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Editor's Note

This edition is full of practical advice regarding the management of otitis externa - a really frustrating condition in some patients. Basically it boils down to treating the underlying cause and not just the symptoms. There are also guidelines on when to admit defeat and recommend a total ear canal ablation.

There are also some exciting new developments on the clinical pathology front with the launch of the IDEXX SDMA™ renal function test. New literature on the the Spec cPL™ and SNAP™ tests are also evaluated and information regarding their diagnotic sensitivity and specificity is summarised.

This was an interesting and challenging issue to edit.

I hope you enjoy it.

Regards

Liesel

Index:

PREVIOUS EDITION: February 2016
- Canine Idiopathic Dilated Cardiomyopathy
- General Anaesthesia in Patients with a Heart Murmur
- Employee Relations within a Vet Practice - Part 2

NEXT EDITION: June 2016
- Breed Specific Hepatopathy in Scottish Terriers
- Ultrasounding of the Gall Bladder
- Diagnosis of FIP in Cats

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Part 3

Employee Relations Within A Vet Practice

Andrew Christie
BComm (Industrial Psychology)

This article is the third in a series of three which deal with maintaining positive and mutually fulfilling employee relations within a Vet Practice.

The first article dealt with the start of the employment relationship (recruitment, selection and appointment); the second explored the managing of the employment relationship (performance management, the grievance procedure and the creation of an HR policy) and this, the third one, will examine the end of the employment relationship (resignations and dismissals, as well as the disciplinary procedure).

The employment relationship can be terminated for various reasons:

a. On completion of agreed period or task
b. By mutual agreement
c. By notice
d. On grounds of impossibility of performance
e. Because of insolvency

1. Dismissal

Fair versus Unfair Dismissal

The following are considered by the labour courts to be "fair" reasons for the termination of the employment contract:

- Misconduct
- Incapacity to perform job
- Operational requirements of the business

While most other reasons for dismissal will be unfair, the following are the automatically unfair reasons that apply to a vet practice:

- A worker is pregnant or intends to become pregnant
- An employer discriminated against a worker because of race, gender, sex, ethnic or social origin, colour, sexual orientation, age, disability, religion, conscience, belief, political opinion, culture, language, marital status or family responsibility
- An employer cannot prove:
  - a worker's misconduct or inability
  - that the employer's operational needs are valid
  - that the dismissal procedure was fair
- Any other reasons

Dismissal for Misconduct

The following are grounds for dismissal for misconduct:

- The accused employee did commit the misconduct
- The misconduct was serious enough to merit dismissal
- The employee knew or should have known that the incident constituted misconduct
- The rule or standard was valid or reasonable
- The rule has been consistently applied in previous cases.
- If the dismissed employee takes action against the vet practice, you will be asked the following:
    - Were there any mitigating circumstances?
    - Can you prove that the employee's conduct made a continued employment relationship impossible?

The above refers to the "substance" of the dismissal; however the dismissal must also be "procedurally" fair. Procedural fairness refers to following the correct procedure when effecting the dismissal.

This procedure is outlined by the labour legislation, as well as the vet practice's own disciplinary code. It includes holding a disciplinary hearing, allowing the employee to appeal, etc..

Dismissal for Incapacity

Incapacity can arise from two places; first, a newly appointed employee shows that they are unable to perform their job adequately or, second, when injury or ill-health prevents an employee from performing their duties.

As discussed in the first article in this series, recruitment is the foundation on which employee relations is based. This is very evident in the case of dismissing for poor work performance as the employer will have to show the following:

- The employee was given appropriate evaluation, instruction, training, guidance or counselling.
- The employee continues to perform unsatisfactorily after a reasonable period of time for improvement has been given.

This means that the probationary period should be seen as a time for skilling the employee, rather than just "checking them out".

Incapacity due to ill health or injury can be temporary or permanent

- Temporary: The vet practice needs to assess the extent of the illness or injury as to whether the employee will be absent for an unreasonably long period of time. If so, the vet has to explore all possible alternatives to dismissal. For example, if an employee will be absent from work for 3 months due to illness, an alternative to dismissal could be employing someone on a temporary basis.
- Permanent: Before considering dismissal, the vet has to look at whether the employee's workplace can be adapted to accommodate their disability or, alternatively, whether the employee can perform another job within the practice. For example, in the case of an practice manager who becomes wheelchair-bound, the vet would have to investigate making her office easily accessible to the wheelchair.
- In the case of injury or sickness caused by their job, even more accommodation should be made.

One particular area to be aware of is that, while being drunk can be a dismissable offence, alcoholism and drug addiction can be seen as illnesses and therefore counselling or some other intervention should be considered before dismissal.

Dismissal for Operational Requirements

Because small to medium sized businesses have dismissed unproductive employees by saying "We just can't afford to keep you on" to avoid disciplinary action, the labour legislation is very specific about what constitutes dismissal for operational reasons and how it must be conducted. Dismissal for operational requirements are categorised as "no fault" dismissals as they are not in any way linked to the performance of an employee. As they are "no fault", the legislation requires that businesses explore all possible alternatives to dismissal are explored.

To this end, the process should be collaborative, between employer and employee, as far as possible.

To achieve this collaboration, the process must include:

- The opportunity to meet and report back to employees
- The opportunity to meet with the employer
- The request, receipt and consideration of information

Of course, in a vet practice, the collaborative process is often limited to just two people – the practice owner and the person being retrenched. Nevertheless, the process must still be collaborative.

Vets should also be aware that they might have to justify why they selected the person(s) for retrenchment. Apart from a job becoming redundant, selection criteria that would be fair include length of service, skills and qualifications. The courts will generally accept the use of the "last in first out" (LIFO) principle.

An employee who is retrenched is entitled to a MINIMUM of one week's salary per completed year of service at the practice. They must additionally be paid out for unused annual leave and either be paid one month's notice, or be given unlimited time to find a new job. If the employee rejects a reasonable alterna-

tive position at the practice, they do not have to be paid a severance package.

2. Disciplinary Code

In the previous section, dismissal for misconduct was outlined. However, it is important to remember that dismissal in these circumstances should only be used as a last resort. A formal disciplinary code will provide guidelines as to how deal with discipline in the workplace, ensuring fairness as well as preventing unwanted comebacks by disgruntled employees.

The purpose of a disciplinary code, then, can be summarised as follows:

- To ensure that all employees conduct themselves properly in the interests of workplace harmony, safety and effectiveness.
- To guide employees and management as to the conduct expected and the appropriate corrective measures

The Reasons for Disciplinary Action

While the reasons for disciplinary action being taken are almost limitless, they can all be summarised as follows:

- A vet practice must have certain rules and regulations to enable it to carry out its activities in an orderly and meaningful way.
- To achieve this, it is essential that all employees are aware of the behavioural norms and standards expected of them.

The above will only be successful if the disciplinary policy and procedure are applied fairly and consistently.

The Aims of a Disciplinary Code

When creating and applying a disciplinary code, rather than seeing it as a guide to punishment, the code should be centred on the following

- Rehabilitation
  Correction and rehabilitation should be the cornerstone of good discipline and is prescribed before harsh penalties are unnecessarily applied.
- Deterrence
  For example, despite counselling, failure to improve poor attendance may ultimately lead to dismissal, thus acting as a deterrent to other employees.
- Prevention
  This is achieved via progressive discipline because warnings identify and give employees the chance to stop the offending misconduct.
- Fair Discipline
  The LRA, backed up by the CCMA, Labour Court and Industrial Court decisions made of the past few years, have stressed that disciplinary action must be both substantively fair, as well as procedurally fair.
  - Procedural Fairness is achieved by the correct application of disciplinary steps. Adherence to disciplinary procedures helps to ensure that discipline is administered consistently, fairly and promptly.
  - Substantive Fairness is achieved by adherence to the disciplinary policy, guidelines, common practice, the laws of South Africa, fairness and consistency. This applies when determining innocence or guilt and arriving at the corrective measures.

  The LRA and the Courts require that employees must know the rules and the consequences of infringements before any penalties are applied.
- Progressive Discipline
  While some offences merit a disciplinary hearing and possible dismissal on the first offence, other infringements need to be dealt with via a process of one, two or three warnings at progressively higher levels of severity, depending on the seriousness of each specific offence. In this way, warnings accumulated on a progressive basis could culminate in a disciplinary hearing and possible dismissal. It is stressed that not all warnings may be accumulated in this way. That is, only warnings for infringements of a similar type may be accumulated in a particular line of progression.

Andrew Christie
BComm (Industrial Psychology)

After completing his studies Andrew spend 2 years at a leading bank in South Africa where he was involved in the implementation of the new labour legislation that was rolled out in 1997, as well as managerial training.

He followed this with a stint at a computer-based training company where he assisted in the development of learning centers for major South African corporations.

Over the next five years he drove the development of that shop into a successful chain of six. In 2005 he re-entered the corporate world by forming his own consultancy, specialising in providing financial, tax and human resource services for SMME's, as well as growth management for medium corporates.

Before the End

Prepare highly attached clients to face their pets' death

Sarah J Wooten, DVM

Planning for the inevitable can alleviate the complications of uncertainty and grief, allow clients to better assess quality of life, grieve in a way that honours the human-animal bond and provide you with the privilege to guide clients through what can be one of the most difficult decisions of their lives.

The importance of animals in the lives of humans has never been so pervasive or celebrated—spend two minutes on the Internet for countless examples. It seems this emotional public perception of animals as connected to humans, seen also in the aversion to animal deaths and killing, will inevitably become more emotional and more contentious.

In many ways, animals are playing a larger role in peoples' lives. People are more isolated. The growth in one-person households (people living alone) is responsible for most of the increase in non-family households over time — and the corresponding decrease in family households[1]. But people still need companionship and emotional support, and they are finding it in their pets.

This personal and emotional attachment—even a feeling of being more bonded to their pets than they are to their human family—can make end-of-life decisions for these beloved pets overwhelming. In order to guide pet owners through difficult decisions, veterinarians need to be able to navigate the relationships people have with their pets and understand the current expectations and needs that pet owners have regarding humane euthanasia and end-of-life decisions.

I recently attended a session on euthanasia and end-of-life care by Elizabeth Colleran, DVM, DABVP (feline) at the CVC in San Diego and came away with the following thoughts on this arguably most difficult aspect of practice.

"Doctor, what would you do?"

Pet owners give us remarkable trust and authority, and during loss, look to us to provide strength, guidance and leadership. Given these expectations, compassionate communication should be considered both a core clinical skill and a standard of care for veterinarians, says Colleran.

But when a client asks a veterinarian for advice, what do we do? Veterinarians usually choose from three basic options when guiding clients on end-of-life decisions:

- We deflect: "It's your decision. I can't decide this for you."
- We give options: "You can try steroids …","I can send you to hospice care …", "You can elect amputation …", "Humane euthanasia is not a bad option."
- We tell them what to do: "Your pet is suffering. You should euthanase."

All of these can be the right way to handle the question depending upon the circumstances. For the client's sake, and ultimately for our own, Colleran says we must not try to solve the owner's problems by making decisions for them, rationalising their choices or rescuing them. What becomes essential to assisting clients is our ability to educate, support, guide and facilitate during this often-difficult time.

The emotional toll

On the one hand, euthanasia is a tough subject that we face almost every day and don't talk about enough. Clients are stressed and emotional and looking to you for guidance. In one study, more than one-third of pet owners said that their major source of emotional support was their veterinarian. Mental and emotional stressors associated with euthanasia take their toll on veterinarians as well. We have a saying at our clinic: no one cries alone. I almost always cry at a euthanasia, even when I don't know the clients very well.

Euthanasia is hard

- First, recognise the moral stressors involved in euthanasia that can affect you
- Convenience euthanasia. Euthanasia for reasons we can't accommodate in our minds (e.g. "My cat doesn't match my drapes").
- Severed relationships. The client has ended the mental and emotional relationship with the pet before they have arrived, leaving nothing on which to base decisions.
- Financial constraints. The ability to afford—or a lack of desire to pay for—the care a pet needs causes a client to choose euthanasia.
- Client guilt. Veterinarians often deal with client guilt associated with euthanising too soon or causing suffering by waiting too long.
- Technology zeal. Just because we can continue to treat and diagnose doesn't mean we should, even if the client desires it.
- Inability to let go. Client won't stop or won't quit seeking treatment even when the pet's condition cannot be cured or managed pain-free.

Second, realize you have little to no control over these moral stressors, and take care of yourself mentally and emotionally.

- Take a time out.
- Talk to someone who understands.
- Learn mindfulness-based stress reduction techniques (i.e. breathing, meditation).

Uncertainty and grief

The strongest desire of highly attached pet owners facing the loss of a beloved pet is to do what is best for the animal. It is an elusive goal and one that requires the owner's interpretation of the animal's state. There is no easy answer, particularly because we advise our clients based on proxy. As the pet's proxy, the owner makes the decision for euthanasia based on uncertain anticipated events rather than what is known. Uncertainty makes the decision more difficult.

Also, highly attached owners who recognize that their pets have a life-limiting medical condition that cannot be cured or managed pain-free experience strong emotions called anticipatory grief, says Colleran. Anticipatory grief is the psyche's way of preparing for impending loss and is a normal reaction, but unfortunately it can blur judgment. This is often the beginning of a period of powerful emotions, and end-of-life planning should be done before these emotions take over, advises Colleran.

Quality of life vs. prolonging life

In euthanasia, it is best to let clients make decisions, says Colleran, but in order to do this, they need to be able to accurately assess their pets' quality of life before their judgment is impaired by emotion. According to Colleran,

> "…one core belief that must be developed in clients is that preserving quality of life takes precedence over measures to prolong life."

Medical and surgical therapy that was previously unattainable can now prolong suffering, making it more important than ever to educate clients on the importance of quality of life over quantity.

Colleran says the best practice is to teach people early on how to recognize quality of life with assessment tools and diaries, even as early as puppy and kitten wellness visits. At the end, we want to be able to remind people with that point of reference what quality of life looks like in their pet, instead of trying to teach them about quality of life during an often-emotional moment. Quality of life assessment tools give clients power and confidence by allowing them ability to quantify something that is typically unquantifiable. I use this quality of life scale.

