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MEDICINE

Hypothermia: Foe More Than Friend

IMMUNOLOGY

Understanding the Use of Antibody Titres in Veterinary Practice

CPD

Common Dilemmas in Epilepsy

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Editor's Note

Another few months, still in lockdown and a whole brave new world out there.

We adjust quickly though. When looking at programs regarding Chinese cities - instead of scoffing at the masks people used to wear against the pollution, I find myself examining the designs and wondering how comfortable they are!

We have an extension of last months' doodle regarding hypothermia written by Dr Justin Grace, an anesthesiology resident. Also, the first of a 2-part series on IMHA. I also found an interesting article on titre testing - something I initially didn't see sufficient scope for - but there definitely is a place for this in your practice. The AAHA vaccination guidelines also has a stepwise approach to 12 situations where it may be of value in your practice.

The CPD article is from Dr De Decker - who lectured us on neurological conditions at the last NVCG congress and also again on a SAVA webinar. I can really recommend all of his lectures. His approach is extremely practical and relevant. His message - get the basics right first before you throw money at expensive tests, this is my message as well. Attention to detail with the basics!

The imaging article has some valuable advice on diagnosing pneumothorax and the dental article shows you what options are available for advanced dental corrective procedures.

I hope you enjoy the read.

Liesel

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We welcome any comments, contributions, topic suggestions and letters for publication. Send them to:
The Editor, PO Box 232, GROENKLOOF, 0027
Tel: (012) 346 1590, 082 575 6479. Fax: 086 671 9907
Email: lieselvdmvet@gmail.com
(Dr Liesel van der Merwe)

Advertising Enquiries: The Publisher. Vetlink.
Madaleen Schultheiss: madaleen@vetlink.co.za

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Madaleen Schultheiss

Understanding the Use of Antibody Titres in Veterinary Practice

Antibody titres are a tool to assess an individual patient's immune response to vaccination for some common diseases. As titres grow in popularity, veterinarians must learn when to perform them and how to interpret the results to maintain patient health.

Kate Boatright, VMD

As concerns about over-vaccination and vaccine-associated injuries have grown in human medicine, vaccine compliance has decreased. In some areas, these concerns have spilled over into veterinary medicine and led pet owners to question the vaccine protocols recommended by their veterinarian. Due to these concerns, vaccine titre may be requested in lieu of vaccination. In other cases, the veterinarian may recommend a titre due to a patient's history.

No matter who suggests performing a titre, it is imperative that we as veterinarians understand which titre are reliable and how to interpret them to best protect our patients. The AAHA Canine Vaccination Guidelines, published in 2017 and updated in 2018, include a section on antibody testing for vaccine-preventable diseases.

In a lecture at the 2020 Midwest Veterinary Conference, held in February in Columbus, Ohio, lead guidelines editor Richard Ford, DVM, MS, DACVIM, DACVPM (Hon), discussed utilisation of antibody titres in companion animal practice. He encouraged practitioners to shift their thinking away from the dichotomy of vaccination versus titre testing and instead consider "using antibody testing to assess response to vaccines."

Which titres are reliable?

The only titres for which a positive test result has a high correlation with protective immunity are for the vaccine-preventable diseases canine distemper virus, canine parvovirus, canine adenovirus and feline panleukopenia virus. In practice, both quantitative and qualitative titres are available for these diseases.

Quantitative titres are laboratory-based tests that provide results in a matter of days. Dr. Ford cautions that the amplitude of the antibody titre should not be equated to a level of immunity. A patient is either immune or not.

Qualitative titres are also available as point-of-care tests that can be performed in the clinic and provide results much more quickly. These tests have been validated, and a positive result correlates well with the results of quantitative tests.

How should titre results be interpreted?

Dr. Ford cautions practitioners that a positive result can have different meanings depending on the titre performed. Only positive antibody titres for canine distemper virus, parvovirus, adenovirus and feline panleukopenia can be interpreted as protection. Based on the current laws, a positive rabies antibody titre can only be interpreted as exposure to the vaccine. These results cannot be interpreted as an indication of protective immunity.

When a titre produces a negative result for canine distemper, parvovirus, adenovirus or feline panleukopenia, clinicians should not assume an adult patient with previous vaccination history is susceptible to the disease. Over time, antibody levels circulating in the blood will decrease in the absence of exposure, but the immune system possesses memory that can produce an immune response should it encounter the disease. A negative titre in a puppy undergoing its initial vaccine series indicates that the immune system has not yet responded to vaccinations or may be unable to respond.

Why should titres be performed?

In Dr. Ford's experience, the most common reason for practitioners to perform titres is at the request of an

owner. The AAHA guidelines discuss 12 indications for titre testing and guide practitioners on how a positive or negative result should be used to guide patient management in each case. Dr. Ford highlighted several of these indications during his lecture.

Assess immunization status in puppies

Dr. Ford repeatedly emphasised that just because we are vaccinating pets does not mean they are immunised against a disease. Immunisation occurs after a pet's immune system has mounted a response to the organism(s) in the vaccine.

Research has shown that at 12 weeks of age, only 50% of puppies have been immunised against canine distemper virus and parvovirus. This lack of immunisation is thought to be due to interference from maternal antibodies. The goal of a puppy booster series for modified-live vaccines is to administer at least one vaccine after maternal antibodies are no longer present, leading to the recommendation of vaccination through at least 16 weeks of age. However, at 16 weeks of age, Dr. Ford stated that 15% of puppies are still not immunised.

When an owner needs to know if immunisation has been achieved, a titre could be performed two to four weeks after completion of the initial vaccine series. These cases could include especially valuable animals, those that will be traveling extensively, or those living in endemic disease areas. If titres are negative, an additional dose of the vaccine should be administered. An additional titre can be performed two to four weeks after this booster to assess immunity.

Identify genetic nonresponders

A small subset of dogs are genetic nonresponders to parvovirus vaccine (but will respond to vaccination for other preventable diseases, including distemper and adenovirus). These pets are at high risk for infection if they are exposed to the disease, and lifestyle modifications to minimise exposure are needed to protect these dogs. At this time, these dogs can only be identified through serial vaccine titres. If a negative titre is obtained to parvovirus following the final puppy booster, one additional booster should be administered. If a negative titre is again obtained, the patient can be identified as a nonresponder.

Assess immunisation status in adults

Titres can be used to determine whether a pet has protective immunity to canine distemper virus, parvovirus, adenovirus or feline panleukopenia. This can be helpful if the vaccination history of an adult dog is unknown or if the client requests a titre in lieu of vaccination. If the titres are positive, the pet does not need to be revaccinated. If a titre is negative, a booster is recommended, especially if the pet's vaccine history is unknown.

Determine whether vaccination is necessary in adult patients with comorbidities

At times, there may be contraindications to vaccinations, including a history of severe adverse vaccine reaction or immune-mediated disease, a chronic illness or a patient undergoing immunosuppressant therapies. In these cases, a titre can help guide patient management.

In cases in which a positive titre is obtained, the pet is protected and revaccination is not needed. When a negative titre is obtained, the pet may not be protected and the clinician and client should work together to make the best decision for the pet in light of the comorbidities present.

Take-home message

Dr. Ford encouraged veterinarians to consider titres as a tool that can be used to assess a pet's response to previous vaccination and determine if revaccination is necessary. Positive titres for canine distemper, parvovirus, adenovirus and feline panleukopenia are indicative of protective immunity and can give a clinician confidence that revaccination is not currently needed.

Questions as to how frequently titres should be performed remain unanswered. The AAHA Vaccine Guidelines offer extensive information on antibody titres for vaccine-preventable diseases and can be used as a guide for practitioners on when to perform titres and how to interpret them.

Dr. Boatright, a 2013 graduate of the University of Pennsylvania, is an associate veterinarian and freelance speaker and author in western Pennsylvania. She is actively involved in the AVMA House of Delegates as well as her local and state veterinary medical associations. She is a former national officer of the Veterinary Business Management Association.

Common Dilemmas in Epilepsy

Steven De Decker, DVM, PhD, MvetMed, DipECVN, FHEA, PGCert Veted, MRCVS
European and RCVS recognised Specialist in Veterinary Neurology

Epilepsy is probably the most common neurological disorder in veterinary medicine. The unpredictable and violent nature of epileptic seizures can cause severe emotional distress to owners of affected animals. Successful management requires an accurate assessment of the patient and commitment of the owner. A good relationship and mutual respect between the veterinarian and client are crucial for the optimal management of epilepsy. It is therefore important that you feel confident about clinical decision making and supply the owner with all necessary information.

What is a seizure?

One of the most common reasons for unsuccessful treatment of epilepsy is that the observed 'collapse or fitting episodes' are not epileptic seizures. Other disorders can be difficult to differentiate from epileptic seizures and include cardiogenic syncope, exercise induced collapse, REM-sleep related disorders, and movement disorders. Movement disorders are increasingly recognised and can be difficult to differentiate from epileptic seizures, and focal seizures in particular. It is therefore important to get a detailed clinical history and good description of the events from the owners. A classical 'text-book' epileptic seizure has the following characteristics:

- Lateral recumbency and tonic-clonic movements ('jerking movements', 'paddling', 'running movements') of all limbs. The limbs and body are typically stiff. Loss of consciousness
- An epileptic seizure typically occurs at rest or during sleep.
- Lasts typically less than 2 to 3 minutes.
- Autonomic signs such as urination, defecation and salivation can be present
- The character of the episodes is typically consistent between each epileptic seizure
- Post-ictal phase. When the actual epileptic seizure is finished, it might take a while before the animal is again completely its normal self. For minutes, hours or even days animals might demonstrate ataxia, abnormal behaviour, barking, aggression, transient blindness, or demonstrate abnormal hunger or thirst.

It is very uncommon for other causes of collapse to be associated with decreased consciousness, be associated with autonomic signs, occur during sleep or be followed by a post-ictal phase.

What is the difference between an epileptic seizure and epilepsy?

An epileptic seizure is the 'event' or 'episode' itself and is not the same as epilepsy. Epilepsy is a disease of the brain characterized by an enduring predisposition to generate epileptic seizures. Practically, it can be defined as two or more unprovoked epileptic seizures more than 24hours apart.

Epilepsy has also been divided into two broad aetiological categories: idiopathic epilepsy and structural epilepsy.

Idiopathic (previously also called primary) epilepsy is the most common type of epilepsy and refers to a disease characterised by recurrent epileptic seizures in which there is no identifiable brain abnormality. Epilepsy is the 'primary' problem. It has been considered that idiopathic epilepsy has a genetic basis in the vast majority of patients. Structural (previously called secondary) epilepsy is defined as epilepsy caused by an identifiable forebrain pathology, including congenital, vascular, neoplastic, inflammatory and degenerative disease.

Easy to recognise clinical characteristics can be used to make a presumptive differentiation between idiopathic and secondary epilepsy. Dogs with idiopathic epilepsy:

- Are of a breed in which there is a high prevalence of idiopathic epilepsy
- Are aged between 6 months and 6 years of age
- Do not demonstrate any other clinical signs than epileptic seizures
- Have an unremarkable general physical and neurological examination
- No significant abnormalities on blood work and urinalysis

It is important to realise that animals will demonstrate neurological abnormalities during the post-ictal phase. Even in animals with idiopathic epilepsy, it will be normal to have an abnormal neurological examination in the first hours after a seizure. It has therefore been suggested that results of a neurological examination should not be considered 100% reliable in the first 24 hours after a seizure.

