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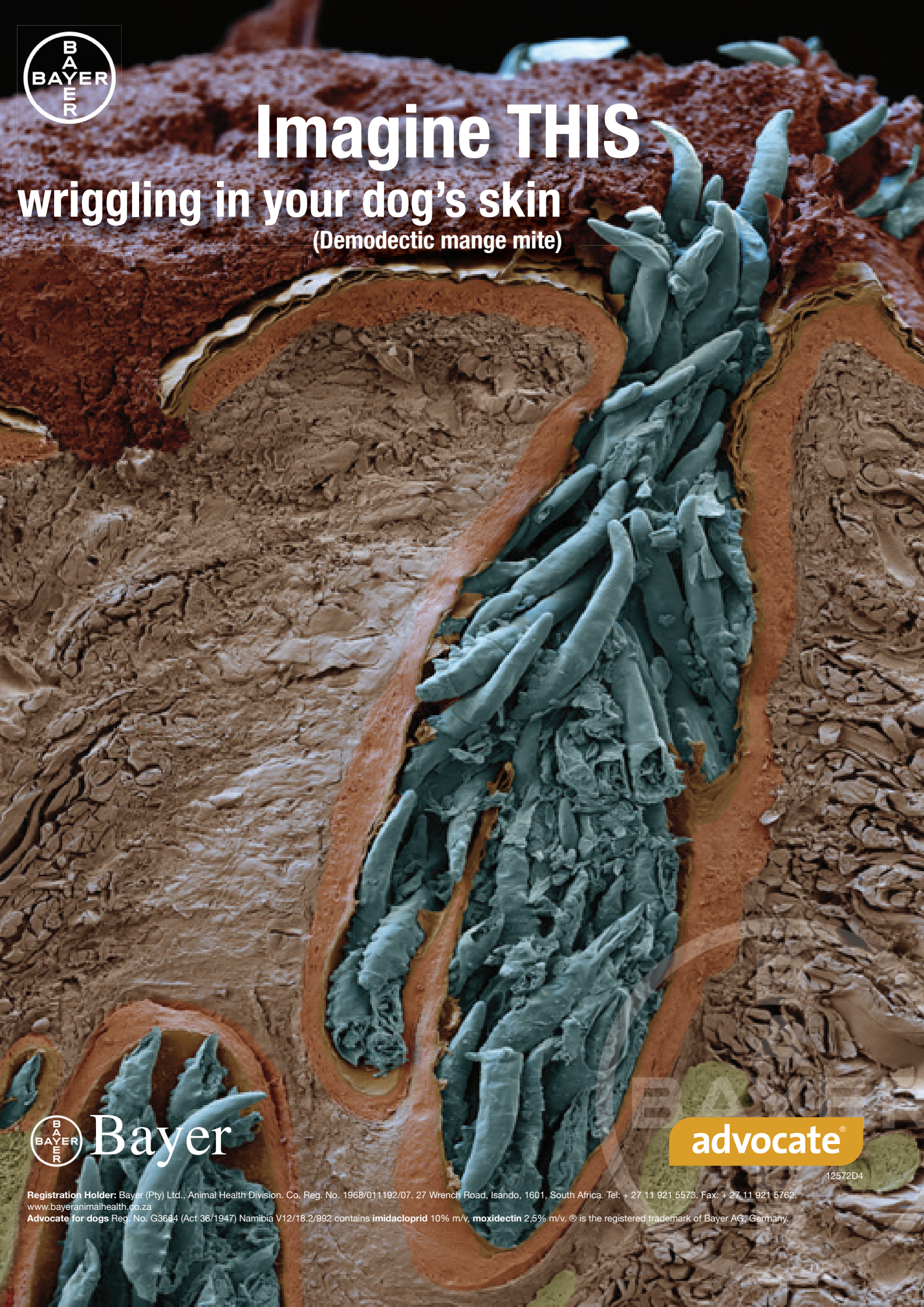
Diagnosis and Treatment of Demodicosis in Dogs and Cats

10 Most Common Pricing Errors

Replacement of the Prolapsed Third Eyelid Gland

Also in this issue:

Why Some Senior Cats Lose Weight | Unclogging The Constipated Cat



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Editor's Note

This edition is full of practical articles on the approach and treatment of common clinical problems. The article on skinny older cats by Dr David Williams is a real eye opener with regards to their nutritional requirements, over and above that required by specific disease conditions. We will go into more detail on this in the next edition.

Cats with constipation present commonly and the causes are varied. The article provides differential diagnoses as well as management tips. The CPD article on the diagnosis and management of canine demodicosis has been expanded upon by Dr Heidi Schroeder and myself and I have also included guidelines from the SAVC on the extra-label use of medications.

I would like to urge readers to continue to give feedback and also comment on content in the form of letters to the editor (editor@vet360.co.za). Please note these letters will need some experiential or scientific backing. Virbac will be assisting with distribution from March as a service to veterinarians - so thank you to them!

Liesel

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The 10 Most Common Pricing Errors Made by Vets

(Full article on Vet360 App or www.vet360.vetlink.co.za)

What is the biggest mistake a veterinary practice can make? This is a question often raised in veterinary discussion forums: congresses, management courses, informal meetings between practice owners...

Probably there is no one correct answer but in the author's opinion the greatest scourge of our sector is an incorrect pricing policy.

Pricing is a complex problem with several possible approaches. Some argue that vets charge little because they have low social status unlike those in other professions. Others state that the excess of veterinary surgeons on the market makes it unfeasible to charge decent professional fees. There are others who also think that the excessive demands on this profession (the vet feels obliged to know about dogs, cats, rabbits, cardiology, traumatology, infectious diseases..) generate a feeling of insecurity in the professional. This ends up resulting in low self-esteem and consequently in having a complex when it is time to charge for services. Some believe that the general ignorance of the profession about the true cost of things also does not help matters. Others even propose the hypothesis that the veterinary profession is so conspicuously vocational that money takes second place, to the extent that vets feel bad about having to charge for their services.

All these are plausible theories and deserve at least the benefit of the doubt. The reality is probably a mixture of these and other related factors. What at least seems clear is that if the profession continues to behave as it has done thus far, the best it can hope for is to obtain the same results.

The rest of the chapter offers a pragmatic view of this problem. It does not try to diagnose the causes, it simply lists a series of errors with respect to prices commonly found in many veterinary practices and suggests tools for those who want to try to obtain different results.

1 To think that most clients make decisions based mainly on price

All the studies carried out on this issue show that this is not true. In a study done in the United States[1] on a thousand households with pets it was found that:

- Only 24% of households with pets had ever changed veterinary practices over the life of their pet.
- Of that 24% that had changed at some point, just 15% (i.e. Less than 4% of total households) did it "because the previous vet was too expensive".