Helping a child say goodbye

Seeing a pet euthanised can be traumatic for young children, even if it is peaceful. If there are young children involved, help them say goodbye, and advise the parents to excuse them for the euthanasia, Colleran says. Be mindful in the way you communicate with kids: children younger than 9 or 10 will not un-

derstand that "I am putting your pet to sleep" means death. Colleran advises veterinarians to use concrete language: "I am helping your pet to die because she is suffering and we can't control her pain any longer. This is really hard and sad thing for us, but it is a good and brave thing to do for your pet. It is OK to be sad."

Plan for the inevitable

Planning ahead also involves making sure that clients have access to medical records and a plan in place if something goes wrong or timing is wrong, says Colleran. In addition, it can help the client to decide ahead of time if children or other pets should be present, where the euthanasia will occur (at home, at the clinic, outside), body care and more.

We may be the only person in that client's life that recognizes the human-animal bond between the client and the pet. We can plan to honour that connection and provide emotional support for the client after the pet has passed on. Celebration of life ceremonies, telling funny stories about the pet, a funeral, special mementos, art work, music or candles can all help a highly attached client cope with the loss, so don't be afraid to ask how the client will be honouring the passing of his or her pet. If your client doesn't have any ideas, don't be afraid to give some suggestions. In doing so, you may be giving that client permission to grieve in a healthy way that he or she may not have considered. Ritual can bring comfort and closure.

By preparing the client for end-of-life decisions, helping the client plan and providing emotional support to your client, you have the opportunity to provide exceptional service and protect the human-animal bond. The manner in which a veterinarian provides care for a client whose pet has died has the potential to alleviate or aggravate grief and influence client and veterinarian satisfaction—even create or destroy long-lasting relationships, says Colleran.

Presenting options gives families control over the process of inevitable loss by helping them define for themselves what constitutes the best way to care for their animal. I believe a sense of control—even if limited—correlates with healthy grieving and emotional healing. Just as humane euthanasia is a privilege to end suffering, guiding clients through the process can be a privilege as well.

Reference

1. Vespa J, Lewis JM, Kreider RM. America's family and living arrangements: 2012. United States Census Bureau. Available at http://www.census.gov/prod/2013pubs/p20-570.pdf

The Top 5 Liver Diseases in Dogs

Craig B Webb
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The liver can be exposed to ingested toxins, blood-borne pathogens, and drugs and their metabolites, providing numerous causes for acute insult.

The liver serves as the control-and-command center for virtually all metabolic processes: production, packaging, and distribution of proteins, lipids, and carbohydrates; hormonal and enzymatic control of metabolic pathways; metabolism of biologics; transformation of xenobiotics; decontamination and removal of toxins; and recirculation, recycling, and refilling of gallbladder contents.

When the liver does not function at full capacity, clinical manifestations are often ubiquitous and possibly devastating.

1 Hepatitis or hepatic insult

Acute

The liver can be exposed to ingested toxins, blood-borne pathogens, and drugs and their metabolites, providing numerous causes for acute insult. Signs can include anorexia, fever, vomiting, and abnormal mentation. Jaundice is the classic sign of hepatic failure, with RBC haemolysis as another important differential for hyperbilirubinaemia.

Leptospirosis appears to be increasingly prevalent and usually involves both the liver and kidneys. Other possible infectious agents include infectious canine hepatitis (canine adenovirus-1), *Clostridium piliformis,* bacteria (especially *Escherichia coli)*, and *Toxoplasma gondii*. Ingestion of *Amanitum* spp mushrooms, blue–green algae, and some drugs (eg, sulfonamides, carprofen, amiodarone) can result in acute and significant liver disease. Consequences of insult may be idiosyncratic and unpredictable, and signs may vary.

ALT and bilirubin are the most pertinent biochemical indicators of hepatic insult, although low BUN, albumin, cholesterol, and glucose levels along with prolonged clotting times are indicative of fulminant hepatic failure. Supportive care is essential.

Glucocorticoids may be contraindicated (infection) or of minimal benefit in the acute setting. Acetylcysteine has been used in critical patients with a loading dose of 140 mg/kg IV, followed by additional treatments at 70 mg/kg.

Chronic

Signs of chronic canine hepatitis, often nonspecific and systemic, include vomiting, lethargy, decreased appetite, polyuria/polydipsia (PU/PD), and weight loss. Increased ALT enzyme activity, usually the most telling biochemical abnormality, is frequently monitored as a quantifiable indicator of treatment response.

Although the primary disease cause may be undetermined, several treatable causes warrant diagnostic examination with histopathology, metal analysis, and bacterial culture. Chronic hepatitis may present as a slow progression of changes started by acute insult. Culturing bile, not liver tissue, may yield better growth.[1]

There is often an immune-mediated component to chronic hepatitis progression, warranting immunomodulatory therapy. Prednisone has been the preferred drug, but Colorado State University has recent-

TOP 5 Liver Conditions in Dogs

1. Hepatitis or hepatic insult
2. Hepatic fibrosis or cirrhosis
3. Copper-associated hepatitis
4. Congenital portosystemic vascular anomalies (PSVA)
5. "Nonhepatic" hepatic disease

ly had success using cyclosporine at a starting dose of 5 mg/kg q24h.[2] Unlike prednisone, cyclosporine does not induce canine liver enzyme elevation.

Hepatitis cases (acute and chronic) can be treated with ursodeoxycholic acid at 7.5 mg/kg q12h to enhance bile flow, dilute toxic bile acids, and provide both immunomodulatory and antioxidant effects. Antioxidants may benefit cases of canine hepatitis: S-adenosylmethionine (SAMe; 10 mg/kg q12h), silymarin (100–200 mg/dog q24h), and vitamin E (100–400 IU/day).

2 Hepatic fibrosis or cirrhosis

Chronic hepatitis may progress to a cirrhotic liver as fibrin replaces liver parenchyma, causing permanent changes in hepatic architecture. This is a morphologic diagnosis of prognostic significance.

The presentation, often severe, can include weight loss, ascites, and jaundice. A PCV/TP can quickly rule out haemolytic anaemia as the cause of jaundice, and a serum bile acids test is redundant if the total bilirubin is significantly elevated. Ascites may be a result of portal hypertension from the cirrhotic liver, hypoalbuminaemia from loss of liver function, retention of sodium or water via stimulation of the renin–angiotensin– aldosterone system, or a combination thereof.

Although the diagnostic work-up (eg, histopathology, metal analysis, culture) is similar to that of less marked cases of chronic hepatitis, it is often too late for specific beneficial treatment, even if a primary cause is identified (eg, copperassociated hepatitis). Supportive care may include palliative abdominocentesis if the ascites compromises respiration, but the newly emptied space will likely refill. Spironolactone (1–2 mg/kg q24h) combined with furosemide may be used when fluid removal is less critical; however, it may increase risk for hepatic encephalopathy from alkalosis or hypokalemia effects.

Cirrhosis and ascites are negative prognostic indicators against which colchicine at 0.025 mg/kg q24h may be tried; however it lacks proven benefit. Prednisone and nonspecific liver protectants are indicated, but the prognosis can be grave.

*Editor's Note: These cases with hypertension will/may eventually develop acquired shunts, managing the ascites clinical sign.

3. Copper-associated hepatitis

As a form of chronic hepatitis, copper-associated hepatitis is thought to result from an inherited enzymatic defect in several breeds, especially Bedlington terriers but also Dalmatians, Labrador Retrievers, and West Highland white and Skye terriers. Excessive hepatic copper is a treatable component of chronic hepatitis cases (common in Doberman Pinschers, Cocker Spaniels, mixed breeds) in which copper accumulation may be secondary to the disease.

The copper chelator D-penicillamine at 10 to 15 mg/kg q12h is preferred for removing excess copper, and elemental zinc at 10 mg/kg q12h can decrease GI copper absorption; however, the two should not be used simultaneously.

Most commonly seen as a chronic hepatopathy, copper-associated liver disease can present as an acute illness or acute hemolytic crisis (rare), so qualitative or quantitative assessment of hepatic copper content should be a standard diagnostic of liver biopsy.

4 Copper-associated hepatitis

The liver is normally the first stop for blood carrying digestive and absorptive efforts of the GI tract to the rest of the body. Various congenital vascular anomalies can result in blood bypassing the liver, causing release of digestive content systemically without screening

or preparation and resulting in decreased liver function. The nomenclature has recently been clarified by WSAVA Standards.

The most common presentation is in young dogs with stunted growth and/or abnormal mentation, but more subtle signs of PSVA (eg, GI, PU/PD, weight loss, lethargy, poor hair coat) in older pets are increasingly appreciated; other signs are generally related to the nervous or urinary system.

Serum biochemistry profile and urinalysis may reflect the dysfunction (ie, low or low-normal glucose, cholesterol, albumin, BUN, microcytic anemia or target cells, ammonium biurate crystalluria); however, pre- and postprandial serum bile acids testing (not liver enzyme elevation) may best establish decreased liver function, although it is not specific to PSVA.

When the clinical presentation and serum bile acids test are consistent with decreased liver function, PSVA can be confirmed and characterized by abdominal ultrasonography.

A single extrahepatic (small breed) or intrahepatic (large breed) shunt is frequently visualised. Advanced imaging or histopathology can be used if ultrasonography is equivocal or if primary portal vein hypoplasia (formerly microvascular dysplasia or noncirrhotic portal hypertension) is suspected. It is necessary to determine whether the PSVA patient requires surgical (most single shunts) or nonsurgical (primary hypoplasia, multiple acquired shunts) treatment.

Medical management includes dietary intervention (low-quantity, high-quality protein) and pharmaceutical manipulation of gut ammonia production (lactulose, 0.5 mL/kg q8–12h; neomycin, 22 mg/kg q8h).

5 "Nonhepatic" hepatic disease

Nonhepatic liver disease includes the following reactive hepatopathies: vacuolar hepatopathy, steroid hepatopathy, and benign nodular hyperplasia. Elevations in liver enzyme activities do not necessarily indicate primary liver disease.

The liver senses various conditions or diseases and reacts with enzyme elevations; hence the term reactive hepatopathy. Overlooking these differentials may lead to unproductive diagnostic efforts. Steroids and anticonvulsants can induce elevated canine liver enzyme activities. Benign nodular hyperplasia in older dogs can mimic enzymatic and ultrasonographic changes similar to those seen in neoplastic disease.

Growth in young dogs, bone cancer in older dogs, and endocrinopathies (eg, Cushing's disease, diabetes mellitus) can result in elevated liver enzymes. (ALP) Idiopathic vacuolar hepatopathy is a histopathologic diagnosis consistent with exogenous steroid administration, Cushing's disease, or other systemic illness. After identifying and treating the primary disease, nonspecific therapy (eg, antioxidants) aimed at the liver may be administered. Elevated liver enzymes in a dog without clinical signs may be reevaluated in 4 to 6 weeks before pursuing a more extensive diagnostic work-up.

Closing thoughts

The liver is involved in most every aspect of life. Therefore, primary liver disease makes the list of differentials for most presentations, while secondary liver involvement must be considered in most nonliver diseases.

References available on www.vet360.vetlink.co.za

Oral Cobalamin Supplementation in Dogs with Chronic Enteropathies and Hypocobalaminaemia

Summarised by Dr Mirinda van Schoor BVSc MMedVet
Journal of Veterinary Internal Medicine 2016 vol 30: 101-107

Why they did it

Cobalamin deficiency has been reported at a prevalence of 6-73% in dogs with chronic enteropathies (CE). Other documented causes of cobalamin deficiency in dogs include exocrine pancreatic insufficiency (EPI), familial cobalamin deficiency of Chinese Shar Peis, Giant Schnauzers, Border Collies and Beagles and short bowel syndrome. Hypocobalaminemia in humans results from many causes such as pernicious anemia, gastrointestinal disease and cobalamin deficient diets (vegetarian and vegan).

Historically cobalamin deficiencies in humans and animals have been treated via parenteral (intramuscular or subcutaneous) supplementation at monthly intervals. Several studies in humans have shown that daily oral cobalamin supplementation is successful in alleviating the hypocobalaminaemia and avoids side effects such as painful injection sites and scleroderma.

No studies on oral supplementation of cobalamin are available in the canine literature. One of the authors of this paper extrapolated treatment regimens from the human literature and started using oral cobalamin in canine patients. This paper represents a retrospective analysis of the usefulness of oral cobalamin supplementation in these dogs.

What they did

They examined the records of dogs suffering from hypocobalaminemia and treated with oral cobalamin supplementation in the form of tablets.

Dogs with cobalamin concentrations below 270 ng/L at presentation and subsequent oral cobalamin supplementation were included in the study. Follow up measurements of cobalamin were done within 20-202 days after initiation of therapy.

Dogs included in this study were treated using a 1mg tablet containing cyanocobalamin. Dogs weighing less than 10 kg were given ¼ tablet daily; dogs between 10 and 20 kg were given ½ tablet daily and dogs weighing more than 20 kg were given 1 tablet daily.

What they found

Fifty-one dogs with CE and hypocobalaminaemia were included in the analysis. Twenty-two of the dogs were on immunosuppressive therapy together with various gastrointestinal medications such as gastric protectants and antibiotics.

The remaining 29 dogs received supportive gastrointestinal medication without immunosuppressive therapy. The difference between serum cobalamin concentration before and after oral treatment with cobalamin was statistically significant. Three dogs did not respond adequately initially, but in two cases serum cobalamin did increase significantly after prolonged oral cobalamin supplementation. The third dog was lost to follow up.

Take home message:

Although prospective studies are required to confirm these retrospective findings, results suggest that oral cobalamin supplementation in dogs with hypocobalaminemia appears to be effective in restoring serum cobalamin levels in dogs with CE. In some cases the rise in serum cobalamin may be delayed and follow up measurements are required to monitor the response.

Appropriate Restriction of Protein in Liver Disease

Dr Liesel van der Merwe BVSc (Hons) MMedVet (Med)
Small Animals.

Protein restriction should not be the default when a patient is diagnosed with liver disease.

Liver disease can manifest in many different forms: portosystemic vascular anomalies (PSVA/"shunts"), chronic or acute hepatitis, suppurative or non-suppurative cholangiohepatitis, toxic hepatitis, hepatic lipidosis, fibrosis and neoplasia.

It is likely that protein deficiency occurs in veterinary patients with active necroinflammatory disorders, and possibly those treated with glucocorticoids. In many of these conditions the patient is ill, not eating properly, and the liver is undergoing active damage due to inflammation and/or infection or toxic damage. The patient will require protein to provide "building blocks" for regeneration and recovery. Protein and sufficient energy will also be required to maintain lean muscle mass.

