from the bone marrow prematurely in response to peripheral need for oxygen carrying capacity. Their presence in circulation indicates normal physiologic process and appropriately functioning bone marrow. A regenerating canine has >80,000 reticulocytes, while felines have slightly less at >60,000.

Duration of anemia needs to be taken into account when evaluating reticulocytes counts. A proper regenerative response takes 3-5 days from onset of a moderate to sever anemia. Patients without a regenerative response may have had inadequate time or an inappropriate response from the bone marrow.

Non-regenerative anemias are most commonly encountered in acute blood loss, chronic inflammation, renal disease, toxins (estrogen), and dysplastic or neoplastic bone marrow disorders; very rarely the immune response may be directed at the bone marrow, as well. Regenerative disorders include hemorrhage, peripheral IMHA, toxins (garlic/onions, zinc), vasculitis, and neoplasia.

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Emergency Management of Arrhythmias

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When interpreting electrocardiograms, a methodical approach is necessary. If one can measure the heart rate, determine whether the rhythm is sinus origin or not, describe whether the rhythm is regular or irregular, and determine the origin of the impulses, most arrhythmias can be easily identified.

To determine average heart rate quickly, remember that the length of a standard Bic pen (with cap) is 150 mm, which is equivalent to a 6 second period of time when the paper speed is 25 mm/second. This means you can lay your pen on an ECG trace, count the number of complexes, and multiply by 10 to obtain the average heart rate. If the paper speed is 50 mm/second, the length of the pen will be equivalent to a 3 second period of time (thus you will need to multiply the number of complexes by 20). To obtain an instantaneous heart rate, count the number of small boxes (millimeters) between two QRS complexes. If the paper speed is 25 mm/second, divide the number of small boxes into 1500 (25 mm/sec x 60 sec = 1500 mm/min). If the paper speed is 50 mm/second, divide the number of small boxes into 3000 (50 mm/sec x 60 sec = 3000 mm/min).

Bradycardia is defined as < 60 bpm in the dog and <140 bpm in the cat. Tachycardia is defined as >160 bpm in the dog and >240 bpm in the cat.

Arrhythmia	Common Underlying Cause(s)	Treatment	Pearls
Sinus tachycardia	**Physiologic:** -Cardiac: CHF -Non-Cardiac: Shock, pain, anxiety, dehydration, fever, exercise, hyperthyroidism, drugs	-**Treat the underlying cause**. -Antiarrhythmic medications are not warranted.	In the dog, if HR persistently >220, probably not sinus tachycardia, if 250 bpm or more → not sinus tachycardia!
Ventricular tachycardia	**DOGS:** -Arrhythmogenic right ventricular cardiomyopathy (Boxers), Dilated cardiomyopathy, Myocarditis (Chagas disease)	**DOGS:** **Injectable:** -Lidocaine: 2 mg/kg bolus IV (can repeat 2-3 times), 25-80 mcg/kg/min CRI -Procainamide : 10-15 mg/kg IV bolus (over 2-5 min, can repeat once), 25-50 mcg/kg/min CRI **Oral :**	Not all ventricular arrhythmias require treatment. Always consider antiarrhythmics if you have documented ventricular tachycardia, see multiform ventricular arrhythmias, or if the patient is