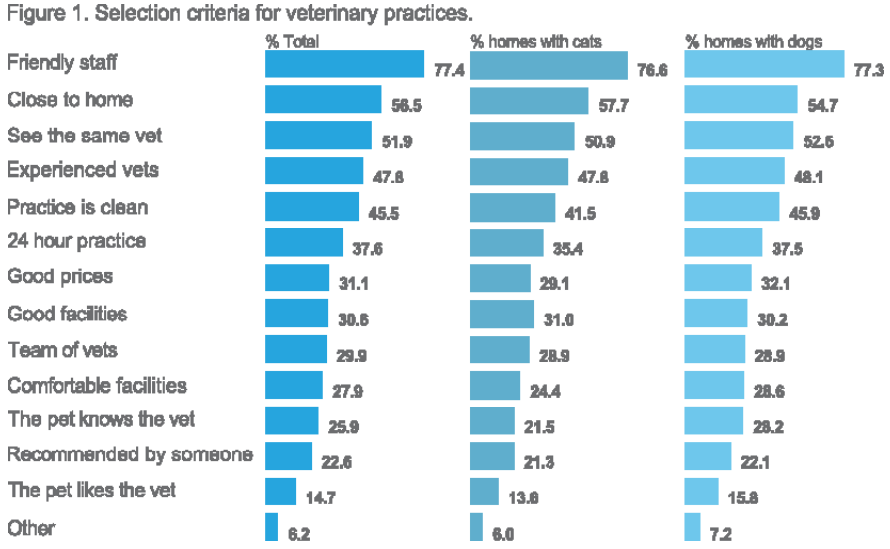
In Spain an extensive study[2] was also carried out which surveyed more than 1,200 households with pets. Some of the findings were:

- Only 19.6% of the households surveyed had changed vet at some point over the life of the pet.
- Of these, 11.6% (i.e. just over 2% of total households) had done it "because the goods sold by the practice were too expensive".
- Price was mentioned in seventh place as a reason for choosing the veterinary practice (figure 1).

Regardless of what the studies show, one only needs a bit of common sense to appreciate that price is not the main deciding factor for most consumers. We only have to look at the restaurants, health clubs, dentists, solicitors, hairdressers *etc* in our local area to realise that there are a number of suppliers offering a range of services at varying prices. There are clients for each one of them (or the same client may choose one or the other depending on the circumstances surrounding the purchase).

Price is just one of the ways to compete, but fortunately it is not the only one. Moreover, the bad news for those that decide to compete on price is that in every economic sector analysed, we find the same pattern:

Figure 1. Selection criteria for veterinary practices.



Note: The percentages add up to more than 100% because responders were allowed to choose more than one answer

There can only be one leader in low prices (for obvious reasons: there is only room for one leader, the rest follow). Therefore, the veterinary practice that leans towards this strategy will always be at the mercy of the next one wanting to do it. The position of leader in low prices is only sustainable in the medium term for those businesses that are also low cost leaders. This is a key point that many vets struggle to understand: economy of scale. In sectors with high fixed costs (like veterinary medicine), the large businesses (i.e. those that have more clients and therefore higher turnover) end up having cost advantage over the smallest ones. This happens because these larger practices or hospitals can dilute their fixed costs (website, advertising, reception, administrative costs, equipment) between a much larger number of clients.

This is not to argue that price is not important to customers. In fact, even for those customers whose decisions are based on other factors, price is a relevant question because everyone likes to think they use their money wisely.

2 Not understanding the nature of the running costs of a veterinary practice

Mark Oppermann[3] proposed a very interesting method to understand the real cost of providing a veterinary service. Most services provided in a practice contain three types of costs:

Veterinary costs: Let's do the following calculation:

A. **Annual practice salary costs for all vets.** To calculate this we will need to consider the cost to the business, not the net salary received by the vets. For the salary costs that relate to the owner/partners, we will have to include, for the purposes of this calculation, a salary at the market rate (for example that of the best paid vet at the practice).
B. **Annual total of chargeable "veterinary" minutes for the practice.** In order to calculate this figure we will set the number of working days per year and multiply it by the number of vets (including the owner(s), by eight hours a day and by 60 minutes per hour. In the case of part time veterinary staff, we will make the appropriate adjustment. Finally, we will multiply the number of resulting minutes by an efficiency factor of 70%. The logic of using this factor is as follows: not all the vet's working hours are directly dedicated to the generation of income. Telephone calls, case research and internal meetings are all examples of important tasks that can take up 30% or more of the vet's time. Therefore, we will multiply the total annual veterinary minutes for our practice by 0.7 in

order to calculate how many minutes are actually available to generate income.

C. Cost to the business per chargeable "veterinary" minute. This is the result of dividing A by B. It reflects how much it costs the practice each minute that one of its vets is available for a client.

Fixed or operational costs

Let's do the following calculation:

D. Annual operational costs of the practice. For this we will take the income statement from the last full year and take the total costs of the business as a starting point. From this figure we will subtract the total annual costs for vets (A) and also the cost of supplies (drugs, pet food and other shop items). The resulting figure will represent the operating costs of the practice (nurses, administration, receptionists, cleaning, facilities, IT) that enable the vets to do their job.

E. Operational costs per chargeable minute of the vet's time. As the reader may have already guessed, this figure is the result of dividing D by B. Its interpretation is how much it costs the practice to provide support in terms of infrastructure (facilities, support staff, *etc*.) for each vet for every minute that they are working for the clients.

Total annual income	450,000	%
1) Vet salary costs (3.5 people) (A)	112,500	25%
2) Purchase of supplies (drugs, pet food, consumables)	103,500	23%
3) Fixed operational costs (D) (facilities,non-veterinary staff)	166,500	37%
Profit	67,500	15%

Figure 2 (b). Cost per minute of the vet's time.

Number of vets (including owner)	3.5
Working days per year per vet	225
Working hours per year per vet	1,800
Working minutes per year per vet	108,000
Efficiency factor (% of strictly chargeable time)	70%
Actual chargeable minutes of veterinary time per year (total for practice)	264,600
Salary cost to the practice per actual chargeable minute of veterinary time	0.43
Operational cost per actual chargeable minute of veterinary time	0.63
Total cost to the practice of each chargeable minute of veterinary time	1.06

Figure 2 (a). Income statement . This calculation only reflects the cost of the service, without any room for profits (margin) and without adding VAT.

Variable costs (cost of supplies)

This refers to the cost of drugs, swabs, vials, wormers, flea control, pet food and all the other things we use to provide a veterinary service. We must keep in mind that the cost of these supplies is not just the cost of buying them but also the financing cost to the practice (the money invested in this stock could have been invested in a bank account paying out interest), the cost of stock losses and things going out of date (it is inevitable that a certain percentage of our stocks will be lost or will expire).

Figure 2 shows the calculation of veterinary costs and operational costs for a medium-sized veterinary practice, based on a fictitious income statement (but that could reflect the reality in many practices). The reader is encouraged to adjust these calculations using real figures from their own practice and to reflect on the result... Are we charging enough for every minute of our vets´ time?

3 To make prejudgments about our clients and make financial decisions on their behalf

A very common mistake among vets is to prejudge how much an owner is willing to invest in the health of their pet based on their appearance.