It is further important to avoid confusing anti-epileptic drug (AED) related neurology side-effects (i.e., ataxia and sedation) with an abnormal interictal neurological examination.

When to start treatment and what to expect?

It is difficult to advise when exactly you should start treatment. This is dependent on many factors of which one of the most important will be owner's preference. Owner compliance is a key factor in treatment of epilepsy and the owner should therefore support the idea that starting treatment is necessary. Although treatment decisions are different for each individual patient, the following general recommendations have been suggested:

- An identifiable structural lesion or prior history of brain disease or injury (i.e. structural epilepsy)
- Status epilepticus or cluster seizures
- Two or more seizure events within a 6-month period
- Prolonged, severe, or an unusual postictal period
- The epileptic seizure frequency and/or duration and/or severity is increasing over 3 interictal periods

Treatment of epilepsy is complicated when your client has unrealistic expectations. It is important to realise that there is no cure for idiopathic epilepsy and only a small percentage of animals will become seizure-free. It is therefore expected that treatment will be lifelong and that occasional seizures will be observed throughout the life of the pet. A realistic aim of treatment is to maintain an acceptable quality of life, which consists of a balance between decreased seizure frequency and avoidance of unacceptable AED side effects.

It should also be noted that 20-30% of cases will be refractory to medical management. The term 'refractory' is used when a patient has poor seizure control despite plasma drug concentrations of two or more adequate AEDs within the accepted therapeutic range. It is unlikely these patients will respond to addition of more AEDs.

Which treatment is recommended?

Although treatment of epilepsy can seem complicated, only a few anti-epileptic drugs (AEDs) are available. Having a good understanding about the indications, side effects and limitations of these AEDs will increase your confidence and success rates. From a practical point of view, AEDs can be divided into three categories:

1. **Very fast-acting, very short-lasting effect**
   a. Benzodiazepines, such as diazepam and midazolam.
   b. These drugs work immediately and are used stop seizure activity in emergency situations. Their therapeutic effect last typically only a few minutes. These drugs should not be used as maintenance drug.

2. **Fast-acting and intermediate lasting effect**
   a. Levetiracetam
   b. Has a therapeutic effect within the first hour and is associated with minimal side effects. This is a very useful AED in emergency situations, but not effective as a maintenance drug.

3. **Slow-acting and long-lasting effect**
   a. These are the classic maintenance drugs. A delay in therapeutic effectivity is seen after administration, but the effect lasts longer.
   b. The most commonly used drugs are phenobarbitone, potassium bromide and Imepitoin (Pexion®). Zonisamide is not often used in Europe, but seems more popular in the United Sates.

There are no evidence-based guidelines regarding which AED drug should be used first and your choice will be influenced by multiple factors, including efficacy, side effect profile, individual preference, need and complexity of monitoring and costs. When you have made the decision to start treatment, you will typically select one of the following three drugs: phenobarbitone, potassium bromide, or Imepitoin. Benzodiazepines and Levetiracetam are not useful as maintenance drug (i.e. daily administration) in dogs. Levetiracetam can however be considered as maintenance drug in cats.

Phenobarbitone:
This is the first-choice AED for most veterinary surgeons. It has a proven efficacy and is relative safe when administered within the dose range. Most side effects are dose dependent and occur immediately after starting treatment or a dose increase. These side effects include sedation, ataxia, polyphagia, polydipsia and polyuria. They typically improve or resolve over the first two weeks after starting treatment. Idiosyncratic drug reactions are very rare. Changes in serum biochemistry profile variables are commonly seen of which (a clinically irrelevant) increase in hepatic enzymes is most common.

The recommended starting dose is 3 – 5mg/kg BID in dogs and 2 – 4 mg/kg BID in cats. There is individual variability in phenobarbitone absorption, excretion and elimination half-life. Absolute dosages (for example 3mg/kg/BID) are therefore not reliable to evaluate response to treatment. Steady state concentrations are typically reached between 10 and 15 days. Therapeutic serum concentrations should therefore be evaluated 2 weeks after treatment initiation or dose changes. Therapeutic serum concentrations are a key factor in evaluating response to treatment and should be used to guide dose changes or consideration of additional AEDs. Phenobarbitone should be titrated until the higher range of the provided therapeutic reference interval before it can be considered ineffective. If, for example, the provided therapeutic reference interval is 20 – 40 mg/dl, then a serum

concentration of 30-35 mg/dl should be reached before considering phenobarbitone to be ineffective. One of the most common mistakes in treating epilepsy is to add another AED before the initial AED has given the opportunity to reach its maximum therapeutic potential. (Ed. - It is no longer considered necessary to time the collection of phenobarb to 4 - 6 hours post treatment unless your dose is reaching 8 - 10 mg/kg)

Phenobarbitone induces an enzyme called cytochrome P-450 in the liver. This has two important clinical consequences:
1) phenobarbitone is contra-indicated in animals with liver failure and
2) higher cytochrome P-450 promotes elimination of phenobarbitone.
Chronic phenobarbitone administration will therefore over time result in quicker elimination of itself.

Historically, there have been concerns about the potential of liver failure in animals receiving phenobarbitone. This is however very rare if the serum concentration is kept within its therapeutic range. For these reasons, it is important to continue monitoring the phenobarbitone serum concentration over time. Haematology, serum biochemistry and serum concentrations of phenobarbitone should be evaluated every 6 months after therapeutic serum concentrations have been reached.

Potassium Bromide:
This drug is typically used as an add-on drug when dogs are not demonstrating a good response to phenobarbitone or if phenobarbitone is contra-indicated (i.e., animals with liver failure). Potassium Bromide is renally excreted and can therefore be given to dogs with liver disease. Potassium bromide should not be given to cats due to a high incidence of severe respiratory complications. The initial dose is 20mg/kg BID. The elimination half-life is much longer than for phenobarbitone and steady-state serum concentrations are only reached after 80-120 days. Therapeutic serum concentrations are therefore only evaluated after 90 days.

Side effects include ataxia, pelvic limb stiffness, sedation, gastro-intestinal irritation. megaesophagus, pruritus and pancreatitis

Imepitoin:
Imepitoin or Pexion© is a relative new drug and is currently licensed in Europe and Australia. It is only registered for the management of single generalized epileptic seizures in dogs with idiopathic epilepsy. It is therefore not registered for use in cats, treatment of cluster seizures, status epilepticus or treatment of structural epilepsy.

It has a similarly reported efficacy as phenobarbitone. Although most side effects are similar in nature as those for other AEDs (i.e. sedation, ataxia, polyphagia, polydipsia, and polyuria), they occur significantly less frequently compared to phenobarbitone. Imepitoin should not be given to dogs with liver, kidney or heart failure. Due to its favourable safety profile, valuation of serum concentrations is not necessary.

Levetiracetam and benzodiazepines:
Benzodiazepines, such as diazepam, should not be used as maintenance AED in dogs and cats. Dogs become rapidly tolerant to the anti-seizure effects of benzodiazepine and a high number of cats develop severe and even life-threating side effects (next to questionable efficacy).

Levetiracetam should not be used as a maintenance AED in dogs due to the development of tolerance after 4-6 months of continuous administration. Levetiracetam is however very useful in emergency situations and is an important AED to treat status epilepticus and cluster seizures. Levetiracetam has been recommended as maintenance AED in cats. A dose of 20mg/kg three times daily is recommended. Due to its favourable safety profile, evaluation of serum concentrations is not necessary
(Ed. - Levetiracetum reaches maximum serum concentration within 90 +/- 60 minutes hours and lasts for 9 hours. It can be given per rectum, is a solution, at 40mg/kg (ref JVIM 201:33,1714 - 1718. Cagnotti G et al, BMC Vet Res. 2018:14, pg189 Cagnotti G et al))

What should you definitely discuss with your client?
An important challenge is to provide your client with all necessary information without overwhelming them. A well-informed client will have realistic expectations and will easier accept AED related side effects and will understand the importance of regular evaluation of therapeutic drug serum concentrations. Receiving the news that your pet has epilepsy can however be emotionally distressing. It might therefore be difficult to understand or remember all information provided during the initial consult. It might therefore be necessary to arrange a separate consult or provide your client with an information leaflet.

The owner should have a good understanding of the following concepts:
- What is epilepsy?
- Nature of idiopathic epilepsy
- Indications to start treatment
- Realistic expectations for treatment
- Expected short-term complications
- Potential long-term complications
- How and when to monitor treatment

References available online: www.vet360.vetlink.co.za

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CPD Questions

Common Dilemmas in Epilepsy

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01. The owner will often confuse other episodic events with a generalised seizure. It is important that the veterinarian clearly differentiates a seizure from the confounders. Which one of the conditions listed below is not a confounde?

a. Cardiogenic syncope.
b. Movement disorders.
c. Behavioural disorders.
d. Cardiac arrhythmias.
e. Sleep disorders.

02. What are the characteristics of an epileptic seizure? Which one of the characteristics listed below is incorrect?

a. Limbs and body are stiff.
b. Loss of consciousness.
c. Urination and salivation can be present.
d. The character of the episodes can differ between episodes.
e. The post ictal phase may last several hours.

03. The classification of epilepsy is broadly differentiated into 2 categories: Which one of the signs listed below is typical of idiopathic epilepsy?

a. There is brain neoplasia.
b. There is an inflammatory meningoencephalitis.
c. There is a viral infection such as FIV , FeLV or FIP.
d. Hypoglycaemia is present.
e. There is no identifiable brain abnormality.

04. The decision to initiate antiepileptic medication is based on several considerations. Which one of the considerations listed below is not considered relevant?

a. Structural epilepsy is diagnosed.
b. The seizure appears especially severe although the duration is short.
c. The patient experiences cluster seizures.
d. The frequency of the seizure episodes is increasing over a short period
e. The post ictal period is obvious and prolonged.

05. Phenobarbitone is often a first line choice for many vets. Which one of the characteristics listed below is not one of this drug class?

a. Proven efficacy.
b. Relatively safe within the recommended dose range.
c. Dose dependent side effects.
d. Side effects typically improve / resolve within 2 weeks of initiation of treatment.
e. Clinically relevant changes in hepatic enzymes typically occurs.

06. Phenobarbitone is used correctly is an effective antiepileptic drug. Which one of the following statements regarding its use is correct?

a. The recommended starting dose is 3-5 mg/kg bid.
b. The recommended starting dose is 2 mg/kg bid.
c. You can taper the drug to an oid treatment frequency.
d. Therapeutic concentrations are a nice to have, but you can use the mg / kg dose as a treatment guideline.
e. If the animal starts seizuring again after dose initiation. A new medication needs to be added.

07. Phenobarbitone used as an AED is based on certain basic premises. Which one of the statements below is incorrect?

a. Dose changes should be guided by serum levels of phenobarbitone.
b. The top 25% of the therapeutic range should be reached before considering the drug ineffective.
c. Serum levels in the top of the normal range mean that the dose must be reduced to once a day.
d. Steady state concentrations are reached within 10 – 15 days.
e. New evidence suggests blood can be collected at any stage during the day.

08. Which one of the following statements regarding imipetoin is incorrect?

a. Imipetoin is called Pexion® in SA.
b. Imipetoin has a good safety profile.
c. Imipetoin has similar efficacy as pehnobarbitone.
d. The side effects as with phenobarbitone therapy do not occur at all with ipimetoin.
e. It is not necessary to check serum concentrations with imipetoin.