As a rule of thumb - protein should not be restricted in liver disease unless hepatic encephalopathy (HE) is present. Hepatic encephalopathy manifests in various ways. Mild changes include poor appetite, being a poor doer, intermittent gastrointestinal signs, and lethargy. More severe clinical signs can include disorientation, central blindness, stupor and seizures.[1, 2]

Don't restrict dietary protein to prevent HE - rather restrict dietary protein if HE is present and try feeding the highest level of dietary protein possible without causing a recurrence of HE.

Hepatic encephalopathy is reversible. Decrease the dietary protein levels and the clinical signs will resolve.

Most veterinary patients with liver disease are not in hepatic failure and do not suffer from hepatic encephalopathy. [1,2]

Common hepatic disorders in dogs, which are not often associated with HE, are vacuolar hepatopathy, chronic infections, chronic active hepatitis, major bile

duct occlusion, primary or metastatic neoplasia and microvascular dysplasia. [1] Less common disorders, which are associated with HE are: cirrhosis ("end stage" liver), portosystemic vascular anomalies and juvenile fibrosing liver disorders. [1]

Common hepatic disorders in cats which are not associated with HE include: mild hepatic vacuolar change, hepatic lipidosis syndrome, cholangitis/cholangiohepatitis (associated with pancreatitis and inflammatory bowel disease), major bile duct occlusion, primary or metastatic neoplasia.[1] Much less common disorders which are associated with HE are biliary cirrhosis and portosystemic vascular anomalies.[1]

Encephalopathic Toxins:

Although a variety of toxins have been identified in influencing HE, the most common one is nitrogen, both dietary and edogenous, which is detoxified in the urea cycle in the liver.[2,4]

Ingestion of a meat-based high protein diet, gastrointestinal bleeding (ulcers or verminosis), azotaemia (increase blood levels of nitrogenous waste products) and hypokalaemia (increases neurological sensitivity to ammonia) are the most common event initiators for HE in animals with severe liver disease with decreased functional parenchyma or with PSVA.[4]

The liver has a very large reserve functional capacity, so this only occurs if a large portion is damaged or if the shunt fraction is significant.

Ammonia that escapes this urea cycle and enters the systemic circulation can be metabolized to glutamine by extrahepatic cells such as astrocytes and skeletal muscle.[5] Glutamine is either excreted in urine or metabolized back to ammonia for re-entry into the urea cycle. In severe liver failure (be it from acute toxic damage or large portosystemic shunts), the astrocytes have to take on a larger detoxification role. Astrocytes contain glutamine synthetase and detoxify ammonia by converting glutamate to glutamine. [5] Glutamine is however able to enter the astrocytes' mitochondria where it is metabolized back to ammonia, leading to mitochondrial damage and cell swelling. Astrocyte swelling is a hallmark histopathologic change observed in acute HE. [5]

Ammonia itself is neutrotoxic, and elevations in blood ammonia cause neurological derangements. Recent studies have demonstrated an important role for ammonia and inflammation in the development of hepatic encephalopathy in dogs with a congenital portosystemic shunt. [5]

Manipulation of nitrogen:

Different proteins have different influences on blood ammonia and amino acid concentrations. Proteins containing haeme groups (red meat) are especially encephalogenic whereas diets containing dairy- and vegetable-protein are less so. Plant and dairy derived proteins have been shown to prolong time to development of HE and lessen its effects in dogs.[3] Condon and colleagues investigated the occurrence and severity of neurologic symtoms as well as the survival time of dogs with experimentally induced portosystemic shunting and HE, when they were fed different protein sources (eg, meat, fish, and milk).[6]

Clinical signs occurred more rapidly and were more severe in dogs fed a meat protein diet when compared to dogs fed a fish diet. The best results were seen with a diet based on milk protein. The survival time of dogs fed fish- or milk-derived proteins was almost twice as long as that of dogs fed a meat-based diet. [6] In patients with HE, non-meat protein sources such as soy or dairy protein may produce lower blood ammonia concentrations than meat sources.[5]

Dietary manipulation of protein is not the only mechanism to manage the build up of ammonia in the blood stream. Protein tolerance can be increased by using lactulose, antibiotics and soluble fibre.

Lactulose lowers the gastrointestinal pH trapping the NH3 as Nh4+in the GIT. Lactulose also changes the intestinal flora from a proteolytic to a fermentative type and decreases food and faecal transit time, preventing constipation and allowing less time for absorption of ammonia.

Antibotics: Urease producing bacteria are also responsible for the production of a lot of ammonia in the GIT and appropriate antibiotic treatment (metronidazole at 7.5 mg/kg oid / bid rather than the normal 15mg/kg bid) or neomycin (22mg po bid will limit the numbers of this bacteria, and thus the amount of free ammonia available for uptake.

Fibre fermentation increases faecal bulk, increases the unstirred water layer adjacent to the enterocyte, decreases intestinal transit time, binds enteric toxins, stimulates enteric IgA production and alters resident enteric flora all of which are beneficial.

Portosystemic vascular anomalies (PSVA)

The most frequent cause of HE are PSVA, which have an incidence of between 1 and 5% of the population and are more common in pure bred animals and certain breeds such as Yorkshire Terriers and Irish Wolfhounds.[5] Furthermore, all chronic liver diseases associated with formation of fibrosis develop portal hypertension, which may lead to acquired portosystemic shunting and then may result in HE.[5]

Most PSVA are diagnosed in young growing dogs and cats. It is especially important to consider maximising protein tolerance by using other modalities of managing HE when dealing with immature animals. These animals still need to grow, so protein restriction to prevent HE needs to be strictly balanced against the

need for protein for growth.

In these patients my approach is to maintain the highest protein diet I can get away with without getting any neurological signs (a puppy diet or an adult gastrointestinal /maintenance diet), whilst adding the other medications to reduce the ammonia such as lactulose and metronidazole/neomycin.

If surgery is not an option for whatever reason, then once the patient is adult, decisions can be made on switching from controlling clinical signs of HE with medical supplements to rather controlling them with dietary protein reduction. Once again, unless HE is present, dietary protein reduction is not indicated.

Chronic inflammatory liver disease

Patients with acute liver disease are hypermetabolic and require a higher protein intake to maintain a positive nitrogen balance.[1,2] Animals with more chronic liver disease, like humans with cirrhosis, are likewise thought to be hypermetabolic, needing protein intake higher than maintenance values. Insufficient dietary protein will result in increased catabolism of skeletal muscle mass which leads to increased ammonia production and a decreased alternative route for ammonia detoxification to glutamine.[1,5]

Because no specific measurement is available for clinical identification of malnutrition, estimation of protein-energy malnutrition is based on sequential assessments including weight changes (unintentional weight reduction); serum concentrations of proteins produced by the liver (albumin, alpha-globulins, fibronectin, fibrinogen); body condition scoring; and the serum creatinine concentration.[1,5]

In patients with liver disease, not exhibiting any signs of HE, a high quality and highly digestible protein source (as can be found in most commercially available gastrointestinal diets should be provided. Patients with acute or chronic liver disease where a large percentage (> 70%) of the liver capacity is reduced, also have decreased glycogen storage and decreased gluconeogenesis, which results in protein catabolism and fasting hypoglycemia. Diets should thus provide of dietary calories in the form of easily digested, complex, soluble carbohydrates such as rice, corn, wheat, or barley. In addition, providing small frequent meals may help maintain euglycemia and a positive nitrogen balance.[1,5]

Hepatic lipidosis

Protein-restricted diets containing high concentrations of lipids are not suitable for cats with hepatic lipidosis (HL).[1] In a small group of obese cats with HL induced by food deprivation, recovery was facilitated by feeding a diet supplemented with protein derived vs. lipid- or carbohydrate-derived energy. Cats with HL, although stressed and ill, are relatively inactive which complicates calculation of energy requirements.[1]

Considering all this, as well as the inability of cats to down-regulate protein turnover, normal energy and protein intake has been fed to most patients with HL. In the rare patient which shows HE, a restricted protein diet can be used as long as it meets minimal protein requirements for healthy cats. Recovery of cats with HE and of those on restricted protein diets is poor.[1]

Conclusion

In patients with HE, most commonly seen in end stage fibrosis or patients with shunts, dietary protein should be limited to minimize excess ammonia production. Titrate to the highest amount possible. [1,2,4]

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Shaping the future of animal health

Managing Chronic Otitis Externa

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Otitis externa (Fig 1), inflammation of the external ear canal, is one of the most commonly diagnosed skin conditions in dogs. There are a number of predisposing factors which render individual pets susceptible to chronic and recurrent otitis. Otitis media is inflammation of the middle ear.

Otitis externa is usually easily recognised by the clinician, while otitis media may be less apparent. It is important to manage otitis externa thoroughly in order to prevent the development of otitis media.

There is no recognised sex distribution for otitis externa. Young animals may be more commonly affected.

There are clear breed predispositions for otitis, which directly reflect the breed predispositions for skin disease (e.g. allergic skin disease in retrievers and terriers, food allergies in German Shepherd dogs). The most common historical findings are headshaking and aural pruritus.

Predisposing factors, primary causes and perpetuating factors are all implicated in the development of otitis.

Predisposing factors

These include defective anatomical conformation, excessive moisture, irritant topical products, and systemic disease. Conformation defects in dogs include

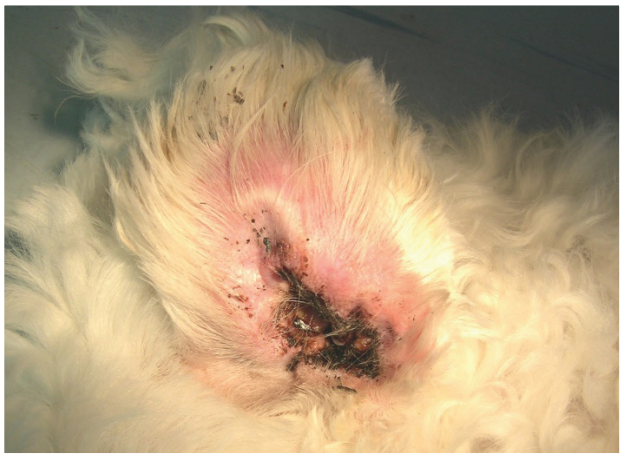

Figure 1. Typical appearance of otitis externa

a hirsute (Fig 2a,b) or stenotic meatus, and pendulous pinnae. Excessive moisture occurs in dogs that swim. Systemic disease such as immunosuppression, renal disease, hepatic disease, and in cats, FeLV and FIV infection, may predispose to otitis.

Primary causes

These include parasites, foreign material, hypersensitivity, neoplasia, polyps, keratinization disorders, and endocrine disorders.

Hypersensitivity is a very common underlying cause of otits externa, even if only one ear is affected. Animals such as golden retreivers and German Shepherd dogs have the most perfect anatomy - wide open canals, minimal hair in the canal and in GSDs' upright ears - yet they are predisposed to otitis. It's because they are predisposed to allergic disease: atopy or food allergic dermatitis. Often just managing this underlying cause will cause resolution of the otitis. Without managing the allergic disease the otitis will be recurrent and chronic changes will develop in the ear canals which will perpetuate the problem. Parasites include mites (*Otodectes, Demodex, Sarcoptes*), ticks (*Otobius*) and biting flies (*Stomoxys calcitrans*). *Otodectes cynotis* (Fig 3) the ear mite of dogs and cats, spend their entire 3-week life-cycle deep in the ear canal of the host. Otoscopic examination reveals these long-legged mites. The mites produce a marked reaction which is probably allergic in origin. Dogs shake their heads, which may lead to the formation of othaematomas. The tympanic membrane may perforate, leading to otitis media and nervous symptoms.

A roll smear of ear canal debris often reveals pathogenic organisms. *Demodex* infestation may intra- or peri-aural. Acaricides (e.g. benzyl benzoate and thiabendazole) can be inserted into the ear canal. Spot-ons containing acaricides (e.g. moxidectin, milbemycin, eprinomectin or selamectin) are advised concurrently, since the mite will often be found in the peri-auricular regions. *Otobius megnini,* the spinose ear tick, may occasionally invade cats' ears. Management is as for *O. cynotis.*

Foreign material may be grass awns, grass seeds or ticks as well as certain irritant topical agents. Atopy, cutaneous adverse food reactions, and contact hypersensitivities frequently play a mayor role.

Sensitivity to otic preparations may occur, although this is rare. Keratinization disorders include seborrhoea and endocrine disorders causing otitis include hypothyroidism, hyperadrenocorticism and sex hormone imbalance. Auditory polyps occur in both the canine and the feline, although they are extremely rare in dogs.

There is increased risk for tumours in pets with a history of chronic otitis. Benign or malignant tumours can develop in the external ear canal of dogs and cats, and arise from the apocrine or ceruminous glands that line the ear canal. The most commonly encountered tumour is the ceruminous gland adenocarcinoma and is more commonly seen than adenomas in both dogs and cats.

Figure 2a. Schnauzers and Poodles typically have a lot of hair lining the ear canal.

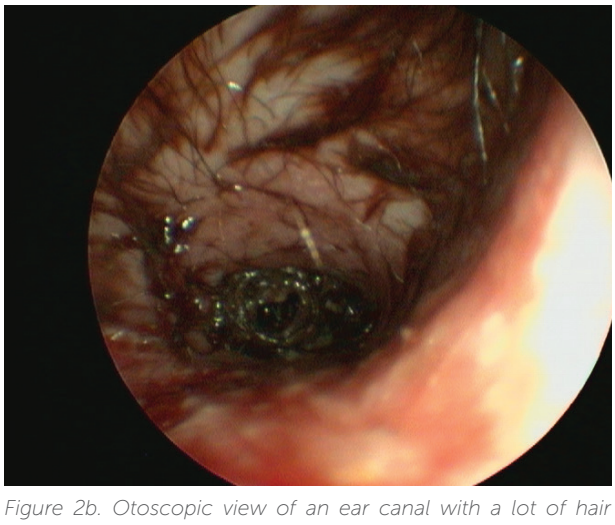

Figure 2b. Otoscopic view of an ear canal with a lot of hair showing how plugs form within the ear canal.