	CATS: Uncommon -Hypertrophic cardiomyopathy, Hyperthyroidism	-Sotalol : 2 mg/kg PO BID -Mexilitine: 4-8 mg/kg PO TID), give with food **CATS:** **Injectable:** -Lidocaine: .25-.5 mg/kg bolus IV (cats very sensitive!) **Oral:** -Sotalol: 2-3 mg/kg PO BID -Atenolol: 6.25-12.5 mg PO BID	symptomatic or hypotensive. Rapid couplets may also prompt therapy. Don't forget to check electrolytes! Keep potassium mid-high end of normal range. Can also supplement Magnesium sulfate (30 mg/kg IV over 20 min) in dogs.
Supraventricular tachycardia	**DOGS:** -Atrial enlargement -Accessory pathways -Sometimes occurs in structurally normal hearts (primary or "lone" atrial fibrillation cases, some atrial flutter cases) **CATS: Uncommon** -Severe atrial enlargement -Hyperthyroidism	**DOGS:** **Injectable:** -Diltiazem: 0.1-0.2 mg/kg IV bolus, 2-6 mcg/kg/min CRI -Procainamide: 10-15 mg/kg IV bolus (over 2-5 min, can repeat once), 25-50 mcg/kg/min CRI **Oral:** -Diltiazem (Cardizem): 1-2 mg/kg PO TID -Diltiazem XR (Dilacor XR): 2-4 mg/kg PO BID -Digoxin: 0.005-0.008 mg/kg PO BID -Sotalol: 2 mg/kg PO BID **CATS:** **Injectable:** -Diltiazem: 0.1-0.2 mg/kg IV bolus, 2-6 mcg/kg/min CRI -Procainamide: 1-2 mg/kg IV bolus (over 2-5 min) 10-20 mcg/kg/min CRI **Oral:** -Sotalol: 2-3 mg/kg PO BID -Atenolol: 6.25-12.5 mg PO BID -Diltiazem XR (Dilacor XR): 15-30 mg PO SID-BID	Typically, SVT is not immediately life-threatening. Oral medications can usually be started with no injectable drugs needed. Atrial fibrillation hallmarks: -No P waves -Narrow QRS complexes -Irregular rhythm Rapid onset and rapid offset can help distinguish regular SVTs from sinus tachycardia. Heart rate control is the primary goal. Tachycardia- induced cardiomyopathy can develop with chronicity if HR not well managed.
Sinus bradycardia	-Elevated vagal tone (physiologic vs pathologic) -Drugs -Hypothermia -Hypothyroidism	Treat the underlying disease. No antiarrhythmic therapy is warranted.	Pathology in the CNS, pulmonary, or GI systems can pathologically elevate vagal tone, as can

			high extraocular pressure. Cats with CHF can present with sinus bradycardia due to hypothermia from poor cardiac output.
AV block	**1st degree:** -Vagal tone -Drugs -Av nodal disease **2nd degree:** -Vagal tone -Drugs -AV nodal disease **3rd degree:** -AV nodal disease -Fibrosis -Myocarditis -Neoplasia (rare)	**1st degree:** None **2nd degree:** May require nothing if mild and vagally-mediated. If symptomatic, withdraw any offending drugs and consider pacemaker if needed **3rd degree:** Pacemaker	Consider screening for myocarditis in cases of advanced AV block with a Chagas titer and/or an ultrasensitive cardiac troponin I (general cardiac biomarker indicative of cardiac damage).
Atrial standstill	**Severe underlying atrial disease** **Hyperkalemia**	Pacemaker Address hyperkalemia	
Sinus arrest	**Exaggerated sinus arrhythmia** (common with brachycephalics) **Sick sinus syndrome** (Miniature schnauzers, Cocker spaniels and West Highland White Terriers are commonly affected)	None **If asymptomatic**: None **If symptomatic (syncope, lethargy, exercise intolerance):** Pacemaker is ideal. Medical management: -Theophylline (10 mg/kg PO BID) -Terbutaline (0.2 mg/kg PO BID-TID) -Propantheline (0.25-0.5 mg/kg PO BID-TID)	Medical management of sick sinus syndrome is often unrewarding.

THE FATE OF FELINE AORTIC THROMBOEMBOLIC DISEASE

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We can all relate to the emotional dread we experience when we hear the unique yowl of the feline in severe distress. That yowl could easily be a urinary obstruction (UO), but it could just as easily be a feline aortic thromboembolism (FATE) or "saddle thrombus". While both UOs and FATEs can be professionally stimulating, FATE is less likely to result in a satisfying patient outcome. While there have been innovations in predicting and diagnosing this challenging condition, there have been few, if any, clear innovations in treating these cats and improving both short and long-term survival.