09. If the initial medication is not providing sufficient control a second add-on drug to phenobarbitone can be used. Which of the follow statements is incorrect?

a. Potassium bromide can be used as an add on drug.
b. Imipetoin is a good choice as an add on drug.
c. Potassium bromide has a long halflife - serum concentrations are only reached after 80 – 120 days.
d. Potassium bromide is safe to use in dogs and cats.
e. Potassium bromide is renally excreted.

10. Medication used for status epilepticus is different to that used for chronic management. Which one of the following statements is incorrect ?

a. Benzodiazepines are effective for long term management of epilepsy in dogs.
b. Cats can develop life threatening complications due to benzodiazepines.
c. Levetiracetum should not be used as a maintenance antiepileptic drug – as tolerance develops after 4 – 6 months of use.
d. Levetiracetum is an important medication in the stabilisation of status epilepticus.
e. Owners must be aware that chronic management will only result in partial (maybe 50%) control and is not curative.

Pneumothorax as an Imaging Diagnosis in Small Animal Patients

Dr Kirstin Dinham BVSC MRCVS
IMV imaging in-house veterinarian

Background

Pneumothorax is the presence of free gas in the pleural space, between the lung and the chest wall. Aetiologically, pneumothorax can be classified as spontaneous, iatrogenic or traumatic[1], with the latter being the most commonly diagnosed[2,3]. Atmospheric air can enter the pleural space through perforating wounds of the thoracic wall or traumatic rupture of the lung parenchyma, trachea, bronchi or oesophagus. Iatrogenic pneumothorax can be caused by sampling procedures such as thoracocentesis or chest tube placement. Spontaneous pneumothorax has been attributed to the rupture of pulmonary bullae or blebs, that can act as a one-way valve and lead to the development of a tension pneumothorax. Other causes of spontaneous pneumothorax include pulmonary neoplasia, various helminth infections, bacterial and viral pneumonia, migrating foreign bodies, pulmonary abscesses, and parasitic granulomas[4]. Siberian huskies were overrepresented in one study on spontaneous pneumothorax[4].

In most small animals, pneumothorax occurs bilaterally due to the presence of mediastinal fenestrations, although unilateral pneumothorax can occur[5].

Radiography

Traditionally, pneumothorax has been diagnosed using two- or three-view thoracic radiography. Lateral views are more sensitive than ventrodorsal (VD) and dorsoventral (DV) views, and DV views are preferable to VD views[6]. The radiological features of a pneumothorax include (Image 1)[1]:

- Radiolucent areas in the periphery of the thorax.
- Retraction of the lungs from the thoracic wall.
- Increased radiopacity of the lungs. This occurs because tension in the pleural cavity caused by the accumulation of air prohibits expansion of the lung parenchyma, leading to a denser appearance radiographically.
- Shifting of gas around collapsed lungs causes the heart to fall away from the sternum resulting in a radiolucent area between the cardiac silhouette and the sternum on a lateral view.
- In some cases, lung bullae may be seen.

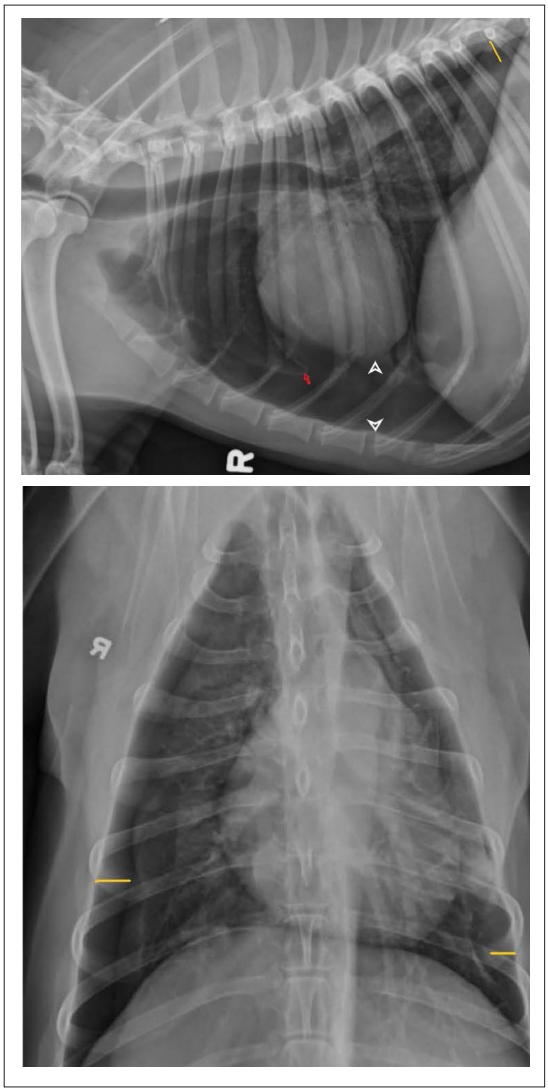

Image 1: Pneumothorax in a dog with classical radiographic findings, on the left is a right lateral and on the right is a dorsoventral view. White arrows, radiolucent area between the cardiac silhouette and the sternum; red arrow, retracted cranial lung lobe; yellow lines, radiolucent areas on the periphery of the thorax. Images Courtesy of Dr Hespel A-M, University of Tennessee.

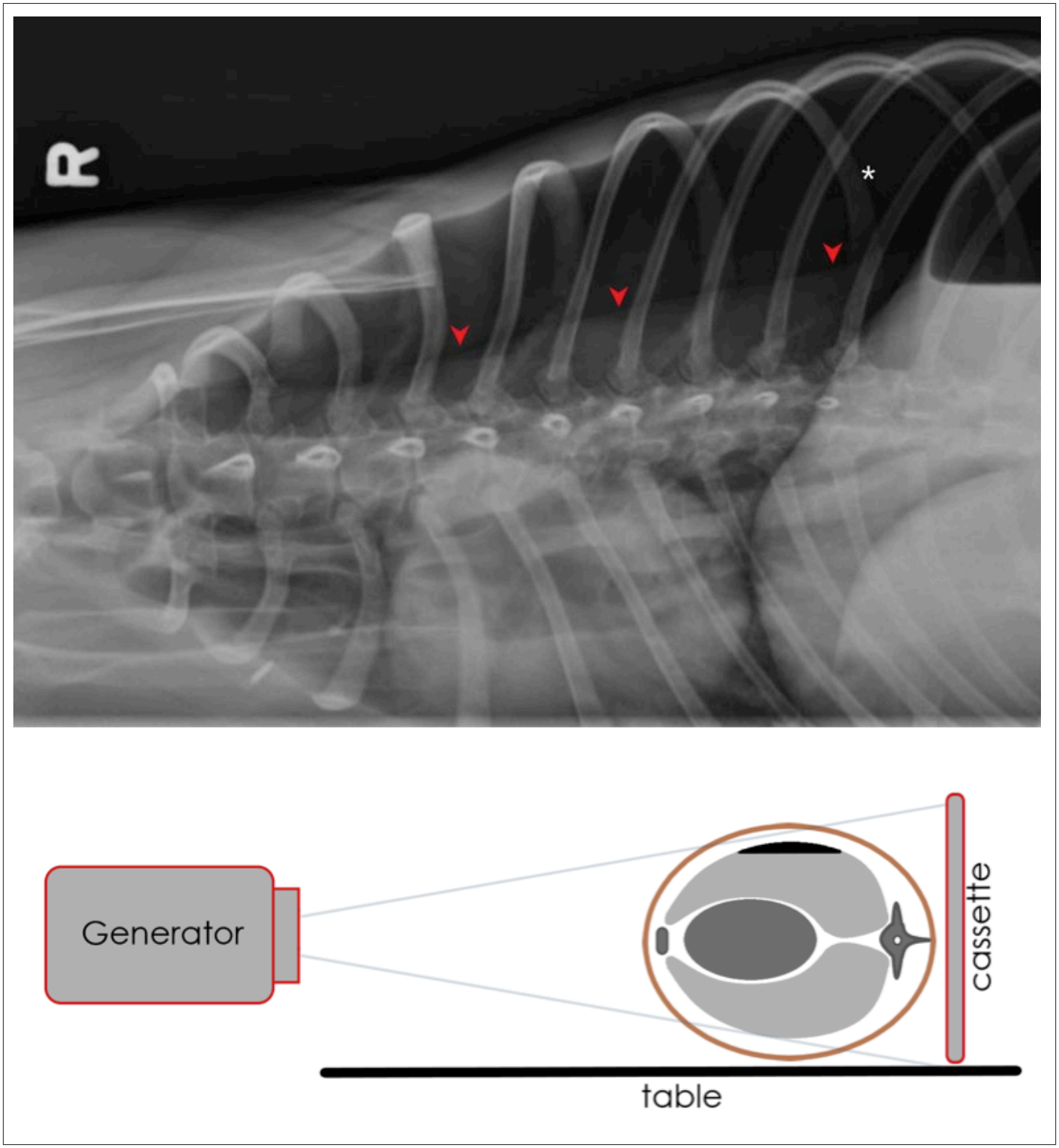

Image 2: Horizontal beam radiography. Top image, schematic diagram demonstrating the technique. The black area in the dorsal aspect of the grey left lung field represents a small volume pneumothorax. Bottom image, resultant horizontal beam, ventrodorsal image of a dog with pneumothorax lying in right lateral recumbency. There is retraction of the right lung field from the thoracic wall (red arrowheads), with an overlying radiolucent area (*) consistent with gas opacity. Radiographic image courtesy of Dr Hespel A-M, University of Tennessee

The presence of small volume pneumothoraces that are radiologically 'invisible' or 'occult' is well recognized in human literature, with estimates of 30 – 55% of pneumothoraces being undetected on radiography[7]. This is of clinical importance because there is increased risk of patients developing worsening pneumothorax or a tension pneumothorax under mechanical ventilation and general anaesthesia[8].

A recent study of 59 blunt trauma small animal patients at a veterinary university hospital published similar findings [8]. The consensus sensitivity of three case-blinded and independent observers (two radiologists and a radiology resident) for pneumothorax was 19% with three-view and 63% with horizontal beam radiography when compared with computed tomography (CT). Notably, radiography of other thoracic lesions commonly seen in trauma patients such as pleural effusion, rib fractures and lung contusions also demonstrated low sensitivity relative to CT of 43%, 56% and 69%, respectively[8]. The low sensitivity of radiography for pneumothorax is likely to occur because of the superimposition of other structures in the thorax over small volumes of free gas and air trapped between the lung lobes and around the mediastinum[8].

The addition of a horizontal beam VD view with the animal in right lateral recumbency has been shown to increase sensitivity for pneumothorax[8,9], and it would be prudent to include in all trauma patients. Horizontal beam radiographs require an x-ray tube head that can be rotated and lowered or a portable x-ray generator, and may not be possible to perform with direct digital radiography systems that are wired under a bucky tray (Image 2).

Ultrasonography

The gold standard diagnostic investigation for patients with thoracic trauma is CT scan and should be performed where possible (Image 3). However, despite increasing numbers of CT scanners in veterinary practices, it remains an underutilized modality in most general practices due to cost and availability concerns.

Thoracic ultrasound has proven to be a sensitive and specific modality for the detection of occult pneumothoraces in human medicine, where sensitivity ranges from 92 – 100%[7]. A meta-analysis of 20 human studies listed the pooled sensitivity and specificity of this modality at 88% and 99%, respectively[10].