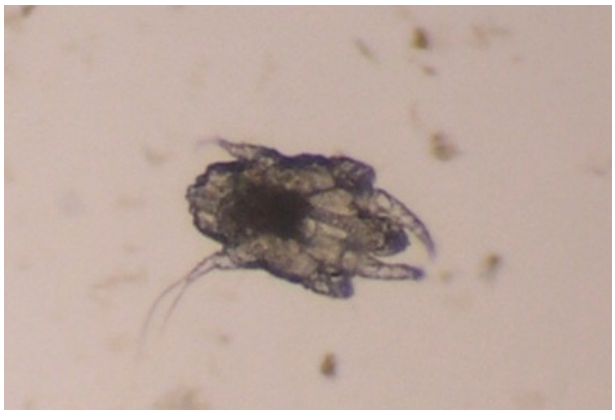

Figure 3. Otodectes cynotis on low power magnification

Perpetuating factors

Bacteria, yeasts, otitis media, swimming, sensitivity to ceruminolytics, and progressive pathological changes such as narrowing of the ear canal due to chronic changes in the epidermis.

Recognition of the predisposing, primary and perpetuating causes allows the formulation of a regime for the management of chronic otitis externa, tailored to the individual pet.

Management

The first step in the management of chronic otitis is to determine the severity of pain. This can be done by gentle palpation or petting of the animal. If the ear is painful or the degree of discomfort is high, the animal should be sedated before performing any further diagnostic testing. The second step is gentle palpation to determine the presence of swelling, pruritus, fibrosis, or calcification since these findings will determine whether imaging is necessary.

The outside of the ear should be examined, noting erythema, oedema, crusts, scales, ulceration, lichenification, hyperpigmentation, or the presence of exudate. The pinnae and peri-auricular regions should be examined for evidence of self-trauma and demodicosis (Fig 4), erythema, and primary and secondary skin lesions. Pinnal deformities, hyperplastic tissue in the ear canal, and headshaking suggest a chronic condition.

If the otitis is unilateral, the unaffected ear should be examined first to prevent iatrogenic contamination of the unaffected ear with organisms (e.g. *Pseudomonas aeruginosa* or *Proteus mirabilis*) that may be present in the diseased ear. The clinically unaffected ear may, in fact, be diseased, meaning that the differential diagnosis list should also include causes of bilateral otitis. Otoscopic examination often requires sedation.

Hyperplasia of the ear canal may prevent tympanic membrane visualisation. In some cases, where there is severe inflammation, swelling and pain it may be appropriate to treat with glucocorticoid anti-inflammatories for a few days prior to performing and otoscopic examination, even if a GA will be given. This allows the swelling to decrease facilitating visulasation of structures within the ear canal The epidermis is also less friable and bleeds and oozes less serum.

Radiographs are normal in many otitis media cases. CT or MRI, if available, should be performed for cases of severe, chronic otitis. Radiographic abnormalities include calcification of the walls of the ear canal, fluid opacity with in the bulla and thickening and sclerosis of the bulla wall.

Roll smears should be prepared using a cotton wool bud to retrieve debris and this applied to a microscope slide. Low power examination (parasites) and high power examination (stained, for pathogens) is necessary.

Selecting appropriate topical treatment

Antimicrobials should be narrow spectrum products to minimise antimicrobial resistance. Whereas culture is necessary for specific identification of causative organisms, the key to determining successful antimicrobial therapy is ear cytology. Repeated cytology is necessary to evaluate the response to therapy. Owner compliance requires careful instruction. Pets should be suitably restrained so that insertion of medication is successful and reaches the target organisms.

Accurate quantities of medication for insertion may be provided by the manufacturer, or correct amounts can be inserted using a syringe. This is especially relevant for ceruminolytics. Separate syringes for each ear prevent cross-contamination.

Since purulent material decreases the efficacy of antimicrobial therapy, ceruminolyitic therapy is an essential part of successful management; purulent exudate is hyperosmolar, and results in a hypoxic, acidic environment within the ear canal.

Prophylactic use of ceruminolytics is indicated in chronic and recurring otitis. Ceruminolytics have surfactant and detergent action and help soften, emulsify and dissolve cerumen and debris.

Dioctyl sodium succinate and triethanolamine polypeptide oleate condensate are potent ceruminolytics. In the presence of a ruptured tympanic membrane saline solution only should be used. Irrigation with a bulb syringe under sedation or anaesthesia may be necessary prior to topical antibacterial therapy. Although irritation of the ear canal can occur, the author has found that diluting ceruminolytics with water can reduce irritation to the ear canal.

Astringents (drying agents) assist in preventing mac-

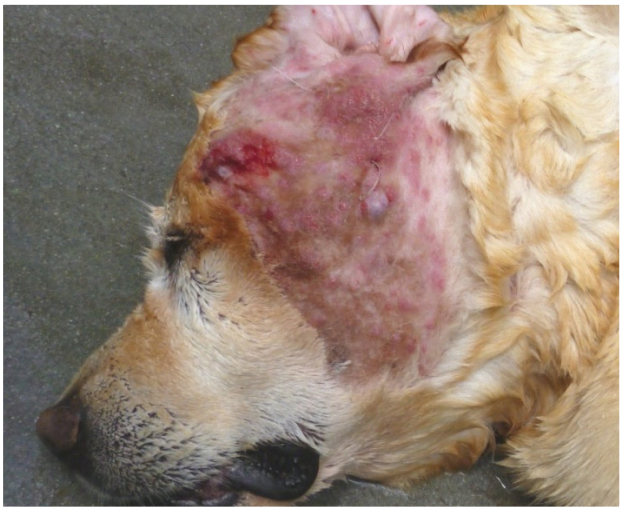

Figure 4. In many cases of otitis externa the skin of the pinna and face can also be severley affected and need specific treatment

eration of the ear canal, and include isopropyl alcohol and mild acids such as benzoic, acetic, boric, salicylic, malic and lactic acids. If the ear canal is ulcerated avoid the use of products containing alcohol or acid. Acetic, malic, boric, benzoic, salicylic and lactic acid also have antibacterial action, however a lower pH inactivates aminoglycosides and fluoroquinolones.

Fungal otitis

Malassezia spp are a normal commensal inhabitant of the ear. Increases in numbers is a secondary change treatment off the underlying cause will normally reduce numbers to normal. In true infections *Malassezia pachydermatitis* is the species routinely involved and this usually responds to azole antifungals. Azoles include clotrimazole and miconazole. However, recently developed triazole antifungals such as variconazole (Vfend®) and posaconazole may be more effective. Candida, although a common cause of fungal disease in humans, is rarely encountered in companion animal otitis. In these cases, polyene macrolides, such as nystatin is advised

Otomax® contains clotrimazole, gentamycin and betamethasone, and Surolan® contains miconazole, polymixin and prednisolone. Fluoroquinolones are broadspectrum and Posatex® is a new product not yet available here which contains posaconazole and orbifloxacin. Panalog® contains neomycin, triamcinolone and nystatin. Caution is necessary, since ototoxicity can occur in the event of a ruptured tympanic. Chlorhexidine, polymixin, and aminoglycosides have been implicated

Bacterial otitis

Coccoid organisms routinely incriminated in bacterial otitis externa include *Staphylococcus, Streptococcus* and *Enterococcus*. Rods (pseudomonads) are often encountered in chronic and recurrent otitis externa. Where Gram negative rods are found, culture and sensitivity should be performed. Generally, aminoglycosides such as gentamycin and fluoroquinolones such as orbifloxacin and marbofloxacin are effective.

However, the selected antibiotic should be continued for a minimum of 6 weeks. TrizEDTA (eromethamine and disodium EDTA dehydrate) is an aqueous preparation with efficacy against *Pseudomonas* spp. as is Flamazine (see Vet 360 May 2015 page 32 for recipes or homemade solution) \(vet360/vetlink/publications).

Glucocorticoids assist in reducing the inflammation and subsequent susceptibility to infection as well as secondary hyperplasia and proliferative changes (Fig 5). Prednisolone can be administered orally at 1 to 4 mg/kg once daily.

Approach to managing chronic otitis

Owners should be informed of correct ear cleaning procedures. Frequency of cleaning usually decreases over time to once or twice weekly as a preventive maintenance procedure. Owners should be warned to keep the pet as dry as possible, and avoid swimming. Dogs with confirmation defects such as hirsute or pendulous ears may need to have regular clipping of the pinnae.

The ear canals should be kept dry and well ventilated. Topical astringents prevent water from entering the ear canals in dogs that swim frequently, minimizing maceration of the ear canal. Chronic maceration impairs the barrier function of the skin, which predisposes to opportunistic infection. Preventative otic astringents may decrease the frequency of bacterial or fungal infections in moist ear canals.

Clipping hair from the inside of the pinna and around the external auditory meatus, and plucking it from hirsute ear canals, improves ventilation and decreases humidity in the ears. Hair, however should not routinely be removed from the ear canal if it is not causing a problem, because doing so can induce an acute inflammatory reaction.

Owners will have to be instructed as to the correct procedure for instilling ceruminolytics and astringents. Since pets predisposed to chronic otitis may require life-long ceruminolytic application, minimising discomfort to the pet during this procedure is necessary. Warming the solution to body temperature in a water bath also minimises the discomfort felt by the patient and controlled "dribbling" administration rather than a rapid squirt may be better.

Control of inflammation minimises discomfort from ceruminolytics, and corticosteroids may be required in the early stages. Non-steroidal anti-inflammatories are not as effective otic analgesics as opiods and should not be used concurrently with corticosteroids. NSAIDS may have to be supplied to the owner for administration prior to instilling the ceruminolytic. Oc-

casionally sedatives may be necessary until the pet has become accustomed to the procedure.

The pet should be suitably restrained, as this allows for speedy instillation of otic products. For dogs, restraint may include a muzzle and cats may need to be wrapped in a towel. The author advises that dogs and cats should be accustomed to a collar, and dogs should have the lead attached while the ears are treated. Dogs should be trained to sit during application of otic products.

A treat given afterwards encourages compliance. Pets may want to irritate (rub or scratch at) the ear and if a collar and lead has been applied they can be taken for a walk immediately afterwards as a distraction and reward for compliance.

Rinsing products should be inserted by holding the container vertical and upside down. The owner holds the ear pinnae with one hand, and the solution is instilled along the grooves of the inner pinnal surface into the canal. This avoids the container tip contacting an inflamed ear.

The owner maintains their hold on the ear pinna to prevent the pet shaking the product out of the canal, and gently massages the base of the ear canal to promote the action. Cotton wool or tissue paper can be used to wipe away excess solution since cats, especially, are averse to topically applied liquids. The owner may not be 100% successful at first, but should be encouraged to persist on a regular basis.

Discuss with the owner the suspected cause of the otitis, emphasising that treatment should be long term or even lifelong. All primary and secondary causes and predisposing factors should be identified and managed. Pain or pruritus should be controlled. Otitis externa is one of the few dermatologic conditions in which glucocorticoids concurrently with antimicrobial use are beneficial. Glucocorticoids such as prednisolone and triamcinolone decrease swelling of the ear canal - a key to successful treatment.

Duration depends on the severity. Ear hygiene is important; in particular, the hair in the peri-auricular area should be clipped as well as hair on the inner surface of the pinnae. This facilitates cleaning and medication. Plucking hair from the canal is controversial but may be necessary, under anaesthesia, to adequately resolve the infection.

The first ear cleaning should be performed in the clinic, and owners should refrain from topical administration until rechecked in 5–7 days since they may be too aggressive, causing further damage. Owners can administer systemic drugs and then begin to clean the ears after the first recheck, provided the otitis is resolving. Topical medications are inactivated by exudates, and excessive cerumen may prevent medications from reaching the epithelium.

Thick, dry, or waxy material requires a ceruminolytic solution such as carbamide peroxide, dioctyl sodium sulfosuccinate (DSS) or combinations of weak acids. If the tympanic membrane is ruptured, detergents and DSS are contraindicated; milder cleansers (eg, saline, saline plus povidone iodine, the Triz EDTA formulation) should be used to flush the ear.

Effective treatment may require both topical and systemic antimicrobial therapy, along with pain medications and glucocorticoids. In the management of acute bacterial otitis, the administration of corticosteroids in combination with antibacterial agents causes as reduction in exudation, pain, swelling, and glandular secretions. The least potent glucocorticoid at the lowest effective dose should be used.

Most topical products contain a combination of an antibiotic or antifungal and glucocorticoids. Most dogs require the instillation of at least one ml twice daily for effective therapy. Products with an aqueous base are preferable, however, vinegar dilutions and propylene glycol should be used with caution since swelling of the lining of the ear canal and increased glandular secretions may result. Substances that do not usually irritate the normal ear may result in irritation and inflammation.

Powders, such as those used after plucking hair from the canal, can form irritating concretions within the ear canal and should be avoided. It is important to demonstrate to the owner into which "hole" the ointment must be placed - holding the pinna upright and placing the tip into (not necessarily deeply) the most outer opening - which is the meatus of the ear canal. It is not necessary to fill the canal with ointment - a drop or two with a good ear massage, if possible, is required.

Systemic antibiotics should be used when neutrophils or rod-type bacteria are found on cytology, in cases of therapeutic failure with topical antimicrobial agents, in chronic recurring ear infections, and in all cases of otitis media. The most common cause of recurrent otitis externa is undiagnosed otitis media. Failure to use systemic antimicrobial therapy is an important cause of chronic ear disease in dogs.

Yeast infections can be treated with oral ketoconazole 5 mg/kg/day, per os, for 15–30 days. In cats, itraconazole at 2–3 mg/kg/day for 15–30 days or one week on/one week off, is recommended.

The best treatment of chronic otitis is prevention. In addition to identifying the cause of acute otitis, topical and/or systemic medications should be chosen based on cytology or culture. They should have a narrow spectrum and be specific for the current condition.

Aminoglycoside and fluoroquinolone antibiotics should not be used unless absolutely required for successful treatment but are the most common ingredients in topical otic medications. Because many topical products contain a combination of glucocorticoid, antibiotic, and antifungal medications, it is imperative to educate the owner on the frequency and duration of administration.

Polymyxin B and fluoroquinolone antibiotics have shown the best success in controlling Pseudomonas infections in cases in which resistance has been identified through culture. Methicillin-resistant *Staphylococcus pseudintermedius and Pseudomonas aeruginosa* have emerged as frustrating causes of otitis and resistance is developing to fluoroquinolones.

Duration of treatment will vary depending on the individual case but should continue until the infection is resolved based on re-examination and repeat cytology and culture. Animals with bacterial and yeast infections should be physically examined, with roll smears examined every other week until there is no evidence of infection. In some chronic cases a therapeutic regimen must be continued indefinitely.