Abbreviations	
FATE	*Feline aortic thromboembolism*
UO	*Urinary obstruction*
TE	*Thromboembolism/thromboembolic*
AKI	*Acute kidney injury*
tPA	*Tissue plasminogen activator*
UFH	*Unfractionated heparin*
LMWH	*Low molecular weight heparin*

Risk Factors

No discussion of thromboembolic disease is complete without at least a passing mention of Virchow's triad: hypercoagulability, blood stasis, and endothelial dysfunction. In the feline patient, heart disease is the leading cause of thromboembolic disease, with clots suspected to originate within the left atrium or left auricle. Both blood stasis within a dilated cardiac chamber, endothelial injury of the endocardial surface, and increased platelet activation can occur in cats, completing Virchow's criteria for thromboembolism (TE). It's thought that intracardiac clots start out as activated platelets and mature to incorporate fibrin in layers. These layers are less stable, and portions of the original clot can break off, or the entire clot can migrate out into circulation. Sadly, there is no way to reliably assess the risk of TE disease, but the literature reveals that 12-17% of cats with cardiomyopathy will develop FATE. Males are at increased risk of FATE, which likely represents the increased occurrence of heart disease in male cats. Cats with FATE typically have a larger left atrium, larger end-systolic left ventricular diameter, and decreased fractional shortening[1]. There is also thought that the presence of a cardiac "gallop," spontaneous echogenic contrast (smoke), or a visible clot on echocardiography puts patients at increased risk of FATE[2]. Thromboprophylaxis is likely indicated in patients with cardiomyopathy displaying these characteristics.

Clinical Signs

While most feline TE disease manifests as FATE, it is also possible to have migration to a forelimb, kidney, brain, mesentery, or spleen. Forelimb TEs occur in approximately 10% of TE patients, with the right forelimb more commonly affected than the left because of the branching of the brachial arteries. Patients with forelimb TE can have subtle to severe sings, and patients that present for FATE may have a recent history of "forelimb lameness" that was actually a previous TE event. FATE can result in partial or complete occlusion of the aortic and collateral flow to one or both hindlimbs. Up to 75% of FATE cats are affected in both pelvic limbs. Affected limbs are often painful, display paresis or paralysis, absent segmental reflexes, muscle discomfort and contraction, diminished or absent arterial pulses, cyanotic nailbeds, and hypothermia. Severe cases of bilateral FATE can also result in a decreased rectal temperature due to decreased perfusion as a result of cardiac failure or TE arterial occlusion. Clinical signs

often develop acutely and can be concomitant with a new or breakthrough cardiac failure event. Most (90%) cats with FATE have underlying cardiac disease, but only 10% have been diagnosed prior to the FATE event[3]. It can be difficult to determine if respiratory effort in FATE patients is due to pain, stress, or cardiac disease.

Diagnosis

Diagnosis of a FATE event is often pretty straightforward, with an acute onset of pain, hindlimb paralysis or paresis, and the absence of arterial pulses. Patients with unilateral disease or incomplete vascular occlusion (weak but present limb pulses) can potentially pose a challenge. There are both simple and advanced diagnostic options available to clinicians. The most basic of which is to locate and measure the arterial pulse pressure (or lack thereof) in the affected limb with a Doppler crystal. A small blood sample obtained from an affected limb can be used to compare glucose and lactate measurements and compare to a central (jugular) blood sample. The glucose in an affected limb is expected to be significantly lower than peripheral, and lactate is expected to be significantly higher than peripheral blood. Ultrasonographic evaluation of the TE can reveal renal artery occlusion and can be used to serially evaluate blood flow around and through the occlusion. Advanced imaging options such as thrombus contrast-enhancing MRI are also potentially available, but rarely necessary or employed in veterinary medicine.