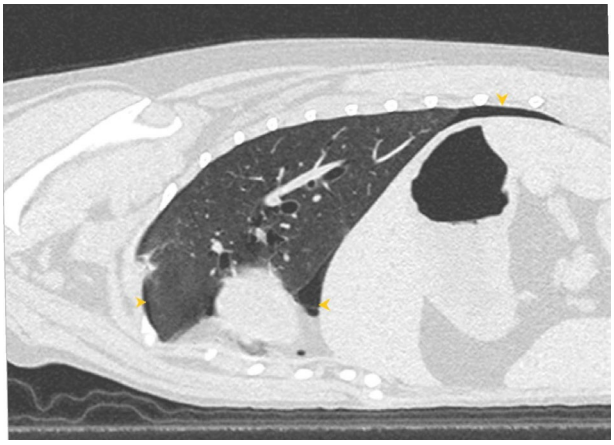

Image 3: Sagittal Plane CT image of a patient with pneumothorax demonstrating low volumes of free gas in the pleural space (yellow arrows). Image courtesy of Dr Hespel A-M, University of Tennessee

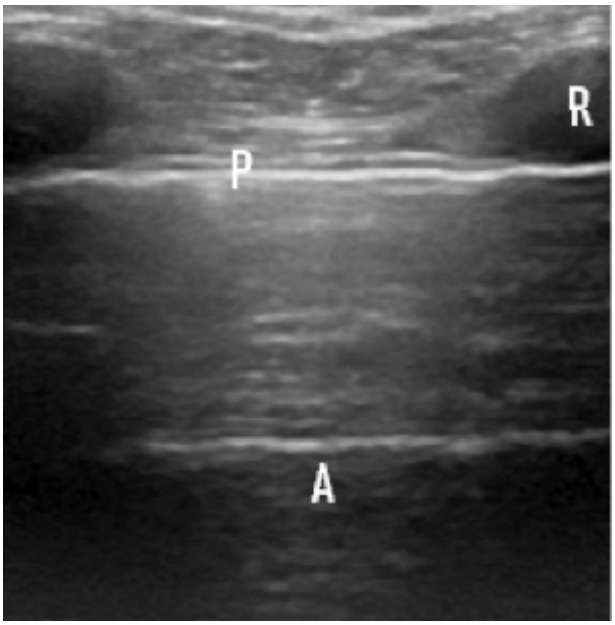

Image 4: Ultrasound image of normal lungs. P, pleural line; R, ribs with distal acoustic shadows; A, A-line reverberation artefacts.

A simple TFAST (Thoracic Focused Assessment with Sonography for Trauma) ultrasound protocol for small animal patients was proposed in 2008[11]. In this study, the sensitivity and specificity of this point-of-care procedure was assessed against radiography; an inherently insensitive but very specific technique for the detection of pneumothorax[8]. A subsequent study was not able to show any correlation between TFAST results and CT diagnosis for pneumothorax[12]. Although TFAST can undoubtedly be very useful in an emergency setting, and particularly with regards

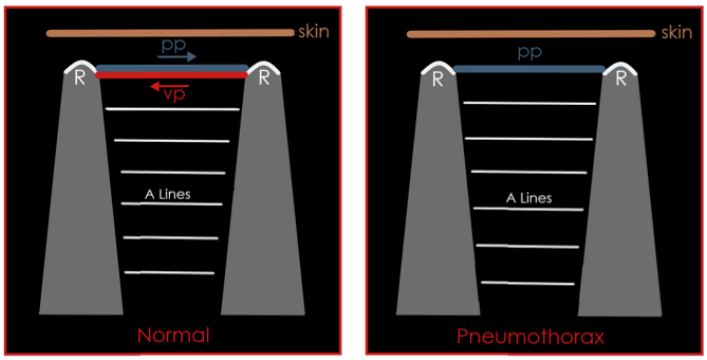

Image 5: Illustration on the left depicting the 'to-and-fro' movement of the interface of the visceral pleura (VP) and parietal pleura (PP) in normal respiration (arrows). A lines are generated deep to the surface of the VP. Illustration on the right depicts a pneumothorax where gas deep to the (PP) obscures the surface of the lung and causes a loss of the glide sign

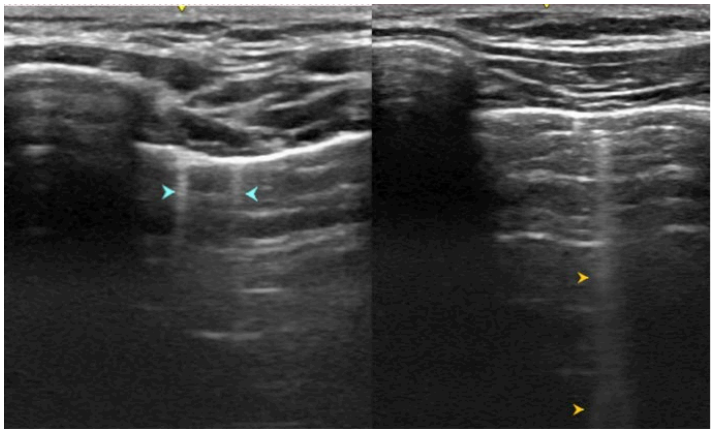

Image 6: Thoracic ultrasound images depicting different types of B lines (arrowheads). These are vertically orientated lines which originate from the surface of the lung and represent penetration of the ultrasound beam deep to the visceral pleura and are a rule-out for the presence of pneumothorax in that area.

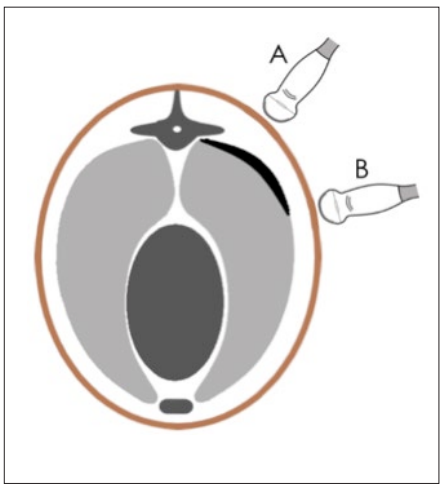

Image 7: Schematic cross section through a canine thorax with black area depicting a small volume pneumothorax. At position A one would expect to see lack of glide sign, and as the operator moved the probe ventrally, the 'lung point' at B should come into view. This finding can provide information on the subjective volume of the pneumothorax

Sonographic Indicators of Pneumothorax

Understanding the sonographic indicators of a pneumothorax is based on knowledge of the anatomy of the pleural space and the physics of ultrasound at a soft tissue and gas interface. To perform a lung ultrasound for pneumothorax, one can use a high frequency linear or micro-convex probe, the patient can be clipped, or the fur soaked in spirit, and accuracy is dependent on operator experience[11].The pleural space is a potential space between the parietal and visceral pleurae, i.e. the inner surface of the thoracic cavity and the outer lung surface. In a normal patient, one is unable to visualize anatomy deep to the visceral pleura because the lung parenchyma is filled with gas. Gas-soft tissue interfaces reflect the ultrasound waves back to the probe, due to the large difference in acoustic impedance values between soft tissue and gas. This contributes to the formation of a reverberation artefact, known as an A-line artefact, comprising equidistant horizontal lines deep to the pleural line (Image 4, 5).

In a normal patient the pleural space does not 'exist' as such, and one is able to visualize the interface of the parietal and visceral pleurae moving against each other causing a shimmering 'to-and-fro' movement called the 'glide sign'. This is a dynamic finding that cannot be captured in a still B-mode image. The presence of the 'glide sign' means that there is no gas present between the visceral and parietal pleura. In the presence of a pneumothorax, visualization of the deeper visceral pleura would be obscured due to reflection of the sound waves, and so one would not be able to visualize the interface of the two surfaces sliding against each other. Similarly, artefacts that originate from the lung surface, such as B-line artefacts are a rule-out finding for pneumothorax in that area of lung (Image 6).

The 'lung point' is considered a highly specific finding for pneumothorax[11] and is the boundary where normal lung meets collapsed lung and visually would be the point where the glide signs return (Image 7). Additionally, this sign can be useful for estimating the volume of the pneumothorax.

The 'reverse sliding' sign has been described as a sensitive and specific sign for the detection of low volume pneumothorax[13]. This sign occurs when a small, trapped pocket of air appears to move in the opposite direction to lung movement during inspiration.

to the detection of pleural and pericardial fluid, further research into its reliability, perhaps differentiated for occult versus radiographically visible (and therefore larger volume) pneumothoraces would be valuable. A prospective study from 2018 induced a low volume pneumothorax in 9 healthy adult beagles, and CT (the gold standard), radiography and ultrasonography were performed sequentially[13]. In this study, pooled sensitivity and specificity of ultrasonography were 88.8% and 88.8%, respectively and 66.8% and 88.8%, respectively, for thoracic radiography. Notably, a more comprehensive thoracic ultrasound examination was undertaken than the 4-point TFAST protocol..

Conclusion

Radiography is a good first line technique for patients with thoracic injury, however the traditional three-views are insensitive for detection of low volume pneumothoraces. The addition of a horizontal beam ventrodorsal view can improve the sensitivity of this modality (63% versus 19%)[8] and therefore should be performed where possible.

Further research is required to enable a more accurate assessment of the value of thoracic ultrasound in the diagnosis of low volume pneumothorax in small animal patients. However, the proven superiority of this modality in comparison to radiography in human medicine for the detection of low volume pneumothoraces, combined with the challenges in veterinary medicine with regards to accessibility and affordability of the gold standard CT, make awareness and familiarity with ultrasound of the thorax and sonographic signs of pneumothorax a useful weapon in one's diagnostic armory.

Special Thanks

Special thanks to Dr Adrien M Hespel DVM, MS, DACVR for assisting with image provision.
Dr Hespel has a veterinary radiology blog, where he posts regular and interesting cases at https://veterinaryradiologymirc.squarespace.com/ . Dr Hespel also shares cases regularly on our IMV Imaging Small Animal Facebook page.

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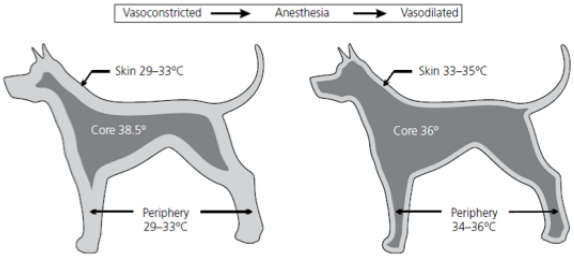
Hypothermia
Foe More Than Friend

Justin F. Grace BVSc, Anaesthesiology resident, ECVAA resident

Hypothermia is one of the most commonly encountered complications during the perianaesthetic period (premedication to recovery). The American College of Veterinary Anaesthesia and Analgesia Monitoring Guidelines highly recommends monitoring core body temperature during the perianaesthetic period as deviations from normal have certain implications and adverse effects.

Hypothermia, defined as a core body temperature below normal (below 36 °C for cats and dogs), occurs when heat loss exceeds heat production or heat conservation. Heat loss is potentiated by cold hospital environments, altered patient thermoregulation (either by pharmacological intervention or pathological causes) and inadequate maintenance of core body temperature close to normothermia. It must be emphasised that heat loss is profound in patients less than 10 kg, because their body-surface-to-weight ratio is much larger compared to larger patients. In dogs, it was demonstrated that they can lose up to 1.9 °C in rectal temperature during the first hour of anaesthesia following normal patient handling but this is highly variable depending on patient size and surgical procedure.