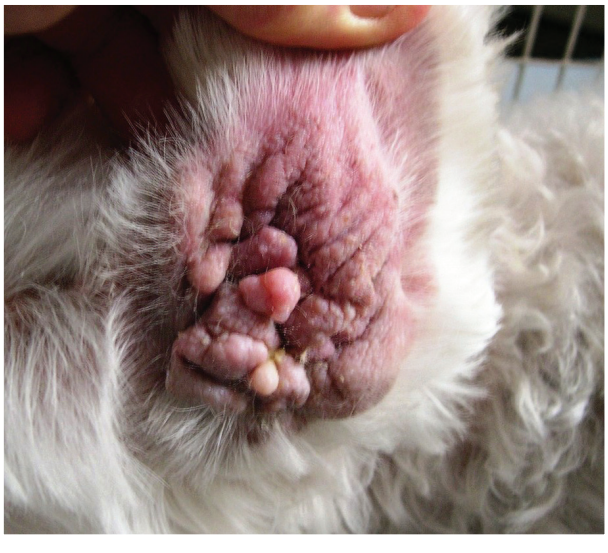

Figure 5. Chronic hyperplastic changes of the external ear canal. Surgery is now the only option

Infections are often chronic in course and associated with marked suppurative exudation, severe epithelial ulceration, pain, and oedema.

Successful treatment is multifaceted and should include the following steps:

1. Identify the primary cause of the otitis and manage it.
2. Remove the exudate via irrigation of the ear canal
3. Identify and treat concurrent otitis media.
4. Select an appropriate antibiotic from the results of culture and mean inhibitory concentration and use it at an effective dosage for an appropriate duration.
5. Treat topically and systemically until the infection resolves (weeks to months).

Non-responsive cases may require surgery. Lateral ear canal resection may be necessary to allow resection of neoplasia or polyps, and may assist in hyperplasia of vertical canal, also known as proliferative otitis externa (Fig 5.).

Summary

The keys to successful management of chronic otitis externa include early recognition and management of of predisposing factors, client education, and the formulation of a consistent management regime. Regular re-evaluation by the veterinary surgeon is essential to prevent hyperplasia of the ear canal, also known as proliferative otitis externa.

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9. Otitis Externa. Dr Martin Briggs. SMS code = a22785
8. Canine Idiopathic Dilated Cardiomyopathy. Dr Alain Carter. SMS code = SMS code = a68068
7. Cushings. Various Authors. SMS code = a20643
6. Cyclosporine in Canine Atopy. Dr Heidi Schroeder. SMS code = a24438
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2. A review of sterilisation practices and impact on the individual animal, in dogs and cats. Drs K de Cramer and K May SMS code = a26581
1. Rehabilitation of neurological patients. Dr Megan Kelly, SMS code = a97907

CPD Questions AC/1473/16

Please read both articles to answer MCQ: (1) Managing of Chronic Otitis Externa & (2) Total Ear Canal Ablation and a Lateral Bulla Osteotomy

1. Which one of the factors listed below is NOT a predisposing factor for otitis?
   a. Defective anatomical conformation such as stenotic meatus and pendulous pinnae.
   b. Excessive moisture in dogs which swim.
   c. Ear parasites.
   d. Irritant topical products.
   e. Systemic disease such as immunosuppression.

2. Which one of the factors listed below in NOT a primary cause of otitis?
   a. Foreign material.
   b. Hypersensitivity.
   c. Neoplasia.
   d. Keratinization disorders.
   e. Excessive moisture.

3. Which one of the following statements is FALSE when applied to otitis caused by cutaneous hypersensitivity reactions?
   a. It is a very common underlying cause.
   b. Both ears are generally affected.
   c. German Shepherds and Golden Retrievers are predisposed.
   d. Dermatitis can be caused by food allergies or atopy.
   e. Recurrent otitis will occur if the allergic disease is not managed.

4. Which one of the following statements regarding bacteria seen on an ear smear is INCORRECT?
   a. Best sampled using a cottonbud and rolled onto the slide.
   b. Best seen under high power magnification on a slide stained with diffkwik.
   c. Are secondary and not the primary cause of the otitis.
   d. Are primary pathogens and need to be managed as such.
   e. Are commonly seen together with Malassezia spp in most otitis cases.

5. Which one of the following statements regarding bacterial infection of the ear canal is INCORRECT?
   a. Narrow spectrum antibiotics reduce the development of resistance.
   b. Antibiogram is the key to successful antibiotic therapy.
   c. Antibiograms are required to evaluate response to therapy.
   d. Ear cytology is required to evaluate response to therapy.
   e. Purulent material decreases the efficacy of antibiotics.

6. Which one of the following statements regarding bacterial otitis is INCORRECT?
   a. Coccoid organisms often seen with bacterial otitis externa include Staphylococcus, Streptococcus and Enterococcus.
   b. Rods seen on an ear smear are generally *Pseudomonas* spp.
   c. Where Gram negative rods are found, culture and sensitivity should be performed.
   d. Glucocorticoids are contra-indicated when rod shaped bacteria are seen on a smear.
   e. Generally, aminoglycosides such as gentamycin and fluoroquinolones such as orbifloxacin and marbofloxacin are effective against *Pseudomonas spp.*

7. Which one of the factors listed below is NOT an indications for performing a total ear canal ablation and bulla osteotomy?
   a. Cases that have failed to respond to long term medical management.
   b. Cases with severe calcification of the cartilage or sclerosis or fluid accumulation in the tympanic bulla.
   c. Cases with severe soft tissue hyperplasia from chronic inflammation extending past the vertical canal.
   d. Cases with severe trauma to the ear canal.
   e. Cases with neoplasia of the lateral wall of the vertical ear canal.

8. An abnormal neurological examination may indicate severe progression of the otitis. Which one of the neurological deficits listed is NOT typical with otitis externa?
   a. Ipsilateral facial nerve paresis or paralysis.
   b. Ipsilateral peripheral vestibular disease.
   c. Ipsilateral trigeminal disease.
   d. Ipsilateral horners syndrome.
   e. Ipsilateral deficits of the sympathetic supply to the face.

9. Which one of the intra and post-operative complications listed below is the most common and serious?
   a. Intra-operative haemorrhage is the most common complication
   b. permanent facial nerve damage is 31%
   c. Draining tracts from remaining secretory tissue in the bulla occur in 5-10% of cases
   d. There can be acute wound complications such as wound breakdown and soft tissue swelling. These can often be controlled with good nursing and wound management
   e. Necrosis of the pinna is seen from damage to the blood vessels that supply the pinna

10. Which one of the neurological deficits listed below occurs **IN** peripheral vestibular syndrome?
    a. Vertical nystagmus
    b. Ipsilateral proprioceptive and motor deficits
    c. Horizontal and vertical nystagmus
    d. Disorientation and loss of balance
    e. Ipsilateral trigeminal nerve deficits (CN V) disease

Total Ear Canal Ablation and Lateral Bulla Osteotomy

Dr Ross Elliot BVSC MMedVet (Surg)
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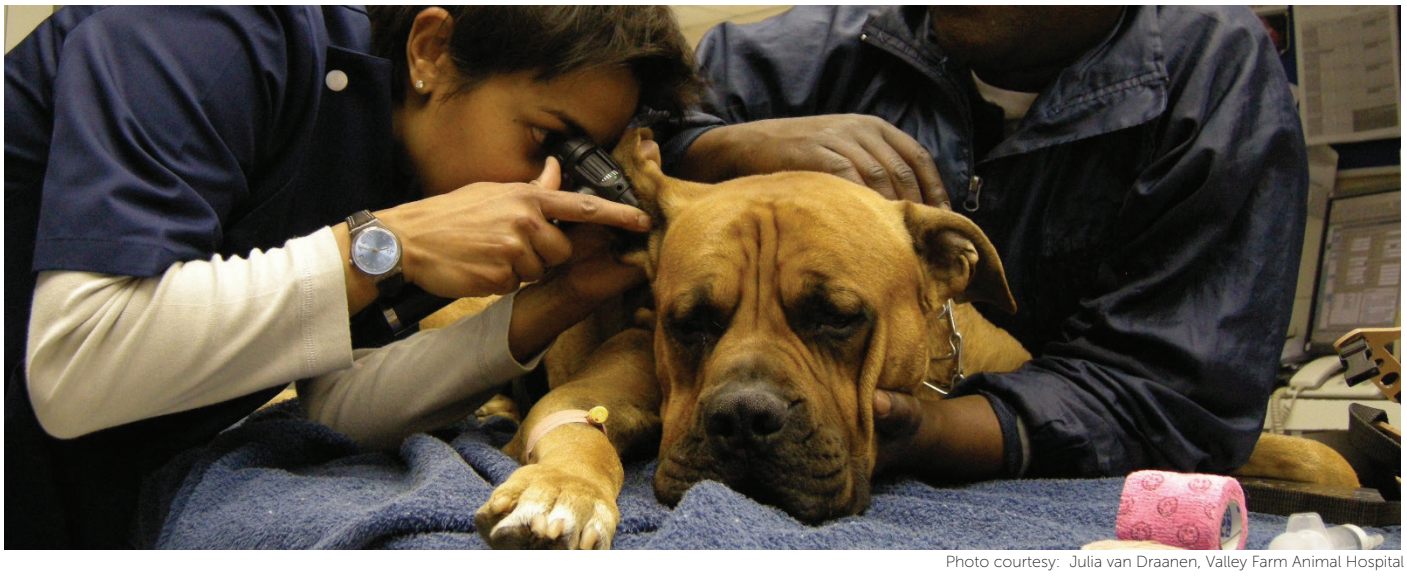

Photo courtesy: Julia van Draanen, Valley Farm Animal Hospital

Total ear canal ablation and a lateral bulla osteotomy (TECA-BO) are two separate procedures which are usually combined as a surgical treatment for otitis externa and media.

The surgical procedure for a TECA-BO entails removal of both the vertical and horizontal ear canal with all the secretory epithelial lining of the middle ear. This surgery has the potential for serious complications and should not be performed unless the surgeon is familiar with the anatomy of the ear and associated structures. A total ear canal ablation should never be performed without a lateral bulla osteotomy. If the bulla osteotomy is not performed all the secretory lining of the middle ear is left behind and this will increase the potential for complications by as much as 82%.

There are very few cases in dogs where a bulla osteotomy is performed without a total ear canal ablation. These are otitis media in the presence of an intact tympanic membrane. Neoplasia of the middle ear or polyps of the middle ear in cats that have recurred after previous removal, generally a ventral bulla osteotomy is performed in these cases as the ear canal cannot be removed and the ventral bulla osteotomy is easier to perform.

A lateral ear canal resection or Zepps procedure has little place in the treatment of otitis externa and media. A study on its effectiveness showed in all dogs it has a 45% success rate in the management of otitis externa and media. Better outcomes have been reported in Sharpei's and Spaniels have a very poor outcome with a just a lateral ear canal resection. The only indication for it is neo-

plasia of the lateral wall of the vertical ear canal.

The indications for a TECA-BO is severe end stage otitis externa and media. All of these cases usually have severe narrowing of the ear canal, which makes topical medical treatment unsuccessful

These are cases that have failed to respond to appropriate long term medical management:

- Have severe calcification of the cartilage of the ear canal.
- Have visible sclerosis or fluid accumulation in the tympanic bulla.
- Have severe soft tissue hyperplasia from chronic inflammation extending past the vertical canal.
- Have neoplasia of the medial vertical and horizontal ear canal.

Severe trauma to the ear canal, or congenital malformations can often require a TECA BO when the integrity of the ear canal is severely damaged.

A high percentage of dogs with severe otitis have associated allergic skin disease. This skin disease should be managed medically prior to resorting to surgery. The otitis will often benefit greatly from management of the allergic skin disease. However if there is marked mineralisation of the ear canal, or secondary changes in the bulla then the ear disease will eventually require surgery.

The advantages of surgery include:
- No need for continued medical treatment with successful surgery (can often negatively affect the pet-owner relationship)
- Relief of pain and improved quality of life for the patient
- Prevents further secondary change and damage to associated structures

The disadvantages of surgery include:
- Surgical complications such as facial nerve paralysis, vestibular syndrome and Horner's syndrome
- Financial cost of surgery
- Surgical complications

Complete ear work up for surgery

A complete physical examination is part of any work up to try ascertain if there are any concurrent disease processes that may complicate the healing of the patient or the ability to tolerate the anaesthesia and surgery. A full neurological exam should always be performed to detect any pre-existing facial nerve or peripheral vestibular involvement. The owners should be informed of these results as they may be present in a significant number of dogs. An abnormal neurological examination may indicate severe progression of the otitis or other differential diagnoses

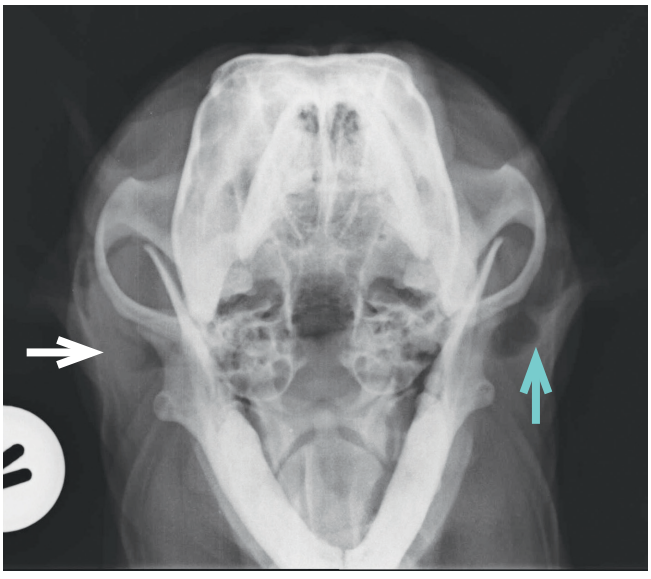

Figure 1. A standard ventro-dorsal radiograph. In this patient the right ear canal shows soft tissues opacities (white arrow) which were diagnosed as severley inflammed tissue on biopsy. The left ear canal (blue arrow) is normal

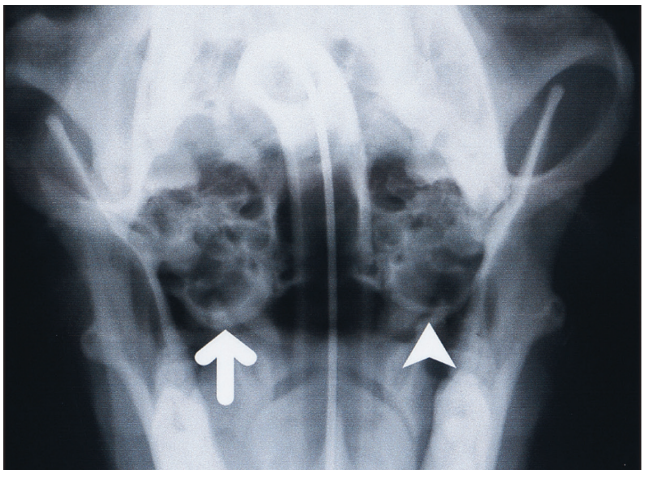

Figure 2. Rostro-caudal skull radiograph. Arrow indicates thickening of right tympanic bulla wall suggestive of otitis media. Arrowhead indicates normal left tympanic bulla

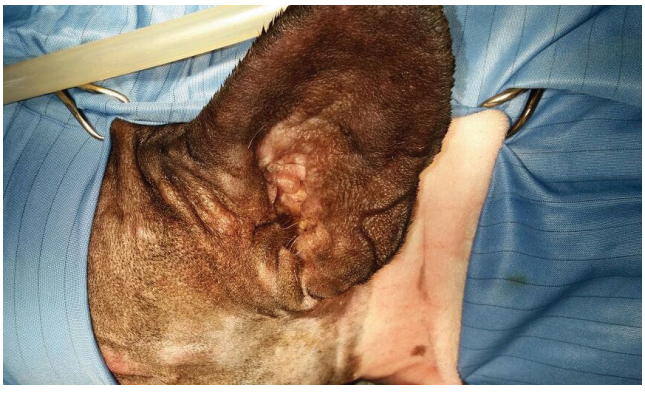

Figure 3. The patient in position prior to surgery

such as granulomatous menigioencephalitis or central vestibular disease. Detecting facial nerve damage prior to surgery prevents its being considered a surgical complication.