Judgments are made according to the way the owners are dressed, the car they drive or the area where they live. No studies have shown a correlation between the level of income and the willingness to spend more on the health of the pet. The key is in the affective bond between the owner and their pet. Vets should not be judges of the affective bond between clients and their pets nor their financial advisors. Clients want to make informed decisions. What they ask of us is that we inform them of the options available and, if possible, recommend one.

A revealing experiment was done in the United States[4], in which a clinical case was proposed to a group of vets. Half the participants were told that the pet owner was an elderly widow of modest means, whilst the other half were told that the owner was a young professional. Next the vets were asked to prepare a treatment plan for the patient, and an estimate for the client. The interesting thing about this experiment is that the vets varied significantly in their clinical approach to the case based on the information they had available about the pet owner. That is to say non-clinical considerations prevailed over clinical ones. Were these vets behaving like clinical vets, or like veterinary economists?

4 To mistake value with price, spending too much time talking about price and very little communicating value

Put simply, we could say that most financial decisions taken are based on two criteria:

- Value (what we get if we make that decision). This can be seen in terms of receiving goods, solving a problem, feeling happier...
- Price (what we must pay if we take that decision). This must not be strictly considered just in financial terms but also as working time, concern, inconvenience...

If the value of a decision exceeds its price it becomes attractive and we will probably take it. We will compare it with other available options and under normal

conditions will choose that with a bigger difference between its value and its price.

In the veterinary practice world, the most common error is to spend a lot of time and effort in arguing with clients (and with our own employees) about the price of services and too little talking about the value we provide in return. Figure 3 outlines a service that offers value to the client.

5 To give money away to clients (and what is worse, without them even realising)

It is estimated that in an average American veterinary practice, the annual amount of uncharged items can amount to more than 40,000 dollars. This may be due to an inappropriate discount policy, a poor administrative system used to account for services provided, or to a combination of both.

In the case of discounts, it would be a good idea if everyone at the practice asked themselves the following questions:

1. How much money did we discount in our practice over the past 12 months?
2. Who are we giving these discounts to? Animal charities? Pensioners?
3. Those on low incomes? Other types of people? Why are we giving these discounts? For charitable reasons or because we have planned to do it that way? Are we perhaps giving them because we made a mistake when preparing the estimate? Is it because we dare not defend the way we have treated a case in front of a dissatisfied customer and think this is the way to appease them? Or is it simply because one of our employees has decided that our price is too high and is giving a discount with our money?

If we do not feel comfortable answering these questions, we must rethink our practice's discount policy. To have a discount policy means having the answers to the following questions in writing:

1. Who is authorised to give discounts at our veterinary practice?
2. To whom and in which specific situations may discounts be given?
3. How much can be discounted?
4. How should discounts be recorded and who should be informed when a discount has been given?

References will be published in Part 2.

IN THE NEXT ISSUE: To give money away to clients; Not giving value to time; Not understanding the impact of inappropriate pricing policy; Not knowing how to communicate to the team importance of proper pricing; Victim mentality; Thinking that profitability and good medical care are incompatible. www.vet360.vetlink.co.za

Figure 3. Offer of a service with value to the client.

PROCEDURE: CAT OVARIOHYSTERECTOMY (EXAMPLE)

- **Pre-anaesthetic clinical exam**
  This involves a full clinical exam carried out by the veterinary surgeon. It involves checking the patient's weight, body temperature, heart and breathing rates. In addition, the clinical status of the patient is also determined by checking the skin, coat, mucous membranes, limbs, pads, abdomen and gastrointestinal and urogenital tracts.
- **Pre-anaesthetic profile**
  The purpose of this is to check that the organs responsible for the metabolism and excretion of the anaesthetics work properly. We will check hepatic, renal and pancreatic functions. The blood and urine of the pet will be tested to rule out anaemia or infections.
- **Anaesthesia**
  This includes the premedication, induction and anaesthetic maintenance using gas anaesthesia. The patient's breathing and cardiac functions are permanently monitored using electronic equipment.
- **Ovariohysterectomy**
  This surgical procedure consists in entering the abdominal cavity by making a midline incision. Then finding and removing the left ovary (found by the left kidney) by ligating the ovarian blood vessels, dissecting them and the ovarian ligament. Then finding and removing the right ovary (found by the right kidney) after ligating its blood vessels, dissecting them and the ovarian ligament. After that the uterine cervix will have to be located and its blood vessels will have to be ligated. Once this is done the uterine body is resected and removed. The incision in the abdominal wall will be closed by suturing all the layers one by one. By the end of the surgery the ovaries, uterine horns and uterine body will have been removed.
- **Post-operatory**
  During the post operatory phase the wound will be looked after and the patient will receive analgesia and antibiotics. Ten days after surgery the sutures will be removed.

Note: The description of this service has been written by Oscar Cortadellas (DVM,PhD) (Clínica Veterinaria Germanías).

Diagnosis & Treatment of Demodicosis in Dogs & Cats

By Karen A. Moriello, DVM, Diplomate ACVD
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PROFILE

Definition

Demodicosis occurs when Demodex mites, which are part of an animal's normal flora, proliferate in the skin (most often the hair follicle).

Causes

- Three species of Demodex mites affect dogs: *D canis, D injai* (long-bodied mite; Figure 1), and *D cornei* (short-bodied mite).[1,2]
- Two species affect cats: *D cati* (long-bodied mite) and *D gatoi* (short- and wide-bodied mite; Figure 2).[3]

Signalment

- Demodicosis can develop at any age.
- No sex predilection is seen in dogs or cats.
- Breeds at highest risk for developing juvenileonset generalised demodicosis due to *D canis* include Shar-peis, Pit Bulls, Boston Terriers, English Bulldogs, Boxers, Miniature Pinschers, Great Danes, and Pugs.[4]

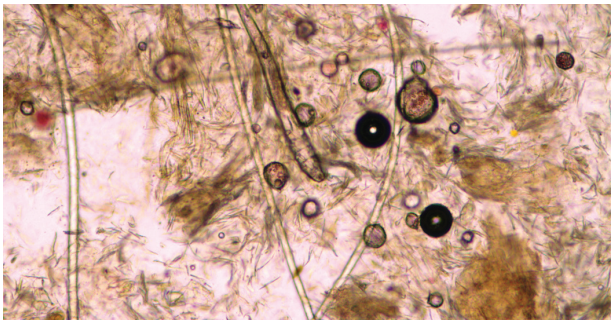

Figure 1: Long-bodied canine Demodex mite (original magnification, 1000×)

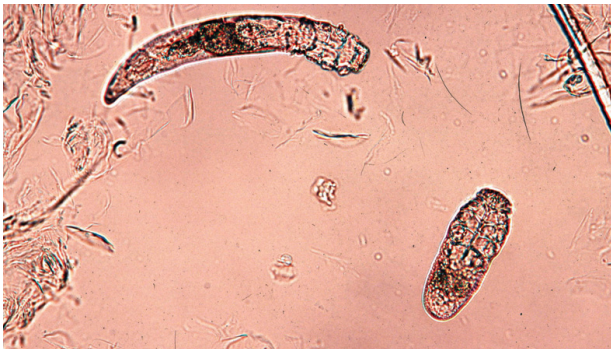

Figure 2: Feline Demodex mites: D cati and D gatoi (original magnification, 40x)

- Emerging evidence suggests that wire-haired Fox Terriers, and Terriers in general, may also be predisposed to demodicosis.[4-6]
- There is no recognised breed predilection in cats.