A healthy individual usually maintains a thermal set-point which is strongly associated with the inter-threshold range of approximately ± 0.2 °C. Should temperature deviate outside of the inter-threshold range, compensatory responses are implemented to



return temperature to normal. These compensatory responses included vasoconstriction, vasodilation, sweating and shivering. However, certain anaesthetic drugs, including opioids, hypnotic agents, and sedatives can alter the set-point temperature, as well as widen the inter-threshold range up to 3.5 °C.

Body heat is not uniformly distributed throughout the body. The core body temperature is 2-4 °C warmer than the periphery. During the peri-anaesthetic period, a reduced core-to-periphery gradient exists which decreases the body core temperature as more heat is transferred to the periphery and released to the surrounding environment. This is because most anaesthetic drugs have an indiscriminate vasodilatory effect. The α2-adrenergic receptor agonists with its vasoconstrictive properties may slow the loss of body heat due to decrease peripheral blood flow.

Heat energy is transferred to the surrounding environment by four main mechanisms. These include: radiation (75% of heat loss), the transfer of energy between surfaces; convection, the transfer

of heat energy by air movement (heat loss increased by clipping of hair); conduction, the transfer of heat by direct contact (cold surfaces and alcohol skin preparations); and evaporation of water (worsened by exposure of abdominal or thoracic viscera). Minimal heat is lost via the respiratory tract and hence airway heat exchangers contribute little to increasing the patient's body core temperature.

Hypothermia has numerous adverse effects on the body and the severity of these adverse effects are proportional to the reduction in core body temperature. It has been shown that core body temperature decreasing down to 36 °C is not associated with detrimental effects in cats and dogs.

Major adverse effects on the body include:

- **Metabolic rate**
  - For every 1 °C drop in core body temperature, there is a 10 % decrease in metabolic rate.
  - Drug metabolism/elimination is decreased which can result in inadvertent drug overdose (e.g. opioid plasma concentration may increase with hypothermia due to altered redistribution and metabolism).
  - The clinic effects from neuromuscular blocking agents (NMBA) is prolonged especially those which are degraded by Hoffman degradation (temperature dependent).
  - Prolonged recovery postoperatively.
  - Reduced sodium pump activity of cells which results in swelling and oedema and leads to electrolyte abnormalities i.e. hyperkalaemia and acidaemia.

- **Blood viscosity increases**
  - PCV increases due to erythrocytes swelling and splenic contraction (accompanies acidosis and hypothermia).
  - Myocardial wo rk increases.
  - Increase incidence of clot formation.

- **Hypocoagulable blood**
  - Platelets sequester in liver, spleen, and bone marrow.
  - PT and aPTT are prolonged.
  - Blood coagulation is impaired due to clotting factors and enzymes in the cascade becoming defective (reduced function) and can lead to a 20% increase in blood loss.

- **Haemodynamic instability**
  - Blood pressure falls due to baroreceptor reflex sensitivity depression and resultant decrease in cardiac output (CO).
  - Reduced CO affects inhalant anaesthetic uptake from alveoli thus level of anaesthesia deepens. This with decreased CNS neuronal excitability also reduces minimum alveolar concentration (MAC).
  - Reduced CO leads to reduced GFR with subsequent reduction in urine output. But this is somewhat offset by diuresis from reduced sodium and water reabsorption.

- **Vasoconstriction**
  - At core body temperature below 34-35 °C, active peripheral vasoconstriction occurs (venous and arterial vessels become harder to palpte/access).
  - Vasodilation occurs below 20 °C as vasoconstriction can no longer be maintained.
  - This also leads to decreased blood flow and oxygen delivery to wound and results in delayed wound healing.

- **Excitable cell membranes are affected**
  - Conduction velocities are slowed.
  - Myocardium becomes irritable at core body temperature below 30 °C (arrhythmias).
  - Bradycardia is non-responsive to anticholinergics.
  - ECG: S-T depression and T wave inversion; P-R and QRS widening; J waves (Osborn waves) form at 30 °C core body temperature.
  - Ventricular fibrillation occurs at 28 °C.
  - Cardiac arrest occurs at around 20-23 °C.

- **CNS activity depression**
  - Reduced minimum alveolar concentration (MAC) of inhalant anaesthetics.
    - » Less inhalant anaesthetic required (between core body temperature of 32-34 °C).
    - » MAC is reduced because inhalants are more soluble in tissue.
  - Confusion occurs below 35 °C (in conscious patients). Different from emergence delirium.
  - Unconsciousness occurs at 30 °C.
  - Cessation of cerebral electrical activity occurs below 18 °C.

- **Reduced immune function**
  - Increase in surgical site infection and metastasis of neoplastic cells.

- **Postoperative shivering**
  - There is a 400% increase in oxygen consumption when a patient shivers.
  - This can lead to hypoxaemia and myocardial hypoxia if the patient is not ventilating adequately.
  - Advised to administer oxygen during postoperative shivering to compensate for increase oxygen consumption.
  - Shivering can also induce pain sensation because of shearing forces on the wound during movement.
  - Thermoregulatory shivering does not occur

at core body temperature below 34-35 °C.

- **Respiratory system**
  - Hypoventilation with respiratory acidosis because of chemoreceptor reflex sensitivity depression and reduced respiratory muscle activity.
  - Bradypnoea can also occur due to excessive levels of anaesthesia or hypothermia. This can result in a deficiency of oxygen and excessive carbon dioxide in the blood, causing acidosis.
  - Apnoea occurs around 24 °C.
  - Reduced mucociliary activity leading to increased respiratory secretion accumulation and infection.

- **Left shift of haemoglobin oxygen dissociation curve**
  - This leads to oxygen off-loading to the tissues being impeded and activation of anaerobic metabolism in tissue (adding to metabolic acidosis).
  - While a lower metabolic rate may mean a decreased oxygen requirement, this is negated by shivering as the patient warms up.

- **Death occurs around 20 °C.**
- Therapeutic hypothermia has been employed in specialised surgeries such as during cardiac bypass or following cardiac arrest, and the scope of which is beyond this discussion.

Prevention

A phrase to live by: 'Prevention is better than cure!'
Once a patient becomes hypothermic, it is difficult to raise the temperature during anaesthesia. The following steps can be used to reduce perianaesthetic hypothermia:

- Prewarming the patient prior to induction of anaesthesia for approximately 30 minutes to 2 hours (patient can however pant etc. and thus not be effective). This is to raise the body temperature so that during inevitable radiation losses, it returns towards normal as a decrease in core-to-periphery gradient is generated.
- Excessive clipping, scrubbing and use of alcohol solution should be avoided. And minimal time should be spent doing so.
- Having a warm environmental temperature during the perianaesthetic period (heater above patient, blanket over patient, air-conditioned room). A warm theatre is ideal, particularly for small patients, but may make the surgeon uncomfortable.
- Minimise surgical and anaesthetic times.
- Using prewarmed fluid when flushing the thoracic/abdominal cavity as this reduces heat loss.
- In-line fluid warmers can be beneficial but must comply with manufacturer's instruction, and generally the volumes of fluid administered are not enough to make a great difference.

Treatment of hypothermia

When rewarming a patient, heat must be transferred from the periphery to the core. This is accomplished more effectively by warming the skin surface. During anaesthesia, drug-induced vasodilation makes it easier to rewarm the patient due to smaller core-to-periphery gradient. However, rewarming a patient under anaesthesia until approximately 36.5 °C before recovery prolongs anaesthetic time and may increase risk of mortality and cost of treatment. For this reason, hypothermic patients must be diligently rewarmed perioperatively using any or combination of below mentioned methods.

There are two main categories by which hypothermia can be reduced:

- **Passive**
  - This method only reduces heat loss and does not generally facilitate rewarming of the patient unless some form of heat is generated by the patient itself i.e. shivering.
  - Blankets, surgical drapes, space blankets, and bubble wrap are practical examples.
  - Even one layer of protection can reduce passive heat loss by 30% and three layers reduce heat loss by 50%.
  - Covering the paws and limbs are essential due to larger surface-to-mass ratio.
  - Care should be taken when active warming devices are used in conjunction with passive and some material can absorb and conduct heat well, leading to thermal injury of the patient.

- **Active**
  - This method reduces heat loss and facilitates rewarming of the patient.
  - Crude techniques to actively warm the patient include hot water bottles, water-filled gloves, home-made wheat bags and microwavable disks but all these should be used with extreme caution and should never be placed in direct contact with the patient as not to cause thermal injury. This is due to vasoconstriction which decreases blood flow and doesn't allow redistribution of heat away from point of contact.
  - Administration of warm fluids (at body temperature)
  - More sophisticated (and safer) devices are available and these include circulating warm water blankets (older technology – can cause thermal injury readily), resistive heat pads (HotDog®), and forced warm air devices (Fig. 4) (Bair Hugger®, Cocoon®). Forced warmed air devices function as a unit with the blankets and should be used as such.

The use of incubators (for small and exotic patients)

and heating lamps can be used, but care should be exercised with the latter.

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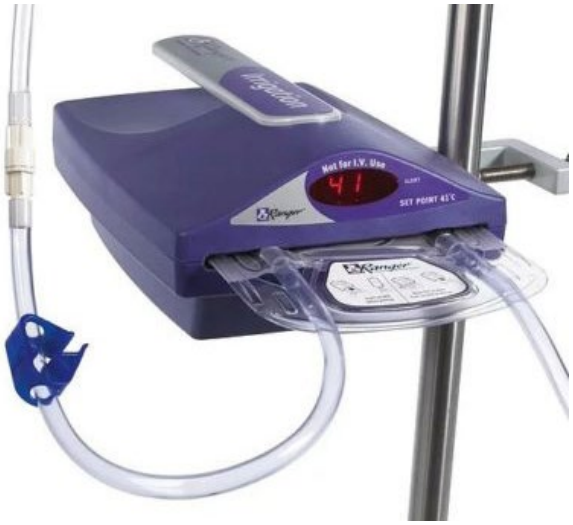

Fig. 01 - Inline fluid warming pumps

Fig. 03 - (HotDog®) Resistive heat pads

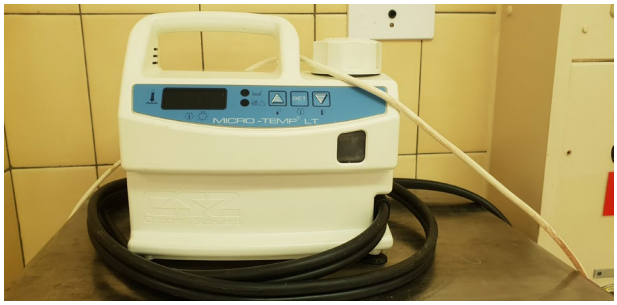

Fig. 02 - Circulating warm water blankets

Fig. 04 -Bair Hugger® - forced warm air devices

Possible IMHA Dog- Now What?

If immune-mediated heamolytic anaemia makes you crazy, you are not alone. Here's the lowdown on diagnosing this confounding disease.