Owners will often be concerned about the ability of the animal to hear after the procedure especially when it has to be performed bilaterally. This can be assessed with a BAER test pre-operatively. However in reported owner perceptions it has been found that there is not a noticeable loss of hearing after the procedure. The reality is that the middle ear is already so damaged in the appropriate surgical candidate for the surgery, that the ability to hear has already been damaged and the patients have already adapted.

Otoscopy and culture are essential in the work up. This is best done under sedation as the ears are often severely inflamed and painful. The patients should be admitted and once sedated, otoscopy can be performed and a culture taken from the middle ear, via a myringotomy if the tympanic membrane is still intact. The tympanic is often found to be perforated and otitis media is assumed. A culture can be taken from the ear canal.

The next step is to perform radiographs or CT/MRI. Plain film radiographs are most commonly used to evaluate the outer and middle ear, but they are not considered highly sensitive. They are however highly specific. CT shows a higher sensitivity especially when combined with plain film radiographs. MRI is once again highly sensitive and specific. In practice the first step is to perform a standard ventro-dorsal radiograph (Fig 1) to assess the ear canal and check for calcification of the ear canal. A frontal open mouth view (Fig 1) is then used to assess the tympanic bulla for fluid opacity and periosteal reaction, which would indicate chronic otitis media in most cases.

In cases where there is calcification of the ear canal, periosteal reaction or fluid in the bulla then a TECA BO is indicated. These cases are unlikely to respond to medical management as this can be considered an "end stage" ear.

Surgical procedure

Otitis externa and media is usually bilateral and cases often require surgery on both ears. It is possible to perform both surgeries at the same time but this has little advantage to the patient. It prolongs the anaesthetic time and doubles the pain experienced by the patient. Generally the disease process has been going on for many months to years and to wait 3 weeks between surgery is of little consequence to the patient.

The surgical site should be widely clipped and aseptically prepared. The ear canal should be thoroughly lavaged with 0.5% hibitane and water prior to surgery.

The patient is placed in lateral recumbency with a towel placed under the head to elevate the head (Fig 3). The entire ear canal is then sharply dissected out from the surrounding tissue, staying as close as possible to the cartilage of the ear canal without penetrating the cartilage (Fig 4).

A recent report of a subtotal ear canal surgical method has been reported, which will maintain the ear carriage but this can leave a small amount of diseased tissue just distal to the pinna which may lead to recurrent dermatological disease.

The facial nerve courses caudo-ventrally to the horizontal canal and should be identified and gently retracted (Fig 5). Where the bulla connects with the horizontal ear canal is easily digitally palpated. Being careful not to transect the facial nerve the horizontal canal is sharply dissected off the bulla (Fig 6 and 7).

The external acoustic meatus can now be seen with a small rim of remaining cartilage of the horizontal ear canal. This cartilage is gently removed with a small periosteal elevator. The meatus is then enlarged in a ventrolateral direction with a sharp bone Rongeur being careful to avoid the branches of the internal carotid ventral to the bulla and the venous sinus caudal to the bulla. The secretory lining of the middle ear should now be visible.

A small curette is gently used to try peel off the membrane from the underlying bone. The dorsomedial section of the middle ear should be avoided with the curette as this will damage the structures of the internal ear. The membrane is usually a grey black colour, once removed there should be a shiny white appearance to the bone of the middle ear (Fig 7).

The surgical site should be irrigated with saline and the subcutaneous tissues and skin closed. The decision to place a drain is the choice of the surgeon. However if the ear has had a culture taken as a part of the work-up, an appropriate course of intra-operative antibiotics is sufficient. This varies from case to case and the amount of contamination during the surgery and the surgical time. I generally don't place a drain after the surgery.

The use of postoperative antibiotics is controversial as if all tissue has been removed and thorough lavage performed there should be no need for continued antibiotics. It would be bad practice to rely on antibiotics to prevent infection from tissue left behind. If there is any secretory tissue left behind regardless of antibiotics used it is likely that the infection will return. A culture can be taken form the bulla once the secretory lining has been removed and lavage has been done.

Postoperative care

Pain control is essential in these patients. This should be titrated on a patient to patient basis. Basic pain control can be managed with injectable opioids, the pure agonists being the obvious choice. Generally I will start the patient on morphine every 4 hours at 0.5mg/kg. The next stage would be a fentanyl CRI. In patients that are still assessed to be in pain then a morphine, lignocaine and ketamine infusion is administered. Pain control is continued as long as is required.

These patients are often hypothermic from the extended surgical time and this complication needs to be monitored and addressed. The best is to warm them up with warm air blankets to prevent thermal burns. I will often use ocular lubricants post-surgery as there can be a delayed blink reflex from neuropraxia of the facial nerve. This tends to return to normal in 24 to 48 hours.

In most cases when there has been no obvious contamination of the surgical site I will use antibiotics based on a culture for 12 hours post-surgery. I generally do not continue them longer than this.

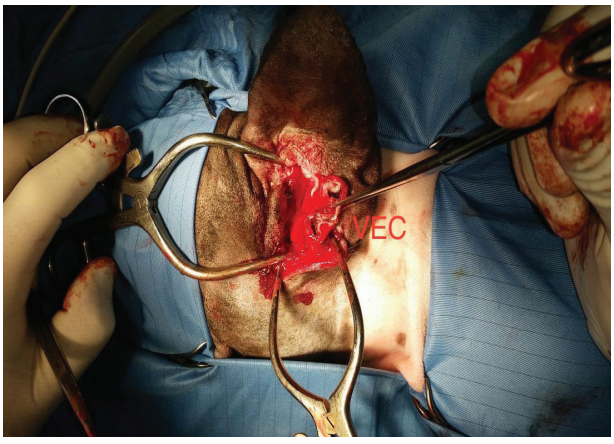

Figure 4. The vertical ear canal is seen with the initial dissection performed. The facial nerve is not visible at this point.

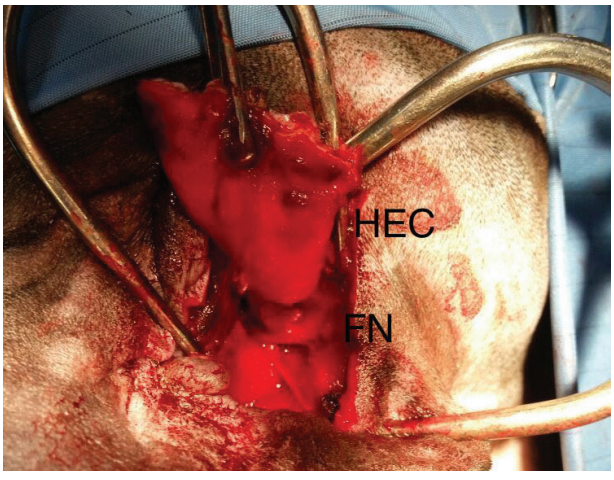

Figure 5. The vertical (VEC) and horizontal ear canal (HEC) have been dissected and the facial nerve can be seen in close proximity (FN)

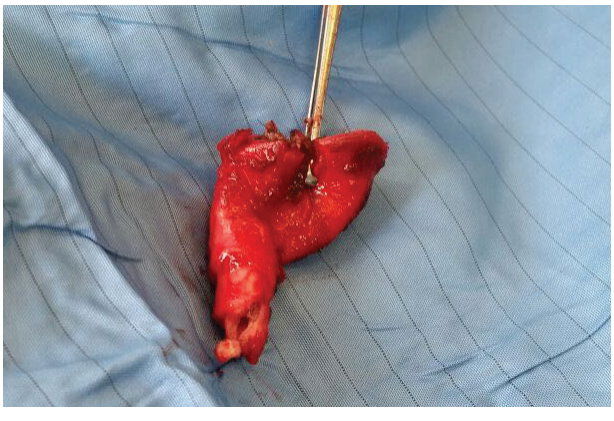

Figure 6. The ear canal removed completely.

However if there is obvious evidence of bulla osteomyelitis the antibiotics should be continued on the basis of a culture for 4-6 weeks.

Once the patient is mobile, alert and preferably eating in hospital I will look to change them onto oral pain medication and send them home. These patients have often been on corticosteroids and hence one should be careful with non-steroidals for pain. If there has been no corticosteroid use in the last 5 days they can be discharged on NSAIDs and oral tramadol.

A buster collar is placed to stop the patient scratching at the wound. If a drain is placed then the head should be bandaged until the drain is removed. This bandage needs to be changed every 24 to 48 hours till the drain is removed.

Complications

The complication rates with a TECA BO are high. Intraoperative haemorrhage is the most common complication, which could be fatal if not managed. However the haemorrhage is usually insignificant and hampers the surgeon's ability to see during surgery more than anything else.

The reported incidence of permanent facial nerve damage is 31% this can be from transection of the facial nerve as it exits the stylomastoid foramina, or severe neurontometesis (traction injury) from inappropriate retraction directly on the nerve. Signs of facial nerve paralysis are loss of the palpebral reflex and hemiparesis of that side of the face. The parasympathetic portion of the facial nerve innervates the lacrimal glands thus damage to the facial nerve will cause decreased tear production. Eye lubricants will be needed to lubricate the eye in the short term until the lacrimal function returns to normal. However they are not essential in the long term as the lacrimal function is normal, passive function of the third eyelid and retraction of the globe are enough to protect the globe.

Draining tracts from remaining secretory tissue in the bulla occur in 5-10% of cases. These will not respond to antibiotics and will require repeat surgery and curettage of the bulla. This can be performed through a repeat lateral approach or through a ventral bulla osteotomy. A lateral approach would have a higher incidence of facial nerve damage when compared to a ventral approach.

There can be acute wound complications such as wound breakdown and soft tissue swelling. These can often be controlled with good nursing and wound management.

Necrosis of the pinna is seen from damage to the blood vessels that supply the pinna. These vessels run under the cartilage on the proximal edge of the pinna margin and can be damaged when removing the vertical ear canal.

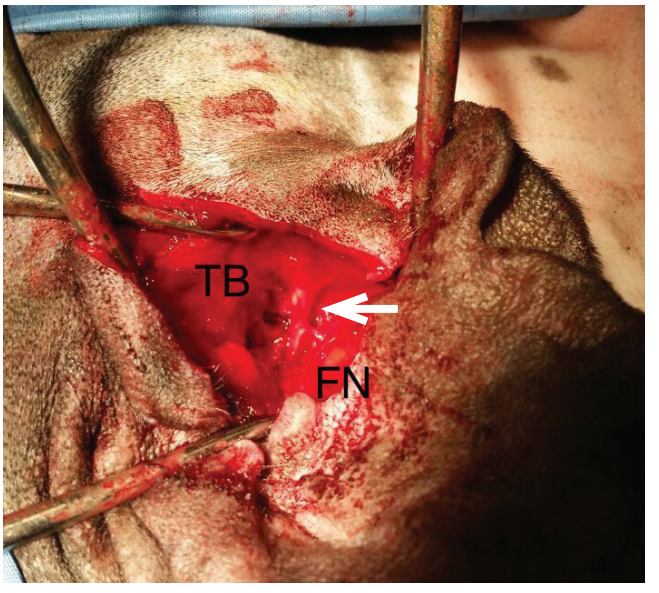

Figure 7. The opening of the tympanic bulla (TB) can be seen. The facial nerve can be seen in close proximity (FN - arrow).

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Nerves Which are Affected by Otitis Media

Dr Liesel van der Merwe BVSc (Hons) MMedVet (Med)
Small Animals.

There are several important nerves which run through and immediately adjacent to the middle and inner ears. These structures are affected with progressive otitis and can also be damaged during surgery.

The middle ear lies beyond the tympanic membrane and consists of the mucosa lined bulla which contains the three auditory ossicles which transmit sound from the external ear to the inner ear. The auditory (eustachian) tube connects the nasopharynx to the middle ear.

The facial nerve and the sympathetic supply to the eye are closely associated with the cavity of the middle ear. Deficits may include facial paralysis, horners syndrome or pain on opening the mouth.

The bony cochlea together with the vestibule and semicircular canals is situated within the petrous-temporal bone and comprises the inner ear. Inflammation of this area will result in peripheral vestibular disease. The facial nerve runs, along with and just above, the vestibuloc ochlear nerve (CNVIII) through the petrosal bone and emerges from the skull through the stylomastoid foramen.

Chronic ear infection can result in pyogranulomatous otitis media - interna with the development of osteomyelitis of the tympanic bulla. This will generally result in peripheral or central vestibular disease - often without any of the other intracranial neurological deficits expected with space occupying lesion:head/neck pain, lethargy, seizures.