Risk Factors

- Risk factors that affect both dogs and cats include chronic severe disease states, neoplasia, long-term glucocorticoid use, and chemotherapy.
- In dogs, risk factors for juvenile-onset generalised demodicosis include pyoderma, coccidiosis, hookworms, short hair coat, and lack of participation in a preventive care wellness plan.[4]
- Focal demodicosis is common in puppies, and physiologic stress and debilitation are risk factors.
- In cats, *D gatoi* is contagious. Cats at risk for this infestation tend to come from high density populations.

Pathophysiology

- Clinical disease results from overproliferation of mites in the skin due to defects or compromise of the skin's immune system.
- Studies using dog leukocyte antigen class II have identified common markers in young dogs with generalised demodicosis, suggesting that this antigen may be an important immunologic risk factor for the disease in dogs.[7]
- Demodex mites found on skin scrapings, plucked hair, ear swabs, and faecal samples are considered clinically significant when interpreted in conjunction with typical clinical signs.[8]

Signalments and Clinical Signs

DOGS

Localised demodicosis

- Occurs primarily in puppies.
- Mites are found only in lesional areas; lesions focal and limited (1–4 sites).
- Signs include focal areas of hair loss, erythema, hyperpigmentation, and follicular plugging (comedones).
- Pruritus varies.

Juvenile generalised demodicosis

- Mean age of onset is 6 months.

- Begins as localised demodicosis and becomes more diffuse
- Mites can be found in lesional and non-lesional areas; all stages of life cycle are commonly found
- Signs include hair loss, erythema, hyperpigmentation, papular rash, follicular plugging, concurrent superficial or deep pyoderma (furunculosis), pain, fever, matting of hair coat, and skin exudation
- Pruritus is variable
- In some patients, presents only as deep pododermatitis (swelling, lameness, pain, exudation, lichenification, proliferation of pedal tissue) and generalised or regional lymphadenopathy
- Dogs can be septic and severely debilitated by disease

Adult-onset demodicosis
- May be focal or generalised
- Occurs in dogs with concurrent systemic illness
- Proliferation of mites may precede signs of systemic illness
- In my experience, dogs with adult-onset demodicosis have no history of demodicosis as a young dog

D injai demodicosis
- Associated with wire-haired Fox Terrier dogs that have dorsal greasy skin of the trunk
- Usually pruritic and may be concurrent with atopic dermatitis[6]
- Some dogs, especially terriers, may present with intense pruritus[7]

CATS

Otic demodicosis
- Affects cats of any age; common in kittens
- Signs include ceruminous discharge and pruritus

D gatoi demodicosis
- Can occur in cats of any age
- Common signs are pruritus and evidence of contagion
- Can present as symmetric alopecia
- Pruritus can be severe and lead to self-trauma
- Often introduced into household after adoption of a new cat or kitten from a rescue center, shelter, or other high-density population

D cati demodicosis
- Not known to be contagious
- Signs can be similar to those of *D gatoi*
- Most often associated with systemic illness (eg, diabetes mellitus) or long-term use of glucocorticoids and progestins

Mixed infections
- Not uncommon

DIAGNOSIS

Definitive Diagnosis

Dogs
- Any finding of mites indicates demodicosis.[5,6] Mixed infestations of *D canis* and *D injai* can occur.
- Mites are found on deep-skin scrapings or via hair trichograms; the latter are useful for sampling sites that are close to the eyes or difficult to scrape (eg, interdigital areas).
- Hair plucking may be the diagnostic test of choice for sampling dogs with greasy hair.[5,6]

Cats
- With otic demodicosis, mites are found on mineral oil cytologic testing of ear exudates.
- *D cati* mites tend to be easily found on skin scrapings.
- *D gatoi* mites can be difficult to find even when the patient is severely pruritic. For these mites, suggested tests include wide superficial skin scrapings, hair plucking, faecal flotations (**Figure 3**), or response to therapy.

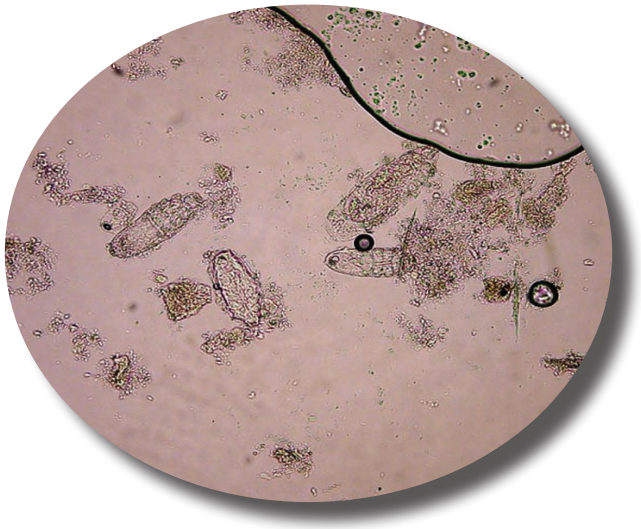

Figure 3: Demodex gatoi mites found on faecal sample from cat with pruritus, self-trauma, and hair loss on ventral abdomen (original magnification, 400×)

Differential Diagnosis

- Dogs: Canine demodicosis can mimic any skin disease; the rule of thumb is "demodicosis until proven otherwise."
- Cats: Consider demodicosis in any cat with hair loss, symmetric alopecia, or pruritus.

Laboratory Testing

- **Bacterial cultures of skin:** Should be performed if deep pyoderma is present or if there is a history of glucocorticoid use or long-term antibiotic therapy (dogs)
- **Complete blood count or serum biochemical profile:** In dogs with deep pyoderma that may be septic or dehydrated
- **Faecal flotation examination:** In pruritic cats to identify *D gatoi*
- **Genetic testing for ABCB1-δ genotype:** To screen for drug sensitivity to avermectins in breed-sensitive dogs (eg, herding dogs, sight hounds); rec-

ommended for dogs with severe generalised demodicosis[3]
- Impression smears: To diagnose concurrent microbial overgrowth in dogs and cats

Additional Laboratory Testing

1. Except for faecal flotation examinations in cats, laboratory testing is most helpful when searching for the underlying cause of adult-onset demodicosis in dogs or a medical condition associated with *D cati* in an adult cat. Testing may include but is not limited to:
    - Blood smear evaluation
    - Complete blood count
    - Faecal flotation
    - Infectious disease titers
    - Radiography of thorax and abdomen
    - Retroviral screening
    - Serum biochemical analysis
    - Urinalysis.
2. Fine-needle aspirates of lymph nodes may contain mites.
3. Skin biopsy may be helpful in Shar-pei dogs and dogs with severe pododermatitis.