Carla Johnson, DVM

Even after many years in practice, the dog with immune-mediated heamolytic anaemia (IMHA) can still leave us baffled. Should we refer to a specialist, transfer to emergency, or send the dog home on medication? When should we recheck—tomorrow, in a week, in a month? What drugs do we pick? Or should we just run screaming from the building?

IMHA cases usually start out in the emergency room, but sometimes they are caught early during routine bloodwork or the patient presents in general practice with mild early disease. A better understanding of how to manage these cases will help with early diagnosis, catching relapses and even managing them on our own. You don't need to become an expert to get the ball rolling on a good diagnostic and treatment plan. At a recent Fetch dvm360 conference, Elisa Mazzaferro, MS, DVM, PhD, DACVECC, shared her approach to IMHA cases.

This is the first of two articles on this topic. Here, we discuss the causes and diagnosis of IMHA. The second article will shed light on best practices for short- and long-term therapy as well as monitoring IMHA patients.

Disease background

IMHA is a disease in which the body attacks its own red blood cells (RBCs). It is among the most common immune-mediated diseases that we see in dogs.[1] IMHA has a 30% to 70% mortality rate despite aggressive therapy, Dr. Mazzaferro noted, and relapse rates range from 11% to 15%.[2,3] Upon initial diagnosis, Dr. Mazzaferro said she tells her clients that their heamolysing pet has roughly a 40% to 50% chance for long-term survival, then she customizes this prognosis based on follow-up diagnostics and response to treatment. Many patients do not make it through the initial heamolytic crisis, but those that do can die later as a result of a thromboembolic disease, renal failure, liver failure, relapse or complications of medical therapy. Dogs with concurrent immune-mediated thrombocytopenia, hypoalbuminemia or very rapid heamolysis can have a significantly worse prognosis.

The median age at presentation is 6 years. Although most cases of IMHA occur in female dogs, males statistically have a worse prognosis. Cocker spaniels, sheepdogs and poodles, among others, are particularly susceptible. IMHA in susceptible breeds is usually far more severe and requires more rapid, aggressive treatment than that in non-predisposed breeds. Pit bull terriers are predisposed to Babesia and have secondary IMHA from this.

Intravascular heamolysis is less common than extravascular heamolysis. The difference between the two—besides the site of RBC destruction—is that with intravascular heamolysis you must be sure to rule out zinc toxicity (and some of the infectious diseases), and you should expect the clinical progression to be more severe, with a higher risk for disseminated intravascular coagulation (DIC). You still treat these patients the same way, but be prepared to be more aggressive, and don't forget to address the thromboembolic and coagulopathy risks using medication and coagulation testing.

Seventy percent to 80% of canine IMHA cases are idiopathic,[4] known as primary IMHA. Secondary IMHA is caused by an immunoglobulin (Ig) M or IgG antibody attack against RBCs in response to infection, neoplasia, an inflammatory condition, drug or toxin, and maybe vaccination. Some toxicities cause direct RBC damage, which can be mistaken for IMHA but is not immune mediated.

Following are potential underlying causes of secondary IMHA:

- **Infection:** *Babesia, Anaplasma, Mycoplasma* spp, *Ehrlichia*

- **Drugs:** Nonsteroidal anti-inflammatory drugs, sulfonamides, cephalosporins
- **Vaccinations:** There is still not substantial evidence that vaccines can cause IMHA, but we suspect that they might, Dr. Mazzaferro said. There is a 2% to 26% incidence of vaccine-implicated IMHA depending on which study you look at,[5] she pointed out. It is also not at all clear what the duration from vaccination to onset of IMHA is supposed to be.
- **Neoplasia:** Any malignancy is potentially causal, either directly or indirectly. In patients that are significantly hypoproteinemic, be sure to rule out erythrophagocytic histiocytic sarcoma, Dr. Mazzaferro said.
- **Toxins:** Zinc, onions and large amounts of garlic (although these toxicities are not truly immune mediated, they cause Heinz body anaemia)
- **Other infections or inflammatory conditions** in the body that can overstimulate the immune system (i.e. cholecystitis, pyelonephritis, pancreatitis)

Diagnosing IMHA

Clinical presentation and history

Clinically, patients with IMHA present with lethargy, weakness, vomiting, diarrhea, pale or yellow gums, collapse, discolored urine and petechiae if they have immune destruction of both RBCs and platelets. Alternatively, patients can appear clinically normal with just spherocytes and a mild anaemia on routine bloodwork.

They can have a regenerative or non-regenerative anaemia. We are encouraged to see regeneration, but it takes the body four to five days to mount a significant regenerative response, so about 30% of cases are nonregenerative on presentation. Spherocytes are present in only 67% to 94% of dogs with IMHA.[6] They are not pathognomonic to IMHA, but spherocytes are great markers for diagnosing and monitoring this disease. Very few other things cause this.

History is very important. Ask whether the dog is or has:

- Been traveling?
- On adequate flea and tick prevention?
- Taking any drugs or supplements?
- At higher risk for tick exposure?
- Had recent vaccinations?
- Been exposed to onions or garlic?
- Eaten something like a coin or other zinc-containing toxin?

Testing

The baseline diagnostic workup is more extensive than for most diseases and includes the following:

- **Routine testing.** Complete blood counts (CBCs)

Slide agglutination testing

This type of test can be tricky. Here's how to do it right.

Place one drop of blood from a purple-top tube on a slide, visualize it for clumping (agglutination), then add four tiny incremental drops of 0.9% saline with a needle-on syringe to the edges of the blood drop to see if the blood cells disperse or stay clumped. If they stay clumped, the result is positive.

Always look for macroagglutination first (you can see it with your own eyes). If macroagglutination is negative (RBCs either do not clump or disperse quickly with saline), then examine for microagglutination. Microagglutination is the same test but under a microscope, wet and unstained. Use a coverslip, and look for RBCs clinging or clumping together.

For both tests, if the blood cells disperse evenly with the saline, the result is negative. For the microagglutination test it is even more important to use the saline to differentiate agglutination versus rouleaux. Assess the slides immediately; letting the blood sit on the slide for even a few minutes can give the appearance of false-positives as the edges dry out.

If test results are unclear, the ACVIM consensus guidelines recommend washing the blood sample and retesting. Agglutination is caused by cross-linking of IgM immune complexes across RBCs, so it should still be happening after washing.

Getting a platelet estimate

To do a platelet estimate, count the number of platelets in each of 10 random views on high-power field (100x oil immersion) near the feathered edge. Add them up, divide by 10 (basically the average number of platelets per high-power field), and then multiply by 10 to 15. That is your platelet estimate range in thousands.

Now look at the feathered edge. If there are huge clumps of platelets, factor these in as best you can as they will falsely lower your total estimate.

and chemistries, a urinalysis with culture and minimum inhibitory concentration; a pathologist's review of your CBC is a must.

- **Packed cell volume/total solids (PCV/TS; in-house)**. Note that the PCV is more accurate than hematocrit because agglutination can interfere with calculated hematocrit.
- **Slide agglutination test (in-house)**. (See Slide agglutination testing)
- **Coombs test.** If both macro- and microagglutination tests are negative, do a Coombs test. If your patient is already auto-agglutinating, a Coombs test will not give you any extra information. (Nobody is going to fault you, though, if you send out a Coombs test because you are unsure of your in-house agglutination test findings. However, there is no good substitute for a slide agglutination test, or for the immediate information that it gives you if it is positive.)
- **Blood smear (in-house)**. It should be a smear with a beautiful feathered edge, dried and stained with Diff-Quik. You are looking for reticulocytes, spherocytes, shistocytes and ghost cells in a monolayer near (but not at) the feathered edge—all of which are consistent with IMHA—and using this to do a platelet estimate while you are waiting for the CBC from the lab. (See Getting a platelet estimate)
- **Radiographs** for metastatic neoplasia or metallic foreign body ingestion.
- **Abdominal ultrasound** to complete your screening for neoplasia.
- **Infectious disease panel**. Always send out an infectious disease panel.

(ED: Escpecially consider tick-borne diseases such as babesiosis, ehrlihiosis and therleriosis, PCR panels are available)

- **Lactate**. This may be of limited availability except at emergency or specialty hospitals. If the lactate level is elevated, your patient may need a transfusion, as elevated lactate can indicate that too little oxygen is getting to the cells (oxygen debt).

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What Lives in the Dog's and Cat's Gut?

Forgotten world of microbiome

Iveta Becvarova, DVM, MS, DACVN
Director, Global Academic & Professional Affairs, Hill's Pet Nutrition Inc.

Have you been wondering why the word microbiome has been increasingly popular topic in human and veterinary medicine? This is because scientists began to uncover that the microbes living on and in the body are not just a random cluster of germs originating from the environment that can make an individual sick. Nor are the gut microbes only important to herbivores to help them extract the energy from food. In fact, gut microbiota is a living ecosystem of commensal microorganisms that provides lots of benefits to every mammal from maintaining gut health to regulating distant organs. In return, the mammalian host (dog or cat) provides the microbiota with nutrients and stable environment. Research is clear that harboring a healthy microbiota in the gut offers many health benefits to dogs and cats.

What is the size of gut microbiota of dogs and cats?

Gut microbiota is the entire habitat of living microorganisms, including not only anaerobic and aerobic bacteria but also archaea, fungi, protozoa, and viruses. They have been evolving for billions of years and there are thousands of diverse species known. It is estimated that the intestine of mammals contains approximately 1010 to 1014 microorganisms which is 10 times more than that number of cells within the body.[1] Due to the small size of microbes and presence in high density, the microbial mass is not as large and heavy when compared to the rest of the body. The total bacterial counts increase along the gastrointestinal (GI) tract of dogs and cats, with numbers being lowest in the stomach and highest in the colon.

What benefits do dogs and cats get from the microbiota?

Traditionally, the gut microbiota are known to be a key player in digestion of foregut and hindgut fermenters like ruminants, horses, or rabbits, helping them to extract energy from otherwise indigestible feed by fermentation. In fact, ruminants are totally dependent on microbial fermentation to survive. Although the energy derived from fiber fermentation in omnivores and strict carnivores is much less, dogs and cats have capacity to ferment undigested material in their colon for their benefit, including pathogen resistance, modulation of immune system, and synthesis of nutrients. Additionally, even in a true carnivore like the cat, microbes inhabiting the feline hindgut have been shown to be very active and have fermentative functions similar to those found in dogs, pigs, and humans.[2]

Where do dogs and cats get their microbiota?

Puppies and kittens are born virtually without GI microbiota and their intestines are colonised by microorganisms from the birth canal and from the environment within 24 hours from birth. Those microbes are necessary to the newborn to establish an oral tolerance to commensal bacteria and food antigens, which ensure that gut immune system will ignore them and will not initiate an inflammatory response. Throughout life, a balanced intestinal ecosystem continues to collaborate with the immune system and among multiple other roles, serves as the defense against invading intestinal pathogens.

Is the gut microbiota of dogs and cats unique?

While microbiota of individual dogs and cats have similar functions, recent studies conducted have shown that each dog and cat harbors a very unique and individual microbial profile.[3,4] The main difference is in bacterial species and strains with minor overlap between individual animals. In one study, 84% of cats harbored Bifidobacterium species but only a minor percentage of cats harbored the same species of Bifidobacteria.[4]

In spite of existing differences in microbial species between individual dogs or cats, the metabolic end products formed do not markedly differ. This is because several members of microbial community

are able to perform similar functions, and if one microbial group is displaced, other members of the community are capable of substituting that function and stabilizing the entire ecosystem. As a result, there is a large similarity in the products of microbial fermentation among individual dogs and cats, in spite of, large differences of bacterial species involved in the process. Using an analogy, it doesn't matter what kind of builders construct the house (or what bacteria do the work), as long as the house (or products of microbial metabolism) is built.