Horners syndrome:

The sympathetic supply to the eye originates in the hypothalamus descends through the brain stem and spinal cord to T1-T3 where it synapses and exits through the brachial plexus nerve roots and travels rostrally again within the vagosympathetic trunk, synapsing on the cranial cervical ganglion. The axons finally travel through the middle ear along the floor of the skull and exit via the orbital fissure to innervate the smooth muscles of the eye.

Miosis: Ipsilateral. Best seen when lights are dimmed and failure to dilate is noted. With aniscoria it is always important to decide which pupil is actually affected - the small pupil or the dilated pupil – and this interpretation will depend on levels of light and stress.

Ptosis: Ipsilateral loss of tone to the eyelid causes them to droop - narrowing the palpebral opening.

Enopthalmosis: Ipsilateral loss of sympathetic tone to the smooth muscles of the orbit resulting in the retractor bulbi muscles pulling the eyes, to sink further into the orbit. This will also cause the third eyelid to protrude.

Vasodilation of ipsilateral aural, facial or conjunctival blood vessels may be noted.

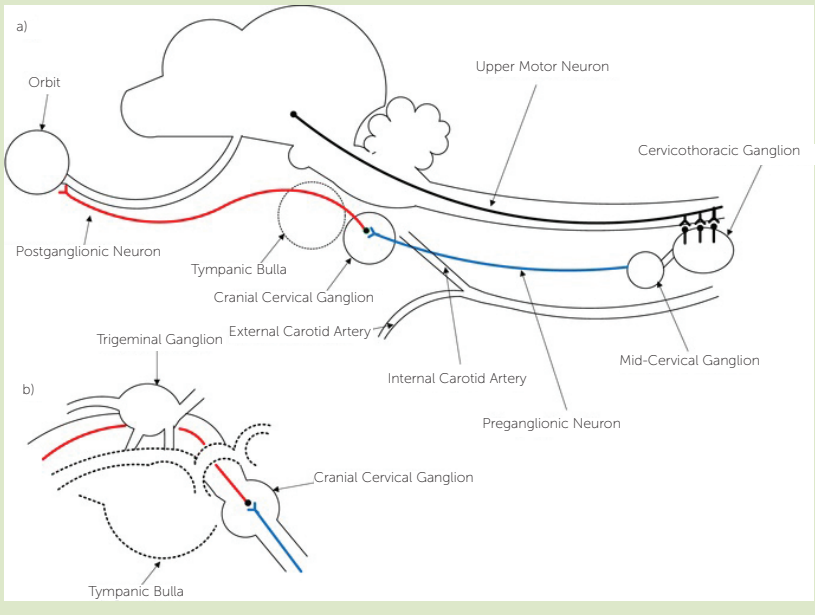

Figure 1. Graphic depiction ot the first (spinal) second (brachial plexus to cranial cevical ganglion) and third order (cervical ganglion to eye) of the sympathetic supply to the eye. The smaller graphic illustrates the close proximity the nerves have to the middle ear.

Source: BSAVA Manual Small Animal Neurology

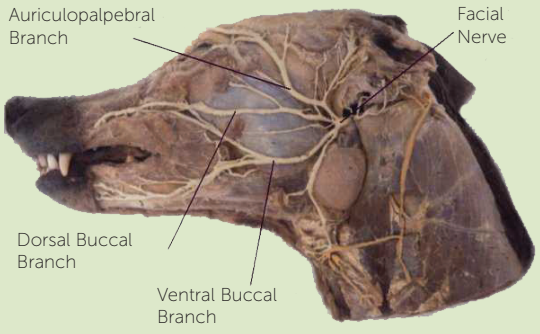

Figure 2. Anatomy specimen of a dogs head, showing dissection of the facial nerve. Note the exit from the skull where the horizontal ear canal inserts into the bulla.

Vestibular Disease Classification

	Peripheral	Central
Paresis	Never	Possible - brainstem disease may affect ascending proprioceptive and and descending motor tracts
Proprioceptive deficits	Never	Possible – brainstem disease may affect ascending proprioceptive and and descending motor tracts
Consciousness	Alert May be confused, distressed, disoriented	May be depressed, stuporous or comatose
Cranial nerve deficits	Facial nerve deficits - runs close to middle ear	Facial nerve deficits - exits brain stem close to vestibulocochlear nerve (CN VIII) Additionally Cranial Nerves V and VI all the way down to XII may be affected (brain stem lesion)
Horners syndrome:	Possible	Unlikely
Nystagmus	Horizontal or rotatory - NEVER VERTICAL Fast phase away from side of lesion Direction doesn't change with head position	Horizontal, rotatory or vertical Fast phase in any direction Direction may change with head position

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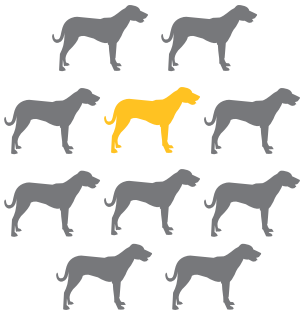

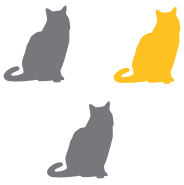

In time, kidney disease will develop in:
1 in 10 dogs and 1 in 3 cats

25%

As little as 25 % of kidney function remaining at time of diagnosis with CREA

60%

Up to 60 % of kidney function remaining at time of diagnosis with SDMA

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IDEXX LABORATORIES

New Test for The Early Diagnosis of Chronic Kidney Disease

By: Hanne Friis Lund, DvM, Nordic Medical Affairs Manager, IDEXX

IDEXX SDMA™, a new and revolutionary biomarker for kidney function in dogs and cats enables diagnosis already in IRIS Stage 1 and 2 thereby opening up for early intervention

Chronic Kidney Disease (CKD) is one of the most prevalent conditions in dogs and cats. Statistics from the US have shown that 1 out of 10 dogs and 3 out of 10 cats will develop CKD within their life span, prevalence obviously increasing with age.

The big challenge with these CKD patients is that the cause is usually incurable and medical care is mainly about prolonging life and making the animal as comfortable as possible.

The earlier we can intervene with diet and medical treatment, the longer the animal is expected to live.

Diagnosing CKD in very late stages gives veterinarians little chance to make a difference and that is what we often see in veterinary practice - the patient being presented to us when it is actually too late to make a difference.

Diagnosing CKD

The classical diagnostic tools in diagnosing renal failure consist of a minimum database including:

Biochemistry and electrolytes: Elevated BUN and creatinine (azotaemia), electrolyte disturbances such as high phosphate and low potassium in the severe cases. Low total protein and high cholesterol are also common findings.

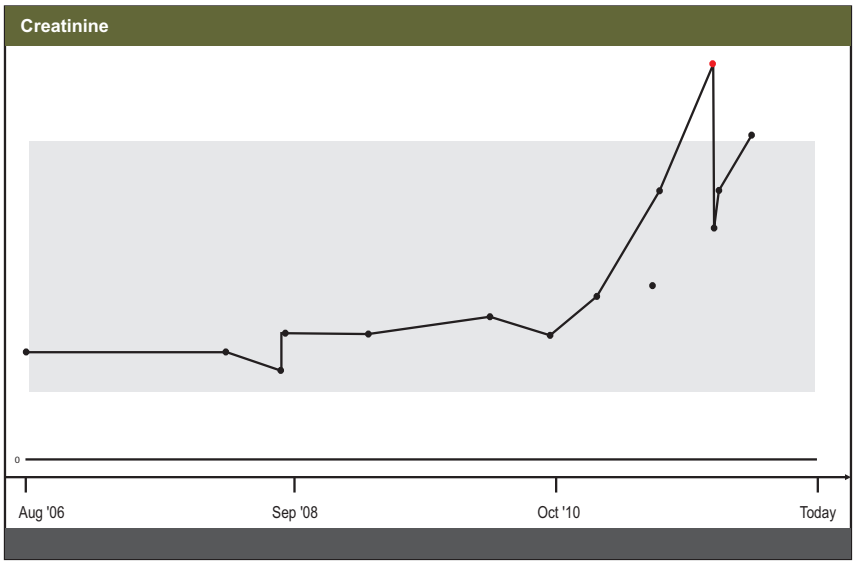
Hematology: CKD patients are often presented with a non-regenerative anaemia and a stress leukogram.

Urinalysis: Lack of urine concentrating ability: low urine specific gravity (USG) and proteinuria is common but not always seen and, depending on the renal pathology, casts may be seen.

Diagnostic imaging and blood pressure: CKD is the most common cause of elevated blood pressure in the dog and cat. Imaging is vital to determine if there are reversible causes such as renal or ureteral urolithisasis, renal neoplasia (lymphoma) and also to evaluate renal parenchyma and size.

Of the biochemistry parameters, creatinine is by far the most specific parameter for renal function as it is primarily excreted through the kidneys. The challenge is that is only increases fairly late in the disease process (>75% renal dysfunction) meaning that up to 75% of the function has been lost before creatinine is actually elevated beyond reference intervals.

Creatinine can vary greatly between different animals and is impacted by factors such as: lean body mass, diet, time of day and breed. For instance oriental cat breeds such as the Burmese cat have naturally quite high creatinine levels without any renal pathology. On the other hand small dog breeds with little lean muscle mass will have a significantly lower creatinine than highly muscled dogs. This parameter is thus bet-



ter measured against "individual normal" than population normal ranges. The sensitivity of creatinine for diagnosing renal failure increases significantly if we establish the patient's own baseline intervals as that enables us to trend creatinine and thereby detect persistent increase in creatinine.

Gold standard in evaluating kidney function

The absolute gold standard in evaluating kidney function in a patient is to perform a clearance test for the calculation of Glomerular Filtration Rate (GFR).

Iohexol and inulin clearance are both looked upon as gold standard tests and a reliable exogenous creatinine clearance test has also been commercially available for some years. The challenge of doing regular clearance tests in veterinary practice is that is often requires special equipment, it takes time, as blood and urine has to be collected several times within a given time interval and it is a costly diagnostic procedure.

For these reasons, although available, it is performed very rarely in veterinary practice outside of referral hospitals and university hospitals.

The big desire for many years has therefore been to find a single test that could give a valid estimate of the GFR, earlier in the course of renal deterioration than creatinine, without doing an actual clearance test.

SDMA and early diagnosis

SDMA is an abbreviation for Symmetrical Dimethyl Arginine. It is not a new test as such, as it has been used to evaluate renal function in humans for many years. It is new to veterinary diagnostics and has now been validated for use in dogs and cats. A veterinary specific analysis has been created and released this year, the IDEXX SDMA™.

SDMA is created by methylation in arginine in any nucleated cell during protein production. SDMA is released into the blood stream and excreted only through the kidneys. This means that the level if SDMA in the blood equals the kidney's filtration rate.

Veterinary research on this parameter started about a decade ago and IDEXX laboratories has worked intensely with different universities and the International Renal Interest Society (IRIS) to look further into this. Hills nutrition has played a very vital role as a great deal of the validation studies has been done on their pet colony.

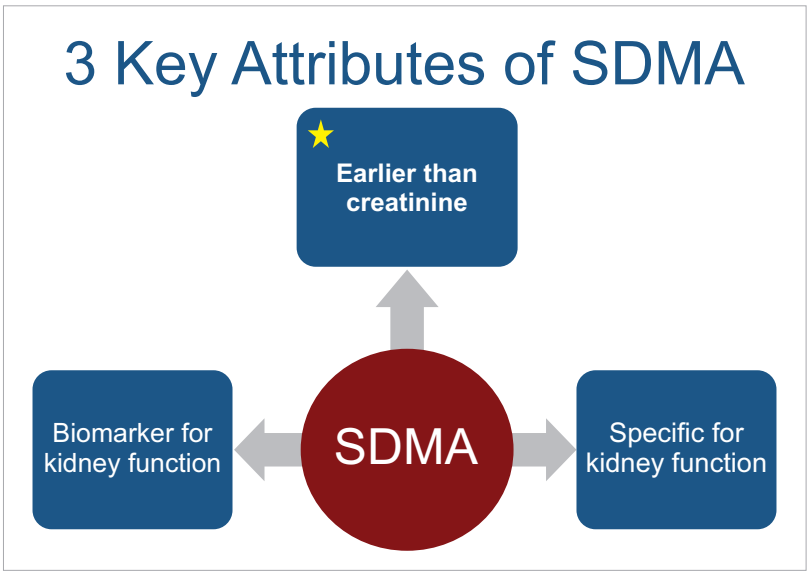
The three key attributes of SDMA

- It correlates with GFR, hence it is a biomarker for kidney function
- It is specific for kidney function
- It is an earlier indicator of renal dysfunction.

Being specific for renal function means that SDMA is not impacted by lean body mass, breed, age or sex. Nor is it impacted by arginine levels in the body, liver function and diet.

The independence of lean body mass is in particular interesting as when patients start going into renal failure, often they lose weight which can falsely decrease the creatinine concentration, SDMA will not be impacted by this.

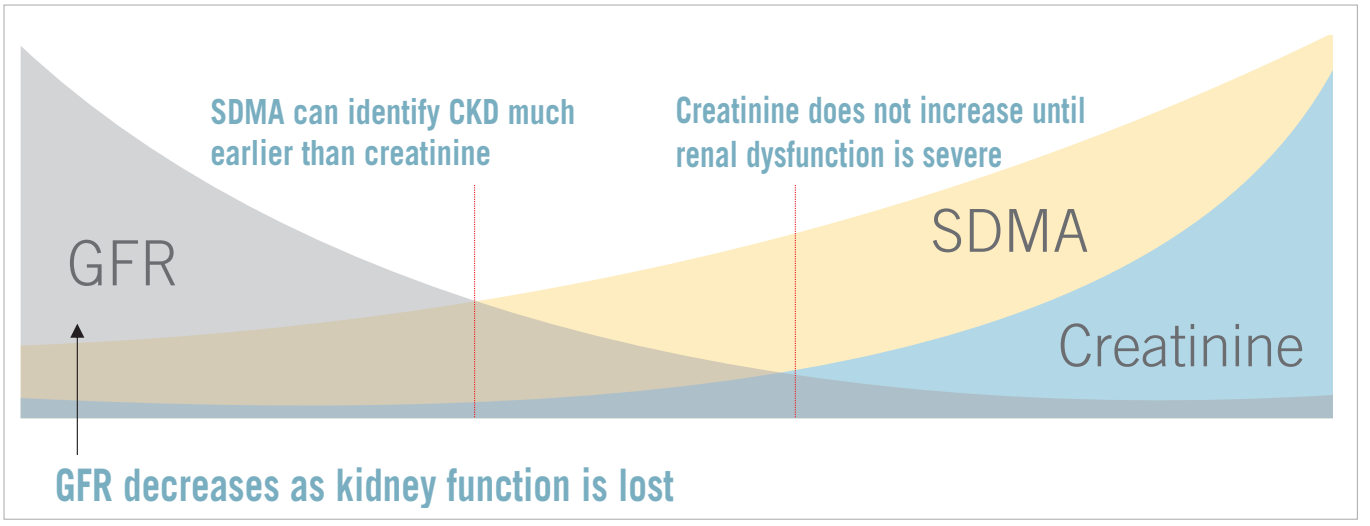
Being and earlier indicator of renal disease than creatinine is also a very promising feature. Studies have shown that in average SDMA is significantly increased when 40% of the kidney function is lost as opposed to creatinine being increased when 75% is lost. SDMA has also been seen to be elevated when as little as 25% of the kidney function is lost.