TREATMENT

Inpatient or Outpatient

- Demodicosis in dogs and cats can be treated on an outpatient basis.
- Concurrent bacterial/yeast overgrowth must be treated so that therapy does not fail. If there is a lack of appropriate response, the skin should be cultured to rule out a possible methicillin resistant *Staphylococcus intermedius* group (SIG) infection.
- Dogs with adult-onset demodicosis and complications from underlying disease or dogs with deep pyoderma, fever, and sepsis may require hospitalisation for supportive care and diagnostic testing. Hospitalisation of cats is rare and is related to medical issues underlying *D cati* infestations.

Medical

- Treat fever, pain, sepsis, and dehydration in dogs with concurrent deep pyoderma.
- Provide pain medication for dogs with pododemodicosis if needed.
- Recommend sedation and clipping of the hair coat (especially long-haired breeds) to facilitate medicated bathing.
- Institute aggressive antimicrobial therapy pending culture and sensitivity.
- Initiate concurrent topical antimicrobial shampoo therapy (eg, benzoyl peroxide, chlorhexidine).
- Monitor patients with severe generalised demodicosis during initial therapy for development of peripheral edema; systemic miticidal drugs can cause massive mite kills and obstruction of lymphatics.

Nutritional Aspects

- Ensure that clients are feeding complete, balanced, age-appropriate diets, especially if the pet's body condition is poor.

Client Education

Dogs

- Explain that localised demodicosis may progress to generalised demodicosis in about 10% of affected dogs.
- Clients need to thoroughly understand the cost and duration of treatment, especially for juvenile generalised demodicosis, and the possibility of relapse or lack of cure.
- Emphasise the need for a thorough workup of dogs with adult-onset demodicosis and the implications of underlying disease; provide the pros and cons of treatment options.

Cats

- Explain the contagious nature of *D gatoi* and the need to treat all in-contact cats.
- There is a strong likelihood of an underlying predisposing disease in cats with *D cati*, but cost of evaluation to uncover the cause needs to be considered.
- Emphasise the pros and cons of treatment.

MEDICATIONS

Dogs

Amitraz[1,2]

- Product availability is variable.
- Use once weekly (extra-label use) by sponging onto the whole body; do not rinse off; apply thoroughly; do not let dog become wet between treatments.
- Clip long-haired dogs to maximise contact with skin.
- Do not use on dogs with deep pyoderma or open areas of sloughed skin.
- Do not use concurrent monoamine oxidase inhibitors (MAOIs), clomipramine, selegiline, selective serotonin-reuptake inhibitors (fluoxetine, sertraline paroxetine), tricyclic anti-depressants (clomipramine, amitriptyline), opioids, or such over-the-counter medications as dextromethorphan.[3]
- Apply the product in the veterinary clinic; use good ventilation. Ensure that individuals applying the solution do not have respiratory disease or blood glucose issues, are not pregnant, and are not taking MAOIs.
- Adverse effects include pruritus, polyuria/polydipsia, sedation, tremors, collapse, and hypothermia.
- Use yohimbine to treat toxicosis.

Metaflumizone plus amitraz is labeled for the treatment of demodicosis in veterinary patients, but the

manufacturer has made the decision to discontinue the manufacture and sale of this product. There have been rare pemphigus foliaceus–like drug reactions associated with the use of this drug combination.[4]

Ivermectin (extralabel use)
- 300 to 600 µg/kg *po* q 24h
- Aqueous formulations are more palatable than propylene glycol–based formulations.
- Adverse effects include lethargy, muscle tremors, mydriasis, ataxia, severe neurotoxicosis (depression, stupor, coma, ataxia, seizures, death), and blindness.
- ABCB 1-delta gene (MDR1) testing can be used to screen for sensitivity.
- Do not use in ivermectin-sensitive dogs or breeds.
- Dogs should have a negative heartworm test result before use.[2]

Milbemycin oxime
- 1.5 to 2 mg/kg *po* q 24h.[2]

10% Moxidectin and 2.5% imidacloprid
- Can be used every other week; however, weekly applications appear to be more effective.[5,6]

Doramectin
- 600 µg/kg body weight SC once weekly. Do not use in ivermectin-sensitive dogs. Shown to be effective in 2 small studies.[7,8]

Cats

Feline otic demodicosis
- Topical ivermectin or topical milbemycin oxime[1,2]

Generalised demodicosis due to *D gatoi* or *D cati*[1,2]
- Lime sulfur (topical leave-on agent) safest treatment: use once or twice weekly for 6 weeks; higher concentration recommended for faster resolution (1:15 dilution in water; mix thoroughly; cats tolerate better if water is warm). Apply thoroughly (rose-garden sprayer can be used) and soak coat and skin.
- Do not rinse off solution. Keep cats warm. Use in well-ventilated area.
- Milbemycin oxime: 1.0 to 2.0 mg/kg q 24h. Well tolerated by most cats; can cause vomiting and diarrhoea and, rarely, neurologic signs.
- Aqueous ivermectin: 300 to 600 µg/kg orally q 24 h; can be mixed in canned cat food; neurotoxicosis may develop.
- Doramectin: 600 µg/kg once weekly by SC injection.[7]
- 10% Moxidectin and 2.5% imidacloprid: used in small number of cats anecdotally; administered weekly or every other week.

Response to treatment trial
- Treat all cats suspected of having *D gatoi* infestation for at least 6 weeks.

Precautions/Interactions
- Dogs without an ivermectin-sensitive genotype can show signs of toxicosis if ivermectin is given with P-glycoprotein inhibitors.
- Some more commonly used agents in veterinary dermatology include erythromycin, itraconazole, ketoconazole, cyclosporine, and tacrolimus. (Note: Oral tacrolimus use to date has been limited but may increase as this drug becomes more affordable.[9])
- In most cases, application of topical tacrolimus will not result in significant absorption. In humans, however, if the agent is used over large areas, significant absorption is possible.[10]
- Do not use glucocorticoids in these patients.

FOLLOW-UP

Patient Monitoring
- Treatment continues in dogs until at least 2 or preferably 3 consecutive skin scrapings are negative at 1- to 2-week intervals.
- The most common treatment error is stopping treatment too soon.

Complications
- Relapse of generalised demodicosis in dogs is not uncommon.
- Adult cats or dogs with demodicosis due to an underlying disease may not be able to achieve remission unless that disease is treated, cured, or controlled.

FUTURE FOLLOW-UP
- Dogs with generalised demodicosis will require lifelong monitoring for relapses. A dog is considered "cured" when no relapses have occurred for at least 1 year.
- Cats with *D gatoi* can be cured, and relapse is not an observed problem. *D cati* infestation will not resolve unless medical disease is treated or managed.