Blood testing performed on cats with FATE may reveal evidence of acute kidney injury (AKI), electrolyte derangement, and elevated indicators of muscle damage (CK, AST, & ALT). Hyperkalemia is not common in the acute phase of disease, but it can be severe and is a common sequalae of reperfusion injury and/or AKI when

Treatment

Currently, no “magic bullet” treatment has been described for FATE. Current recommendations are based on the principles of supportive care: pain management, preventing thrombus propagation (growth), managing underlying cardiomyopathy, preventing mutilation, and preventing TE recurrence. The ischemic injury that occurs in affected limbs is almost certainly painful and should be managed with pure mu agonist opioids initially. Cardiomyopathy and congestive disease should be managed as needed to maintain optimum cardiovascular stability and peripheral perfusion. Limiting thrombus propagation and prevention of recurrence are both accomplished with antiplatelet medications and/or anticoagulants. Physical clot removal has been reported by several modalities, but, but is not currently recommended. Treatment with “clot buster” thrombolytic medications (e.g. tPA- tissue plasminogen activator) has shown efficacy in breaking down occlusive TEs, but complication rates are high, outcomes are not improved, and survival is similar to conservative therapy[4]. It’s hard to know whether or not to recommend tPA for cats severely affected with FATE, as they have the gravest prognosis and potentially the most to gain from thrombolysis, but they also have the highest risk of sudden death, AKI, and life-threatening reperfusion injury.

The antiplatelet agents aspirin and clopidogrel (Plavix®) are the most-studied class of thromboprophylaxis in the treatment of FATE. Anticoagulant agents such as unfractionated heparin (UFH), low-molecular-weight heparin (LMWH), and rivaroxaban (Xarelto®) are also legitimate options for use in these patients. Monitoring of thromboprophylaxis is of limited value and can realistically only be performed with any timeliness in patients treated with UFH. For

patients *at risk* for TE disease, it is probably appropriate to select one avenue of thromboprophylaxis (e.g. clopidogrel), but patients with confirmed TE disease should likely be treated with multimodal the addition of a second anticoagulation agent (e.g. clopidogrel + LMWH or clopidogrel + rivaroxaban) for both treatment and indefinite thromboprophylaxis.

Aspirin- inhibits the production of thromboxane A_2 within platelets. Has a narrow therapeutic window in the cat and can be associated with GI side effects. Typical dosing is 81mg PO every 48 – 72h. In cats with a previous FATE event, recurrence of FATE occurs in17 - 75% of cats (64% within the first year), and median survival times vary from 117 – 192 days. While this option is inexpensive, it has recently fallen out of favor due to its inferior performance when compared with clopidogrel[5].

Clopidogrel (Plavix®) – irreversibly inhibits ADP on the platelet surface to reduce platelet reactivity, granule secretion, and fibrinogen binding. Clopidogrel requires hepatic transformation into an active metabolite via the cytochrome p450 system, which may affect the bioactivity of other medications. A loading dose of 75mg PO on initiation of treatment or a standard daily dose of 18.75mg PO q24h may be prescribed depending on clinician preference. In cats receiving clopidogrel after a previous TE event, 49% of cats had TE recurrence (36% within the first year), and they had a median survival time of 443 days. The recent expiration of the patent on this medication has made it an inexpensive treatment option.

Low molecular weight heparins – are short-chain heparin molecules that only inhibit factor Xa. They have a relatively low risk of bleeding complications, and monitoring is not currently recommended. Enoxaparin (Lovenox®) and dalteparin (Fragmin®) are both used in the treatment of FATE. Both of these medications are given subcutaneously and require dosing every 12 hours. Despite common usage, no data is currently available on the efficacy of LMWH in the treatment or prevention of FATE. Enoxaparin is administered as 1.0-1.5 mg/kg q12h and dalteparin is administered at 100-200 IU/kg every 12h. Both of these medications are quite expensive but potentially manageable due to the small amount administered to feline patients.