This enables us to detect these sub-clinical CKD patients much earlier. Studies together with Hills showed that in average SDMA is increased 9 months earlier than creatinine in dogs and 17 months earlier than creatinine in cats. Seventeen months is a long time in a feline life.

Having this marker available now gives us the unique chance to start studying what will happen to survival if we start intervening earlier in the disease process.

The diet studies by Hills showed significant prolongation of life when animals in IRIS stage 2/3 disease were fed a restricted protein renal diet. We can now evaluate how much it then changes with earlier detection and intervention.



Availability and use in practice

IDEXX SDMA™ is currently available through the IDEXX reference laboratories worldwide. It is incorporated in any profile containing creatinine and also as a stand-alone test if one has done the initial work up in the clinic and wishes to add SDMA to the profiling.

When to measure SDMA?

As SDMA correlates with GFR the indications for measuring SDMA would be any patient where one wants to know what the GFR is. That could be be either to diagnose early CKD or to rule –out early CKD as a concurrent disease in other illnesses.

SDMA is a part of your diagnostic toolbox and should always be interpreted together with creatinine, urinalysis and the clinical picture. In significantly dehydrated animals, the GFR will be decreased because of the viscosity of the blood, hence SDMA will also be falsely elevated. Therefore if you have dehydrated patients, it makes better sense to wait until after re-hydration to measure SDMA.

Suggested patient types for SDMA measurement

- Pu/Pd patients without azotaemia where there is suspicion of CKD
- For sub-staging CKD according to the IRIS staging guidelines (see: www.iriskidney.com)
- In particular feline patients with vague symptoms that could be related to CKD
- Patients with acute renal disease where it is desired to monitor progression
- Patients with lower urinary tract diseases where there is suspicion of ascending infection
- Senior patients where preventative care and wellness blood-testing is being done
- Patients in treatment with NSAID or other drugs that can potentially damage renal function
- Patients with Pu/Pd of other causes where one wishes to rule out concurrent kidney disease
- Hyperthyroid cats before and during medical treatment
- Pre-anaesthetic screening in at-risk patients
- Monitoring of at-risk patients (certain breeds)

Experiences from other countries

IDEXX SDMA™ was released in the US in the summer of 2015. Since then more than a million SDMA tests have been performed. Looking at the data gained form this, an even higher prevalence of renal disease than previously known has been demonstrated, supporting the theory that renal disease is underdiagnosed.

The majority of the cases detected with SDMA are IRIS stage 1 and 2, thus they would not necessarily be diagnosed based just on a creatinine level.

In particular in the older patients we see a higher prevalence of tests results where SDMA is elevated and creatinine normal. This is most likely due to the fact that older patients lose muscle mass, and thereby creatinine is lowered but renal disease is in fact present.

SDMA is a new test and we are still learning. A great deal of research continues world-wide and currently there are studies being performed on topics such as: SDMA and the hyperthyroid cat, SDMA and cardiac function, SDMA in acute renal failure together with other acute renal biomarkers, SDMA as predictor of bone marrow suppression in patients undergoing chemotherapy etc.

The results so far are very promising and the coming years will show how this early diagnosis affects the life of renal patients.

The Clinical Utility of Specific Canine Pancreatic Lipase (Spec cPL™)

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Pancreatitis is a relatively common disease with very non-specific clinical signs such as abdominal pain, anorexia, vomiting and diarrhoea. Currently there is no very specific and sensitive test available to use as a gold standard for diagnosis. Pancreatitis may present as acute and fulminant or be more low grade and chronic in nature. These forms will present with different clinical signs and also cause a different degree of cell damage and resultant lipase release by the affected organ, depending on the degree of inflammation, amount of fibrosis and number of cells remaining and affected.

In a case series of canine random consecutive post mortems the most common pancreatic lesion was hyperplastic nodules (80.2%), followed by lymphocytic inflammation (52.5%), fibrosis (49.5%), atrophy (46.5%), neutrophilic inflammation (31.7%), pancreatic fat necrosis (25.7%), pancreatic necrosis (16.8%), and oedema (9.9%). The most common lesion, lymphocytic inflammation, was mild in most cases. This could help explain why histologic lesions may be more prevalent than clinical signs of pancreatic disease.[5]

Clinically normal dogs have low circulating concentrations of pancreatic lipase in the blood. On the other hand, dogs with acute pancreatitis typically show dramatic elevations in serum pancreatic lipase concentrations, which remain elevated for a prolonged period. Animals with low-grade, histopathological disease may not show any dramatic elevations in serum lipase and may even exhibit no clinical signs.[5] Pancreatitic lipase can be determined by either the spec cPL or SNAP cPL test. The former is a laboratory run test, whereas the latter is a point-of-care in-house test.

Spec cPL is generally considered the most sensitive and specific test currently available for pancreatitis as the specific lipase only occurs within pancreatic tissue, as it is an enzyme that is synthesized and released only by the pancreatic acinar cell. Assays that measure PLI are species-specific. However, no test is perfect

Sensitivity of a test means "positivity in disease", which means a sensitive test has no or few false negatives. The more sensitive a test the more likely you are to get false positives if your test population is not properly selected.

Specificity means" negativity in health" - which means few or no false positive test results. The more specific a test is the more likely you are to get a false positive in a poorly selected test population.

With the Spec cPL test: We know that histopathological pancreatic inflammation is present in many animals, which have mild or no signs of disease. So if we use histopathological changes as the gold standard for pancreatitis the test may be very specific (very few false positives) but if we were to use "clinically confirmed" pancreatitis using best available clinical, biochemistry and abdominal ultrasound evidence as our gold standard the specificity will be lower, more positives in the absence of clinical disease. This makes sense as we know some animals walk around with mild inflammation and no clinical signs. These numbers are not written in stone as they vary depending on what they are measured against.

and the spec cPL can give both false positives and negative results.[1]

SNAP cPL should be performed in any dog with acute signs of gastrointestinal disease which does not have an obvious diagnosis such as intestinal parasites, foreign body, Parvo-virus infection, etc. The SNAP test only gives either a negative or positive result and is slightly more sensitive but less specific than the Spec cPL. If the test is negative then pancreatitis can essentially be ruled out as the sensitivity of the SNAP is 93% which gives a 7% chance of a false negative.[4] A positive SNAP cPL indicates a lipase value of ≥ 200ug/L. However, as a positive Snap cPL, is NOT a 100% diagnostic positive diagnosis for pancreatitis, it is still the responsibility of the clinician to perform a full diagnostic workup. Ideally an abdominal ultrasound should be performed to exclude other abdominal conditions which could cause similar clinical signs and also cause lipase release.

In addition, measurement of Spec cPL should be done to help confirm diagnosis of pancreatitis. The Spec cPL is a quantitative test with 3 diagnostic ranges: normal reference range (< 200 ug/L), questionable range (between 200-400 ug/L), and diagnostic cut-off for pancreatitis (≥ 400 ug/L) (IDEXX package insert). The magnitude of elevation of serum canine pancreatic lipase concentration does, however, not establish a prognosis for a patient with acute pancreatitis nor is there evidence that changes in serum concentrations correspond to clinical improvement.[1]

The sensitivity of Spec cPL in various studies ranges from 21% in dogs with histopathologically confirmed mild pancreatitis; 71% in dogs with severe pancreatitis; and up to 87% in some cases.[4] It would appear that the greater the pancreatic inflammation the greater the test sensitivity, which would make sense as more lipase is release into the bloodstream and these dogs have more severe clinical signs thus the prevalence in the test population is higher. Thus there is about a 15-25% possibility of having a false negative diagnosis. The Spec cPL sensitivity has also been measured against an increase between > 200- 399 ug/L (90%) and an increase > 400ug/L(75%) thus it decreases as the cutoff increases - but the specificity will increase with an increased cutoff. The specificity is 74% for the SNAP, 72% for Spec cPL cutoff between 200 -399ug/L and 78% with a cutoff > 400ug/L.[4]

Spec cPL cannot differentiate between primary and secondary pancreatitis — some dogs with an inflammatory disease process in another organ in the region of the pancreas (liver, gall bladder, mesentery, peritoneum) may have secondary pancreatic inflammation and animals with primary intestinal disease (foreign body, inflammatory bowel disease, lymphoma) may show elevated lipase values.[1,2]

Two recent studies investigating risk factors for pancreatitis showed that many dogs had co-existing disease and that there appeared to be an association between the presence of Cushing's disease and the development of pancreatitis. Dogs with Cushing's disease and no clinically diagnosed pancreatitis had a higher Spec cPL concentrations and more positive SNAP cPL results than clinically healthy dogs with normal ACTH stimulation test results. Spec cPL test concentrations were significantly higher in dogs with Cushing's disease (491.1 ug/L) than in healthy dogs (75.2 ug/L), with more abnormal Spec cPL results in Cushing's dogs. There were more positive SNAP results in dogs with Cushing's disease (55%) than in healthy dogs (6%).[3]

In another study, SNAP cPL and Spec cPL was evaluated in 3 groups of dogs: healthy dogs (n=20), those with signs of Cushing's disease but normal ACTH stimulation test results (n=12), and dogs with confirmed Cushing's disease (n=20). Dogs were excluded from the study if they had any clinical signs suggestive of pancreatitis. Healthy dogs had one SNAP cPL positive test and one Spec cPL in the 200-400g/L range. In the second group, 3/12 dogs had a positive SNAP cPL and 2 dogs had Spec cPL consistent with pancreatitis. In dogs with confirmed Cushing's disease, 11/20 had a positive SNAP cPL and 5 had a Spec cPL

at the 200-400g/L range, and 7 had values >400g/L. Yet none of the "positive" animals had clinical signs consistent with pancreatitis.[5] However, as it has been reported that low grade pancreatitis can be present as a post mortem histopathological diagnosis it is unknown if these dogs had low grade clinical pancreatitis (no biopsies were performed) or if something else was causing the elevation in the lipase.[3]

Miniature Schnauzers with severe hypertriglyceridaemia (> 9mmol/L, 900mg/dL) were 4.5x more likely to have a Spec cPL value consistent with pancreatitis (≥200 µg/L)[4]; however, these dogs did not show any obvious clinical signs of pancreatitis.[6]

Pending further study, SNAP and Spec cPL test results should be interpreted cautiously in dogs with Cushing's disease and hypertriglyceridaemia to avoid a false diagnosis of concurrent pancreatitis.

In one study[4], dogs were enrolled based on clinical signs of pancreatitis and the study showed that the SNAP test had a sensitivity of 91-94% and specificity of 71 – 77%. Using a cutoff of 400ug/L the Spec cPL had a sensitivity of 71-77% and a specificity of 80 – 85%.4 Another study also using a clinical diagnosis (clinical, serum chemistry, abdominal ultrasound) of acute pancreatitis in a group of dogs presenting with acute abdomen, the sensitivity of the SNAP cPL was 82% (18% chance of false negative result) and specificity 59% (41% false positive results).[1] The false positive result dogs may have had pancreatic inflammation due to diffuse abdominal inflammation due to the underlying disease process such as septic peritonitis, conditions causing hypo-perfusion or reperfusion injury, acute gastroenteritis, duodenal reflux and intestinal foreign bodies. In that study, the Spec cPL had a sensitivity of 70% and a specificity of 77% (23% false positives) with the accuracy of the SNAP and Spec cPL in animals with clinically diagnosed pancreatitis calculated at 65% and 75%, respectively.

It is thus evident that is it important to have a positive SNAP confirmed by the Spec CPL test at a laboratory.

Other conditions where Spec cPL is elevated include *Babesia rossi* where 28% of hospitalised cases had a level >400ug/L, patients with more advanced mitral valve disease and heart failure had minor increases (200 – 400 ug/L) and in and a small percentage of dogs on phenobarbitone and potassium bromide treatment which had increases between 200-400ug/L in 13/310 cases and increases >400ug/L in 9/310 dogs.[7,8,9]

Take Home Message

- A negative SNAP test makes pancreatitis highly unlikely and thus consider other causes for the clinical presentation.
- The Spec cPL and SNAP tests are more sensitive and specific in acute pancreatitis.
- Even if it is a true positive result and the dog does have pancreatitis, it does not mean that it is the only condition present. Pancreatitis can be both primary and secondary and neither the SNAP nor the Spec cPL can differentiate between the two. Secondary causes of pancreatitis such as septic peritonitis or intestinal foreign body will need to be specifically addressed. These tests are meant to be PART of the diagnostic evaluation and not the initial test to place the patient into a pancreatitis positive/negative group.
- There are some other disease conditions which appear to cause false positive results and thus decrease the specificity of the test. Cushing's disease, chronic glucocorticoid treatment and elevated triglycerides in Miniature Schnauzers have been shown to cause increased numbers of positive Snap and Spec cPL results in the absence of clinical pancreatitis. Thus a positive SNAP and Spec CPL is not diagnostic for pancreatitis.

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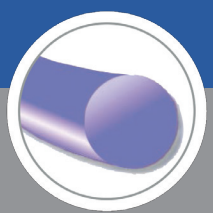

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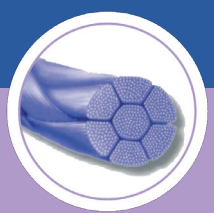

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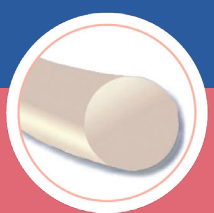

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