The approach to demodicosis and our emphasis in South Africa differs slightly to that in the article. We have outlined additional information according to the subheadings in the article. See page 12. Editor

Additional points on Demodicosis

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Edited - Dr L van der Merwe

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Causes:

Recent evidence has indicated that it is likely that all three forms of canine demodex mites are, in fact, *Demodex canis*. They all however, seem to respond in a similar manner to treatment protocols [1].

Signalment:

In South Africa, the Scottish Terrier and Boerboel also seem to be predisposed to demodicosis. Feline demodicosis is not very common but may be under-diagnosed. The risk factors should probably be expanded. In cases of adult onset demodicosis underlying causes include diabetes mellitus, hyperadrenocorticism (spontaneous or iatrogenic), hypothyroidism, chronic ehrlichiosis, neoplasia, chemotherapy, other immunosuppressive drugs and endo-parasitism. Many dogs with the above conditions never develop demodicosis though and furthermore in about half of the cases an underlying cause is never found. Where an underlying cause cannot be found - continue to monitor during treatment as neoplasia, for example, may only become evident later[1].

Definitive Diagnosis:

1. Skin scrapings: the number of mites per scrape (dead or alive), of each life stage should be recorded at each visit to help monitor response to treatment.
2. Trichograms (hair pluck) are used to find adult mites on hair shafts in areas difficult to scrape e.g. periorbital and interdigital areas. Hairs are plucked with a forceps and placed into a drop of mineral oil on a slide. Trichography is easy but is not as reliable as a skin scraping.
3. Skin biopsies for histopathological examination may be necessary when skin scrapings are negative for mites, but the clinical suspicion is high. This is often necessary for patients with thick skin, e.g. Shar-Pei dogs and in chronic pedal demodicosis.
4. Cytology of exudates can reveal mites in cases where mites are abundant. Secondary bacterial pyoderma is diagnosed where bacteria are found inside the neutrophils

Laboratory Testing:

Serology or PCR for *E canis* is indicated, as South Africa is an endemic region. The MDR -1 gene (multidrug resistant gene) is now called the ABDB-δ genotype. **Testing for this genetype is recommended for all dogs, but especially shelties, collies and other herding breeds being placed on macrocyclic lactone therapy.** Some products are registered as safe to use insensitive breeds, but especially those medications being used extra-label, may cause some side effects. Local laboratories which perform this test include **Inqaba Biotechnical Industries** and **Molecular Diagnostic Services (MDS)**

TREATMENT:

Amitraz:

Amitraz is an approved treatment for canine demodecosis. It is a topical medication applied as a rinse every 7-14 days. The recommended concentration varies between 0.025% - 0.06%. Clinical efficacy increases with shorter treatment intervals and increasing concentrations. A benzoyl-peroxide shampoo prior to the rinse is beneficial due to its follicular flushing and keratolytic "degreasing" effect.

Amitraz should be made up with warm water before each rinse – it cannot be stored in the made up concentration as it is rapidly oxidised and altered by ultraviolet light. Protective clothing and gloves should be worn and the medication applied with a sponge to soak the skin properly. The patient should be allowed to dry naturally and not be towelled off.

Studies have shown that amitraz rinses are less effective in adult onset demodicosis as only 33% of dogs showed a response in contrast to 66% of dogs with juvenile onset demodicosis[2]. Higher concentrations with shorter treatment intervals may be required in these cases. Daily topical footbaths with amitraz are indicated for dogs with pedal demodicosis. The use of amitraz is not recommended in younger dogs (<4-6 months) and signs of toxicity/adverse effects include depression, sedation, ataxia, bradycardia, polyuria, polydipsia, hypothermia and hyperglycaemia. Patients with diabetes mellitus should ideally not be treated with amitraz. Certain toy breeds e.g. Chihuahuas, are more likely to experience the side effects of this drug. Yohimbine, a α2-adrenergic antagonist at 0.1mg/kg im, is the antidote when side effects are severe.

Ivermectin

Ivermectin is the first choice for many dermatologists, although it is not licensed for use in canine demodicosis. It is easy to administer and is cost effective. The oral route is most effective and a number of studies evaluating daily administration showed good treat-

ment success[3]. This is in contrast to studies evaluating weekly injections of ivermectin, which gave variable and inconsistent results[4]. The most current practice guidelines recommend oral ivermectin at a dose of 0.3 – 0.6mg/kg daily (300-600µg/kg) for the treatment of generalised demodicosis. The lower dose of 0.3mg/kg is usually effective. Some veterinarians use the injectable ivermectin formulation orally. A pharmacokinetics study has determined that the terminal drug half-life, as well as mean tissue residence time is the same using the oral or SQ route of both injectable ivermectin as well as doramectin. In fact, the oral administration of the drug actually showed an increased maximum plasma concentration and more rapid absorption[5]. The advantage of this is that the volume of administration is much less as the injectable is more concentrated. However, there are no publications on the effect on efficacy. Two-thirds of dogs with adult onset demodicosis respond to ivermectin therapy. Due to the severe side effects, especially in herding breeds, it has been recommended to gradually increase the daily ivermectin dose over 5 days. A starting dose of 0.05mg/kg on day 1, 0.1mg/kg on day 2, 0.15mg/kg on day 3, 0.2 mg/kg on day 4 and 0.3mg/kg on day 5 is recommended. Mydriasis and ataxia are signs indicating ivermectin sensitivity.

Doramectin (Dectomax®)

Doramectin is reported as successful in treating canine demodicosis but is also not licensed for use in dogs. The recommended dose is 0.6mg/kg SQ once a week or 0.3mg/kg orally twice a week. The drug is not safer than ivermectin - thus the same incremental increase is recommended.

Milbemycin Oxime (Milbemax®)

Milbemycin is a licensed drug for the treatment of demodecosis and an alternative in ivermectin and doramectin sensitive breeds. The recommended dosage is 1-2mg/kg/day orally and it is therefore very expensive.

2.5% Moxidectin + 10% Imidacloprid (Advocate®)

A study has shown that the weekly application of Advocate® can be recommended as effective for the treatment for canine demodicosis without the potential for toxicity associated with ivermectin, where the 10mg/ml solution was used orally at 500ug/kg oid[6]. It is important to note that this product is not very effective against ticks.

When to stop treatment: Treatment should only stop when no parasites can be demonstrated on multiple skin scrapes taken 2 weeks apart. Clinical signs will subside long before this and the disease will relapse if treatment is stopped too early.