Rivaroxaban (Xarelto®)- factor Xa inhibitor available in an oral formulation. No published data on the use of rivaroxaban for FATE is currently available in cats, but studies are underway to evaluate the use of this medication as a sole-therapy for TE prevention in cats with previous TE events. Current evidence suggests that 2.5 – 5mg/kg q24h is well tolerated in cats. This is a relatively expensive medication that is reasonable for many clients because of the small dose required in feline patients.

Outcome

Overall survival for cats with FATE are reportedly between 27 – 45%. Hypothermia, loss of motor function, bilateral arterial occlusion, and bradycardia are all negative prognostic indicators. Cats with only one affected limb and/or intact motor function can do significantly better, with a 68 – 93% survival rate, while cats with bilateral occlusion are reported to survive only 15 – 36% of the time. Up to 40% of cats will die spontaneously despite treatment, and up to 35% of cats are euthanized during hospitalization. With these dismal numbers, it's easy to understand clinician bias and see how up to 90% of cats with FATE are euthanized shortly after

diagnosis. Patients are most likely to die or acutely worsen within the first 72 hours. Patients that survive for 72 hours are more likely to survive to discharge, which can have variable endpoints. Some patients will improve rapidly and regain motor function and blood flow in the affected limb(s). Other patients will require 6-8 weeks to maximize clot dissolution and recanalization. Regained limb function may remain absent or incomplete in some patients and predicting survival and outcome goals are impossible on presentation. Surviving cats may require physical therapy, supportive nursing care, lifelong thromboprophylaxis, and management of underlying cardiomyopathy. Even with thromboprophylaxis, cats with TE disease remain at lifelong risk of TE recurrence, which is often life-limiting. The uncertainties of treatment, cost, quality of life, and recurrence of TE disease are understandably significant obstacles for many owners and clinicians.

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ACUTE KIDNEY INJURY

Dr. Igor Yankin

Acute Kidney Injury encompasses a continuum of conditions from a subclinical kidney injury to a complete loss of function.

AKI Criteria

- Increase in serum creatinine of 0.3 mg/dl within 48 hours or >150% (1.5 times) from baseline within 7 days
- Urine output <0.5 ml/kg/hr for ≥6 hours
- Independent from etiology

AKI Classification schemes

- RIFLE (human)
- AKIN (human)
- KDIGO (human)
- VAKI (veterinary)
- IRIS AKI (veterinary)

Azotemia is not a prerequisite for a diagnosis of AKI. AKI is an independent factor of mortality in humans and animals including those with a non-azotemic stage of AKI.

The gold standard in assessment of renal function is a measurement of glomerular filtration rate (GFR). The GFR measurement is inconvenient, time consuming and laborious. Creatinine, BUN and SDMA are the examples of biochemical surrogates of GFR. A urine output can be used for assessment of renal function, however it lacks sensitivity and specificity.

Biomarkers are the measurable substances that indicate the presence of injury in a certain part of the nephron (e.g. renal tubules or glomeruli). As opposed to functional tests, they don't assess the kidney function. The major benefit of biomarkers is an early diagnosis of AKI.

The relationship between GFR and creatinine may be represented by the exponential curve that consists of the horizontal portion and the steep vertical portion. The reduction in GFR by 50% will double the creatinine level.

Drawbacks of serum creatinine

- Needs a steady state of production to be accurate
- Depends on muscle mass
- May be affected by a breed (mean sCr in healthy non-Greyhound breeds is 1 mg/dl vs. 1.6 mg/dl in Greyhounds)
- Affected by age, diet, critical illness, hydration status

Etiology of AKI in dogs and cats - a proportional meta-analysis of case series studies (*Legatti et al. PLOS 2018)*
Infectious causes – 30%

- *Leptospirosis 44%*
- *Pyelonephritis 6%*
- *Pyometra 38%*
- *Sepsis 3%*

Non-infectious – 60%

- *Nephrotoxins 30%*
- *Obstructive – 16%*
- *Unknown – 37%*

Diagnostic approach to AKI

Step #1 – Establish a diagnosis of AKI

- High index of suspicion is important
  - Any patient with unexplained anorexia, vomiting, or lethargy should be suspected to have an AKI
  - Any critically-ill patient
  - Any patient on therapy with nephrotoxic potential
- Majority of veterinary patients develop community-acquired AKI (as opposed to humans)