Why does gut microbial ecosystem matter to dogs and cats?

Intestinal microbiota play an important role in the health of dogs and cats. Most of its action is located in the colon where the flow of ingesta slows down, which provides the ideal environment for the microbes to grow and multiply. Microbes are excreted with each defecation which means that this ecosystem is in a dynamic and constant state of turnover, growth,

Examples of functional contributions of the gut microbiota

Function	Products	Benefits of products
Harvest of otherwise inaccessible nutrients and energy from the diet	Production of short chain fatty acids (SCFA; acetate, propionate, butyrate)[2]	Acetate and propionate are energy substrates for microbial growth but are also absorbed from the colon and provide source of energy for the body. Butyrate is important energy fuel for colonocytes. SCFA facilitate the absorption of sodium and water in the colon.
	Release and metabolism of fiber-bound plant polyphenols[5]	Polyphenols are molecules from fruits and vegetables with antioxidant and anti-inflammatory properties.
Synthesis of vitamins	Microbial synthesis of vitamin K and water soluble B vitamins[6,7]	Vitamin K is a fat soluble vitamin involved in blood coagulation and in binding calcium in growing bones. B vitamins are a class of water-soluble vitamins that play important roles in cell metabolism and help the body to utilize energy from food.
Development and activity of the immune system	Intestinal bacteria present early in life is necessary to establish oral tolerance to commensal bacteria and food antigens[8,9]	In healthy animals, the microbiota and the immune system maintain a balance so that excessive immune and inflammatory responses are avoided.
Protection against harmful microbial species	1. The microbiota induces gut immune system to produce IgA[10] 2. The microbiota stimulate intestinal secretory cells to produce antimicrobial compounds (beta-defensins, cathelicidins, bactericidal/ permeability-increasing protein and chemokine[11,12] 3. Beneficial bacteria competitively exclude pathogens by occupying receptor sites, competing for space and nutrients[13]	Ig A limits local epithelial bacterial colonisation and prevents penetration of bacteria through the epithelial layer. The secreted antibacterial activity is confined to the mucus layer, which provides physical and antibacterial barrier while allowing the presence of luminal microbiota[14]
Improved integrity of the mechanical mucosal barrier	The microbiota stimulates the mucus layer[15] and alters the mucin chemical composition[16]	Mucins (highly glycosylated macromolecules) form the first barrier between the gut contents and epithelial cells, protecting them from direct contact with commensal bacteria and their components.

and replenishment. The balance of this ecosystem is established via various cooperative strategies that microbes developed and through this dynamic process, thus, the microbial community is capable of competitively excluding potentially pathogenic bacteria.

What are some problems associated with an unhealthy gut microbiota?

Most of the commensal bacteria in the canine and feline gut are symbiotic; however, after translocation through the mucosa or under specific conditions, such as immunodeficiency, commensal bacteria could cause pathology. A state when the bacterial populations within the GI tract become imbalanced is called dysbiosis. Clinical disturbances of dysbiosis include indigestion, anorexia, diarrhea, and malabsorption of nutrients[17] Dysbiosis in dogs and cats has a number of causes and has been associated with both acute and chronic GI diseases[18-21], antibiotic administration[22], or can be diet-induced.[23] In people, dysbiosis is also associated with atopy, obesity, liver disease, or GI malignancy. Conversely, normal gut microbial equilibrium is associated with health.

Evidence is accumulating to consider the gut microbiome as a central player in the gut-kidney axis. Some microbial products, such as advanced glycation end products, phenols, and indoles, are uremic toxins which are absorbed into the circulation and cleared by normal-functioning kidneys. In kidney disease, these products can accumulate and may contribute to the uremic load and increased morbidity.[24]

Bacteria in the gut also play a role in whether or not an individual becomes obese. This is linked to the ability of the intestinal biome to extract additional energy from undigested food and to regulate energy expenditure and storage.

Evidence increases that there is a gut microbe and the brain communication. Neuroscientists are probing the idea that intestinal microbiota might influence brain development and behavior, as well as anxiety. More research is needed to investigate this relationship in dogs and cats.

What is the best way to maintain gut microbial health in dogs and cats?

Microbial growth in the gut is influenced by multiple factors but the major determinant is the substrate for their nourishment, such as dietary fiber, residuals of undigested food, sloughed mucosal cells, or enzymes released into the gut lumen. Altogether, gut microbes use these substrates for their own benefit and growth. In return, they manufacture and release number of compounds called postbiotics, with many of them providing benefits to the host.[25] Gut microbiome of dogs and cats can be modulated with diet composition, antibiotics, prebiotics, probiotics, or synbiotics. Prebiotics, probiotics, or synbiotics can be administered as dietary supplement or added to the commercially formulated pet food. Since dogs and cats have to eat every day, choosing a complete and balanced foods specifically designed to promote healthy microbial fermentation is the most practical way to nourish healthy gut microbiota from day to day. Inclusion of prebiotic fibers into the food offers the most practical and lasting strategy to positively influence the intestinal microbiome. Not every prebiotic is created equal. Fibers with prebiotic benefits commonly found in pet foods include beet pulp, flaxseed, fruit fiber, rice fiber, fructooligosaccharides, inulin, oats, or barley.

Conclusion

Traditional viewing on gut microflora in veterinary medicine focused mainly on the role of microbes to ferment herbivorous diet and help herbivorous animals to extract energy from poorly digestible plant matter. However, research has shown that even omnivores and true carnivores like dogs and cats do benefit from balanced ecosystem in their gut. Gut microbiota is an important living ecosystem within the body, which influences both gut health and extra-intestinal organs. Gut microbes metabolize and ferment substances that travel to the hindgut in the form of undigested substrate. The composition of the intestinal microbiota ecosystem and postbiotics produced are strongly affected by dietary patterns. Composition of the diet and inclusion of prebiotic fiber represent a long-lasting strategy to consistently maintain balance of gut microflora and deliver health benefits.

Key Messages

- Microbiota of mammals contains trillions of bacterial cells, 10 times more cells than the number of cells constituting the body.
- Gut microbiota influences many areas of dog and cat health from nutritional benefits, modulation of immune system, and protection of host against pathogens.
- The benefits of microbiota to the host are mediated through postbiotics, or products of microbial metabolism with biological effects.
- A dysbiotic state of the gut microbiota is associated with inadequate diet and in pathological conditions of gastrointestinal tract in dogs and cats.
- Microbiota of dogs and cats should be considered a key aspect of animal health and nutrition.
- Prebiotics in the diet produce lasting and positive impact on microbiota with a number of health benefits.

GLOSSARY

Commensal microorganisms - live in a relationship in which one organism derives food or other benefits from another organism without hurting or helping it. Commensal bacteria are part of the normal gut flora.

Microbiome – the collective genomes of the micro-organisms in a particular environment.

Microbiota – the community of micro-organisms themselves.

Short chain fatty acids – fatty acids with two to six carbon atoms that are produced by bacterial fermentation of carbohydrates and dietary fibers.

Dysbiosis – a term for a microbial imbalance or maladaptation on or inside the body, such as an impaired gut microbiota.

Prebiotic – a dietary prebiotic is an ingredient selectively fermented that results in specific changes in the composition and/or activity of the gastrointestinal microbiota thus conferring benefit(s) upon host health.

Probiotics – live bacteria and yeasts that, when administered in a viable for and in adequate amounts, are beneficial to animal health.

Postbiotic - a relatively new term that refers to the metabolic product or byproduct secreted by live commensal bacteria, or released after bacterial lysis (e.g., enzymes, peptides, polysaccharides, cell surface proteins, organic acids) which are responsible for many of the biological effects.

Synbiotics – contain a mixture of prebiotics and probiotics.

Polyphenols – naturally occurring micronutrients found in plants with anti-oxidant and anti-inflammatory activity when consumed in the diet. Some are fiber bound and unable to be used by the host until cleaved by enzymes held only by resident bacteria in the colon.

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Advanced Dental Procedures for Pets: What's Possible?

Flap surgeries, root canals and orthodontic techniques are all legitimate alternatives to extraction in many veterinary dental cases. We owe it to our clients to let them know their options.

Benita Altier, LVT, VTS (dentistry)

As veterinary practices have become more modern, sophisticated and technologically advanced, so has our ability to perform veterinary dentistry at a much higher level than previously thought possible. As veterinary professionals, we need to be fully aware of the dental treatments available and understand when to offer a referral for advanced dentistry.

In this article, we'll cover some of the many options for advanced dental care for pets. Let's get started.

Preparing for advanced periodontal therapy

Statistics show that periodontal disease is the most prevalent dental disease in dogs and cats. Left undiagnosed and untreated, it can progress until there is no other option but to extract affected teeth. However, when dental disease is diagnosed early, teeth can be salvaged through periodontal surgical techniques and home care, and patients can benefit over the long term by retaining important teeth for their function.

Larger and more essential teeth can be difficult to extract even when affected by significant periodontal disease, which can result in horizontal or vertical bone loss, furcation (the area between a multi-rooted tooth's roots) bone loss, and tooth mobility due to loss of attachment. When we examine teeth and the surrounding bone through clinical observation and full-mouth dental radiographs, we must assess the true extent of the pathology, noting bone loss, attachment loss, and tooth mobility.

Radiographs and clinical findings help the practitioner determine which teeth are good candidates for therapy, and when extraction is the best option. We can evaluate a tooth on a root-by-root basis as well as examine individual sides of each tooth root. A tooth with significant bone loss (>50%) on the root's surface may have a poor prognosis even with advanced therapy, especially if the bone loss is around the root, or what's called a four-walled defect.[1,2] If the bone is lost from the furcation, this reduces the chances for success even further.[1,2]

Evaluating total attachment loss

Total attachment loss is the sum of the measurement of any gingival recession on the root's surface, as well as any pocket depth beyond that recession. If gingival recession is not present, then it's just the measurement of any periodontal pocket depth beyond what may be considered a normal sulcular depth for that specific tooth in that particular pet's mouth.

Normal sulcular depth ranges from 0 to 1 mm; however, "normal" can differ depending on the size of the animal, size of the tooth and length of the tooth root.

A periodontal probe with clearly marked 1-mm increments is used to measure from the marginal gingival edge to the bottom of the sulcus or periodontal pocket if there is attachment loss.[1,2] The bottom of the sulcus is attached to the tooth's surface at or very near the cemento-enamel junction.[1] Attachment loss at this point creates a periodontal pocket and a pathological process.

Using a gentle hand and holding the periodontal probe in line with the vertical axis of the tooth, the clinician walks the probe around the tooth's structure and takes measurements in four to six places around each tooth root. Whenever these measurements are greater than what would be considered normal for that particular tooth, they're recorded on the patient's dental chart.[1]

Note that conditions such as gingival enlargement can create a false pocket depth and not actual attachment loss, so excess gingival tissue must be measured carefully; take note whether the bottom of the sulcus is at the cemento-enamel junction to determine the extent of attachment on these teeth.[1]

If the combined loss of bone and soft tissue attachment is less than 50% and the furcation loss of bone is less than halfway through, it may be possible to "save" important or strategic teeth through advanced periodontal surgical techniques, frequent follow-up care (possibly under anaesthesia) and a commitment to daily home care by the client.