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CPD Questions

AC/1320/15

Question 1:
Which of the breeds listed below is NOT predisposed to juvenile canine demodicosis?
a. Scottish terrier
b. Labrador retriever
c. Pug
d. Shar pei
e. Bull dog

Question 2:
Which of the statements below is incorrect?
a. Demodex can be contagious is cats
b. Demodex can be contagious in dogs
c. Demodex is a normal inhabitant of the skin.
d. Chronic use of immunosuppressive drugs is a risk for developing demodicosis
e. Focal demodex is common in puppies

Question 3:
Which of the clinical signs listed below is NEVER present in juvenile demodicosis?
a. Patchy Alopecia
b. Superficial pyoderma
c. Deep pyoderma
d. Lymphadenopathy and fever
e. Muco-cutaenoeus dermatitis

Question 4:
Which of the statements below is **not of value** when making a definitive diagonsis of demodicosis?
a. Seeing all stages of parasites on a deep skin scraping
b. Seeing parasites in a faecal flotation
c. Seeing parasites on the hairshafts in a hair pluck
d. The presence of an eosinophilia in the blood smear.
e. Performing a skin biopsy

Question 5:
Which of the conditions listed below is NOT one which would predispose a patient to developing demodicosis?
a. Addisons disease
b. Chronic ehrlichiosis
c. Hypothyroidism
d. Canine cushings disease
e. Diabetes mellitus

Question 6.
Which of the following is NOT true of juvenile demodicosis.
a. Affects puppies at about 4-6 months of age
b. Short-haired breeds are predisposed.
c. The history of dogs with adult onset demodicosis normally shows an episode of juvenile demodicosis.
d. May present as localised or generalised demodicesis
e. May present only as severe pododermatitis

Question 7:
Which of the facts listed below does NOT apply to Amitraz?
a. Amitraz is registered for use in canine demodicosis
b. Amitraz is indicated as initial treatment in demodi cosis complicated by deep pyoderma
c. Amitraz is more effective if used after a benzoyl peroxide shampoo bath.
d. Amitraz has no known breed sensitivities
e. Amitraz causes depression and ataxia as a mild side effect

Question 8:
With regard to ACBC-δ-1 gene-deficient dogs – which of the statements below is incorrect?
a. Herding dogs, collies and sight hounds are sensitive breeds
b. Doramectin is safer than Ivermectin
c. The test to evaluate a patient for ivermectin sensitivity is available in laboratories in South Africa.
d. Gradual titration of the dose will help limit side effects
e. Moxidectin preparations are safer than the avermectins

Question 9:
Which of the following statements regarding demodicosis in dogs is correct?
a. Underlying disease conditions are found in all dogs affected with adult onset demodicosis
b. The recommended treatments amitraz and Ivermectin are 100% effective in curing the disease
c. Affected dogs have an inherent immunological defect allowing infection to occur, and should preferably not be used for breeding
d. Side effects of medication, if present, will decrease in severity during the first week of treatment.
e. The disease can always be managed on an outpatient basis

Question 10
Which of the treatments listed is the safest treatment for demodicosis in cats?
a. Topical ivermectin
b. Topical lime sulphur "leave on" agent
c. Topical milbemycin
d. 2.5% Moxidectin +10% imidaclopramide
e. Ivermectin per os

ANSWER the questions on the Vet360 App. Available from the Itunes/Play store!
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EXTRA-LABEL USE OF PHARMACEUTICAL PRODUCTS

Extracts from SAVC Code of Conduct

New rules regarding dispensing, compounding and extra-label drug use are currently being formulated by the SAVC. The Veterinary Code of Conduct does however address the issue and the relevant extracts are listed below.

7.1 EXTRA-LABEL USE OF PHARMACEUTICAL PRODUCTS

Definition: *Extra-label use: the use of pharmaceutical products in animals in different species for conditions, or in dosages and administration routes other than those indicated on the labels of these drugs.* (See Council Newsletter Volume 23 December 1998.)

The Registrar of Medicines advised that the Medicines Control Council (MCC) was of the opinion that the MCC cannot advocate the off-label/extra-label use of medicines. However, in the light of international acceptable practice to use medicines in such a manner, the right/obligation for the off-label/extra-label use of medicines by veterinary prescribers is recognised by the MCC. **It must be noted that accountability, when this practice is applied, remains with the prescriber.** (See Council Newsletter Volume 23 December 1998.)

"Whether the extra-label use is justified is therefore not dependent on the manufacturer's instructions but rather whether the veterinarian was acting in the best interest of the patient. Therefore in the case of the veterinarian not complying with the instructions of the manufacturer it would be incumbent on the veterinarian to justify, preferably by scientific data of by conclusive empirical evidence, that he/she acted in the best interest of the patient. In other words, the onus is on the prescribing veterinarian to be accountable when this practice is applied. Should Council find that acting contrary to the manufacturer's instructions was not justified and that the prescribing veterinarian had no grounds for extra-label use, then this could be judged as unprofessional conduct."

When a veterinarian considers the extra-label use of a pharmaceutical product the following guidelines should be considered:

7.1.1 Make sure that no alternative exists. There may be other registered drugs available.

7.1.2 Ascertain whether the animal is insured and what the implications may be should the animal die.

7.1.3 Obtain consent, preferably written, from the rightful owner.

"Veterinarian's duty to inform the owner of an animal.

The amount of information that a veterinarian must give the owner of an animal in order to obtain a proper consent must be sufficient for the person to understand fully the nature and effect of the treatment or procedure consented to. This means that the veterinarian must inform the owner of an animal about all the "material risks" involved in the proposed treatment or procedure. Using an analogy from the medical profession, material risks are those that: (a) a reasonable person in the position of the owner of the animal would have regarded as significant, and (b) a reasonable veterinarian would have been aware that the owner of an animal, if warned of the risk, would attach significance to it.

Unlike in the medical profession where there may be scope for non-disclosure based on the "therapeutic privilege", in the case of veterinarians there can be no excuse for failing to make full disclosure to the owner of an animal concerning the risks involved in any particular treatment or procedure.

7.1.4 Make sure that a good veterinarian/client/patient relationship exists. The veterinarian has assumed responsibility for making veterinary judgements regarding the health of the animal(s) and the need for veterinary treatment and the client (owner or agent) accepts this and has agreed to follow the instructions of the veterinarian.

7.1.5 The veterinarian must demonstrate his/her responsibility to the community when prescribing or using pharmaceuticals for food animals to ensure appropriate withdrawal periods and the safety of the food products to man or other animals.

7.1.6 A veterinarian prescribing the extra-label use of medicines must comply with all the relevant legal requirements for the supply, labelling and disposal of medicines.

7.1.7 When reviewing extra-label uses, the clinical pharmacology and safety of potential drugs should be considered in the context of their proposed use. The significance and consequences of varying the target species, dose rates, routes of administration and duration of treatment for formulations need to be evaluated.

7.1.8 Whether extra-label use is justified is not dependent on the manufacturer's instructions but rather whether the veterinarian was acting in the best interest of the patient. Therefore in the case of the veterinarian not complying with the instructions of the manufacturer it would be incumbent on the veterinarian to justify, preferably by scientific data or by conclusive empirical evidence that he/she acted in the best interest of the patient."

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Skinny Old Cats:

Why some senior cats lose weight. What's going on?