Step #2 – Rule out pre-renal and post-renal causes of AKI first

Post-renal (blockage and/or rupture)

- Palpate the bladder
- Rule out uroabdomen
- Rule out hydronephrosis and upper urinary tract obstruction

Pre-renal (volume-responsive AKI)

- Measure USG (ideally full UA) +/- FENa
- Correct dehydration, hypovolemia, low cardiac output state
- Reassess sCr within 48 hours
- Don't forget about Addison's disease

FENa (fractional excretion of sodium) = (Urine Na x SCr / Urine Cr x Serum Na) X 100%

FENa <1% - prerenal

FENa >1-2% - intrinsic

Diuretics and fluids may skew the results

Step #3 – Further investigate possible causes of intrinsic AKI

- 10-40% of intrinsic AKI cases may have an unknown cause (discuss it with the client)
- Collect a thorough history in regards to a possibility of exposure to nephrotoxins
  - Grapes/raisins, EG, Vitamin D rodenticides, mushrooms, lilies, NSAIDs, ACEi, heavy metals, methotrexate, various antibiotics
- Collect urine for culture (aerobic)
- Submit blood and/or urine for leptospirosis
- Perform full abdominal ultrasound if possible
- Perform FNAs of the kidneys if neoplasia is suspected

Diagnosis of leptospirosis

1. **Urine and/or blood PCR**
   - Highly specific
   - Low sensitivity during the late stage or after the treatment
2. **MAT**
   - Paired titers are required if the first test is inconclusive

3. Lepto WITNESS (JVIM Lizer et al. 2018)

- Detects IgM only (active infection; as early as 4-6 days)
- Should be repeated in 7-14 days if negative
- Outperformed SNAP Lepto
- Comparable with MAT

AKI Management: Create a perfect homeostatic environment for kidneys to heal

1. **Optimize oxygen delivery (correct dehydration/hypovolemia, hypoxemia, anemia)**
   - Correct dehydration/hypovolemia with balanced crystalloids
   - Avoid synthetic colloids
   - Avoid high-chloride containing solutions if possible
   - Potassium-containing balanced replacement solutions are not contraindicated in hyperkalemic patients
   - Reassess azotemia within 24-48 hours
   - Once euvolemia/euhydration achieved: decrease or discontinue fluid therapy (if oligoanuric or fluid-overloaded)

2. **Avoid fluid overload**
   - Monitor body weight every 6-8 hours
   - Maintain a zero balance once the patient is fluid-resuscitated and euhydrated
   - Pay attention to clinical signs of overhydration (peripheral edema, chemosis, cavitary effusions)
   - Discontinue fluid therapy if there is any evidence of overhydration or patient is anuric

3. **Correct electrolyte derangements**
4. **Treat arterial hypertension**
5. **Empirically treat pyelonephritis/leptospirosis with antibiotics (e.g. ampicillin/sulbactam) while waiting for labs**
6. **Provide nutrition (NG or esophagostomy tube)**
7. **Eliminate medications with nephrotoxic potential (likely for life)**

When do I refer an AKI case?

- Anytime you are uncomfortable to manage it
- Unable to provide 24 h care
- Oligoanuric AKI
- Fluid overload
- Severe electrolyte derangements
- Progressive azotemia despite conventional therapy
- Unable to rule out a ureteral obstruction

Indications for Renal Replacement Therapy

- Oligoanuria
- Fluid overload (especially in combination with oligoanuria)
- Severe hyperkalemia
- Severe metabolic acidosis due to AKI
- Progressive azotemia (sCr >7-8 mg/dl, BUN >100-150 mg/dl) refractory to conventional therapy

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