For a periodontal pocket depth greater than 5 mm, it's recommended that open root planing and subgingival curettage be performed with the use of flap surgery to facilitate visualization of the bony defect and exposed root surface (more on flap surgery to come). Open root planing allows the practitioner to treat the area to the best of her ability to get the best possible outcome from periodontal therapy.[1]

Obtaining dental radiographs

Besides measuring attachment loss, the veterinary team must obtain radiographs to assess the extent of any suspected bone loss. Evaluating a full set of intraoral dental radiographs will help determine the success of any proposed procedure and also provide a baseline for monitoring treatment progress. If your veterinary practice cannot obtain those dental radiographs and the client is interested in advanced dental care and saving teeth rather than extraction, consider a referral from the onset—it may be in the best interest of the patient.

Advanced periodontal flap surgeries

Techniques to perform flap surgeries are fully described in several dental textbooks and can be learned at labs taught by veterinary dentists on the subject. However, if surgical procedures that are beyond the practitioner's skill level are indicated, referral may be the best option.

Apically repositioned flap

This technique can be used to help the attached gingiva lie over any remaining alveolar bone; it requires at least 2 mm of gingiva to extend toward the crown.[1] This surgery moves the gingiva down onto the root surface after the area is cleaned of unhealthy bone, granulation tissue and debris; then the area is allowed to heal.[3] This procedure can be performed on mandibular incisors to allow for a reduction in periodontal pocket depths, easier cleaning of furcation exposure areas on multi-rooted teeth, and daily cleaning by the client.[3]

Contraindications include more than 50% bone loss (especially on a four-walled defect), grade 3 tooth mobility and the presence of less than 2 mm attached gingiva before surgery.[1]

Laterally positioned (pedicle) flap

When the root surface of a single tooth is exposed significantly due to a cleft of bone and soft tissue loss that extends to or near the muco-gingival line, a laterally sliding flap surgery may be indicated.[1] This procedure requires carefully planned and executed vertical releasing incisions, as well as the creation of a donor flap that's moved laterally over the area and sutured.[1] The goal is to partially cover the exposed root surface and allow for at least 2 mm of attached gingiva to help preserve the health of this particular tooth; the area of exposed tissue from the donor site will heal by second intention.[1,2]

Contraindications for this procedure include tooth mobility due to loss of bone on more than one wall of the alveolar socket, furcation bone loss and lack of commitment on the client's part to daily home

care and more frequent follow-up professional dental care.[1]

Free gingival graft

This technique is indicated in specific individual teeth with a cleft-like defect that is free of endodontic disease and where tooth mobility is not present.[2] A gingival graft is obtained from a donor site separate from the site to be treated, often the buccal surface of attached gingiva over the maxillary canine—this site offers the largest expanse of tissue.[2] The donor graft is harvested carefully using a template, employing a careful technique to avoid damage to the periosteum.[2] The donor tissue is then carefully grafted over the recipient site using specific surgical techniques.[2]

This procedure is contraindicated if endodontic disease is present and not treated first.[2] Concurrent periodontal disease must also be treated and controlled; if there is tooth mobility, this technique is unlikely to have a good outcome.[2] Success will also depend on the client's willingness to comply with daily recommended home care and follow-up veterinary treatment.[2]

Guided tissue regeneration

The goal of guided tissue regeneration is to help facilitate the development of cementum on the root's surfaces and the regeneration of healthy periodontal attachments.[1] Once the tooth root surface has been cleaned and treated, barrier membranes that are either absorbable or non-absorbable are positioned to prevent granulation tissue from invading the area. The goal is to encourage bone induction behind vertical bone walls and periodontal ligament cells to develop in areas where they've been destroyed by periodontal disease.[1]

The use of bone inductive materials can assist in such procedures where significant bone has been lost in two- and three-walled bony defects and areas of class 2 furcation bone loss in multi-rooted teeth.

Endodontics

Endodontics is the dental discipline that treats disease involving the internal tissues of a tooth.[1] These inner tissues are highly neurovascular and susceptible to trauma, which can cause significant inflammation and lead to irreversible damage, including tissue necrosis and death.[1,2]

Concussive injury to a tooth can cause the pulp to bleed inside the tooth into the dentinal tubules or expose the pulp to the oral cavity, as in the case of a complicated crown or crown-root fracture.[1] Other causes of endodontic disease are near-pulp exposures such as in an uncomplicated tooth fracture into the porous dentinal tubules, a carious lesion or cavity, severe abrasion or attrition, or bacterial invasion through the animal's bloodstream through the apical delta into the root canal and pulp chamber of the tooth.[1,2]

Root canal procedures are performed to retain important strategic teeth in their alveolar bony sockets to maintain function. The vital or once-vital endodontic tissue is removed and replaced with an inert filling material; the access and fracture sites, if applicable, are also filled with a composite material.[1,2] The tooth is not restored to its original height because the weakened tooth would be more prone to further damage or fracture. Annual monitoring is recommended after root canal treatment, and dental radiographs should be obtained to evaluate the continued success or failure of the procedure.[1,2]

Restoration of teeth affected with carious lesions

True carious lesions are not common in dogs and especially not common in cats; however, if they are found, they can be restored with cavity preparation and restoration material after careful evaluation of dental radiographs.[1] Some carious lesions involve the pulp and should not be restored without conventional endodontics.[1] If concurrent periodontal disease is also present, the prognosis for these teeth may be significantly worse.[1]

Restoration of enamel defects

Enamel defects can be acquired from tooth wear due to abrasion or attrition or a congenital condition that prevents enamel from forming correctly before adult tooth eruption.[4] Trauma, infection, hypocalcemia or the use of certain drugs during the enamel-forming period can also cause defects or malformations on the enamel of unerupted permanent dentition.[5]
Amelogenesis imperfecta is an inherited defect that is fairly rare in dogs and also rare in people. It is most common in species located in remote areas where the genetic pool is less diverse.[5] Hypoplastic, hypomaturation and hypocalcification are the three types of amelogenesis imperfecta.[5] Enamel hypocalcification leaves the enamel very fragile even though the depth is within normal limits; it has failed to undergo mineralization and is easily removed from the dentin below it.[5]

The goal of enamel restoration is to prevent any further destruction of the surrounding enamel and to protect the pulp and dentin from damage due to changes in temperature, bacterial invasion, and further wear or loss of enamel substance.[1] Occasionally in the case of attrition, we will choose to restore an important tooth, such as a canine tooth, and extract a less important tooth, such as an incisor, if these two teeth are rubbing on each other and causing a lesion or enamel defect.[4] If the defect is caused by forces other than another tooth, the source of that wear

must be removed to prevent further wear and loss of the restorative material on that tooth.[4]

A flowable type of composite can be used to repair areas of enamel hypoplasia or enamel defects on the crown.[4] First, the enamel defect must be prepared to accept and retain the composite material, which will help restore the tooth to a more normal contour and function.[2,4] Preparation entails the debridement of diseased or damaged enamel and contouring the edges with a pear-shaped bur used in a high-speed handpiece, cooled with water spray.[4] An excavator is used to prepare the area further.[4] An acid etchant is then used to remove the smear layer from the exposed dentin and create an environment where the restoration will bond more effectively through micromechanical interlocking.[1,4]

An unfilled resin—a bonding agent that helps the flowable composite attach more readily—is then light-cured onto the surface of the prepared defect.[4] Composite is flowed into the defect and allowed to overfill the area slightly, then cured with a special dental curing light and finished with a fine diamond bur or polishing disks, so the edges of the composite are not detectable when investigated with the tip of a shepherd's hook exporer.[4]

Enamel restoration may help increase the durability of the tooth. The client should be informed of the possibility of lost restorations, the likelihood of further treatment and the necessity of preventing the habit or behaviour that caused the defect (if applicable) to avoid further damage to these teeth.[4]

Orthodontics

Orthodontics deals with the correction of malocclusions or abnormally positioned teeth that are causing trauma to teeth or soft tissues. Trauma results in either tooth-on-tooth wear and damage (attrition) or tooth-on-soft tissue trauma. These ongoing traumas cause patient pain and can lead to tooth damage and endodontic disease.[1]

It is important to become familiar with the basic canine skull types, how these occlusions are evaluated and categorised, and what constitutes a comfortable, pain-free occlusion for our patients.

Orthodontic abnormalities should be recognised by the general practitioner early on in the pet's life. During the primary, mixed or early eruption of permanent dentition phases, the teeth should be carefully evaluated and any abnormalities noted. Persistent primary or retained deciduous teeth can further exacerbate occlusion problems.
At-risk breeds are those with jaw relationships outside the normal limits associated with mesocephalic skull types. These dogs and cats should be monitored closely for any signs of malocclusion, and early intervention through interceptive orthodontics should be performed if indicated. If the practitioner diagnoses a malocclusion, referral to a veterinary dentist may be the best option.

Common malocclusions in dogs and cats are:

Lingually displaced mandibular canines (MAL/LV).
When this occurs, the cusps of the canines are tipped too far lingually and may cause soft tissue trauma to the hard palate.[1] Occasionally this condition occurs due to the lack of space, or diastema, between the lateral maxillary incisors and the maxillary canine teeth either bilaterally or unilaterally.

Rostral crossbite (MAL/CB/R)
In this condition, some or all of the mandibular incisors are positioned in front of or rostral to the maxillary incisors rather than the preferred scissor bite.[1]

Caudal crossbite (MAL/CB/C)
When one or more of the mandibular premolars or molars occlude buccally to the maxillary teeth above them, a caudal crossbite has occurred. A more extreme example is when the maxillary fourth premolar occludes palatal to the first molar of the mandible on that same side of the mouth.[1]

Level bite (MAL1)
In this condition, the maxillary and mandibular incisors occlude right on top of each other, causing attrition to the crown cusps over time.[1] A malocclusion could also occur when the maxillary premolars occlude with the mandibular premolars on the same side of the mouth, which can cause potential problems with mouth closure and pain.[1]

Mesioversion of maxillary canine teeth (MAL/MV)
In these patients, the maxillary canine teeth are tipped too far forward, causing a reduction in the diastema between the maxillary third premolar and the maxillary canine tooth on that side.[1] The crown tip can also cause trauma to the patient's lip.

Orthodontic movement should not be undertaken by untrained professionals, but a basic understanding of occlusion can assist veterinarians in making recommendations for treatment rather than disregarding these conditions as unavoidable and untreatable. Early and careful intervention may be required even when only primary dentition is involved in preventing more complicated and costly orthodontic intervention later on in life.

Summary

Keeping the patient's best interests in mind helps veterinary teams make the proper observations, and then recommendations, for advanced dental treatment and procedures. Developing a working relationship with a referral veterinary dentist can be very helpful as advice and insights are exchanged via consultations on specific cases. Having a financial quote ready for clients who choose referral for more advanced treatment options will also be important when they need to make treatment decisions. Open communication with clients increases their understanding of your findings and diagnoses and helps them feel more comfortable with your recommendations and referral.

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Benita Altier LVT, VTS (dentistry), is the owner of Pawsitive Dental Education in Easton, Washington, and a frequent speaker for the Fetch dvm360 conferences.

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[1]Hill's data on file. Two-month clinical study evaluating dogs with chronic diarrhoea. [2]Hill's data on file. Clinical study on microbiome changes in dogs.

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