By David Williams, MA, VetMB, PhD, DACVIM, DECVIM-CA, Nov 12, 2014

Decline in body weight is common in cats that are older than 11 years.[1] Sometimes this loss is readily attributable to apparent disease, but in many cases, there are no obvious signs of illness and routine diagnostic approaches fail to reveal evidence of an underlying problem.[2,3] Energy requirements of older cats apparently do not decline as markedly as they do in dogs and people, perhaps because physical activity does not decrease as much with age in cats. Indeed, the maintenance energy requirement of older cats may increase rather than decrease.[3,4] Although cats might be expected to regulate their energy intake to compensate for these changes to maintain body weight, that clearly is not always the case.[4,5]

It has been recognized for many years that both protein and fat digestibility decrease in many apparently normal cats after 10 years of age. While the cause of the decreases remains unclear, the changes are quite marked in some individuals and in particular can be dramatic with regard to fat digestibility.[4,5] Often these changes are not readily apparent from casual observation of faeces and may only be verified if faecal fat content is quantified by appropriate analytic testing. Methods for such testing are rarely available for evaluation of veterinary patients, even at referral centers. Whatever the explanation for weight loss and decline in nutrient digestibility in older cats, progressive decline in body weight has been reported in the two years before death from a variety of seemingly unrelated diseases. As cats live increasingly longer lives and receive attentive health care, this weight loss is more frequently recognized. In this article, I will review what is known about common age-related changes and what may be done to halt or reverse the decline in body weight that is apparently a predictable prelude to death.[3,4,6]

Attributable weight loss

Well-recognized causes of weight loss in old cats include chronic renal disease, diabetes mellitus, hyperthyroidism, inflammatory bowel disease (IBD), exocrine pancreatic insufficiency, and dental problems. Most are readily suspected and confirmed based on physical examination and routine laboratory testing. At times, selected additional testing of parameters such as serum thyroxine, serum trypsin-like immunoreactivity, cobalamin and folate, dental radiography (Figures 1A and 1B), or gastrointestinal (GI) endoscopy and biopsy may be necessary. Despite thorough investigation, however, the underlying cause of even severe weight loss can be remarkably difficult to establish conclusively. Subtle weight loss may not even be noted unless careful records of body weight and body condition scores are kept over repeated veterinary examinations. Similarly, moderate increases or decreas-

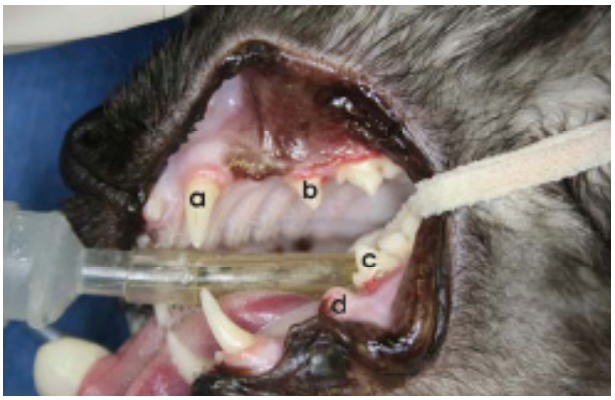

Figure 1A. Dental disease can contribute to weight loss in senior cats. Tooth resorption of the left maxillary canine (a) and 3rd premolar in a cat (b). Small focal areas of inflammation are apparent on the left maxillary 3rd premolar (b) and the left mandibular 4th premolar (c). The left mandibular 3rd premolar is missing, leaving focal residual inflammation (d).

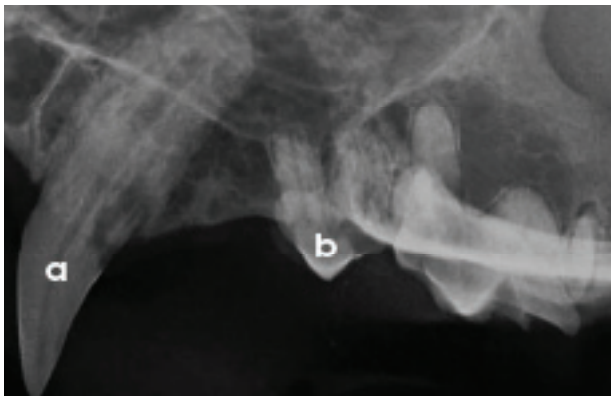

Figure 1B. In the same cat, an intra-oral bisecting angle dental radiograph of the left maxilla shows an unsuspected extensive tooth resorption at the cementoenamel junction of the distal left maxillary canine tooth (a). Tooth resorption affecting the root and crown of the 3rd premolar is also apparent (b).
(Courtesy of the Veterinary Dental Service Library, University of Illinois)

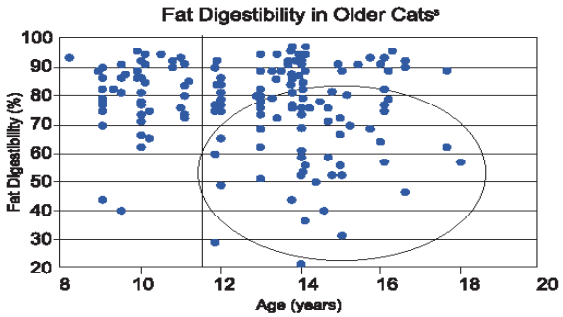

Figure 2. Fat digestibility (by percentage) in cats ranging in age from 8 to 18 years (n = 208)

es in food or water intake will probably go unnoticed by most owners. Even when the most attentive owners provide the best veterinary care for their cats, a substantial proportion of senior cats will experience weight loss, despite being in otherwise good health and exhibiting no detectable change in food intake. Evidence indicates that, in these older cats with no apparent classic diseases to explain the weight loss, there is an age-related decline in food digestibility.[3]

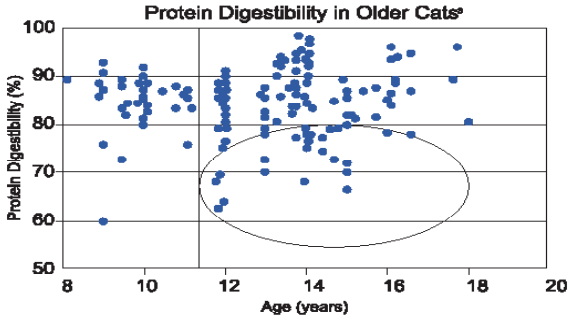

Figure 3. Protein digestibility (by percentage) in cats ages ranging in age from 8 to 18 years (n = 208).

There is a significant ($p < 0.0001$) negative correlation ($r = -0.76$) between age and fat digestibility (Figure 2). The incidence of low fat digestibility increases with age, affecting 10% to 15% of mature cats (8 to 12 years old) and 30% of geriatric cats (> 12 years old). In some geriatric cats, fat digestibility was found to be as low as 30%, and the only clinical signs were large stools (not frank diarrhoea) and low body weight. There is also a significant ($p < 0.0001$) negative ($r = -0.66$) correlation between age and protein digestibility (Figure 3).

Low protein digestibility also seems to affect mature and geriatric cats. Although the incidence of low protein digestibility is lower than that of fat digestibility, about 20% of cats older than 14 years show protein digestibility lower than 77%. The incidence of low fat and protein digestibility tends to occur in the same cats. A marked decline apparently becomes particularly prevalent after around age 10 (Figures 4 and 5).

It is perhaps not surprising that these changes were

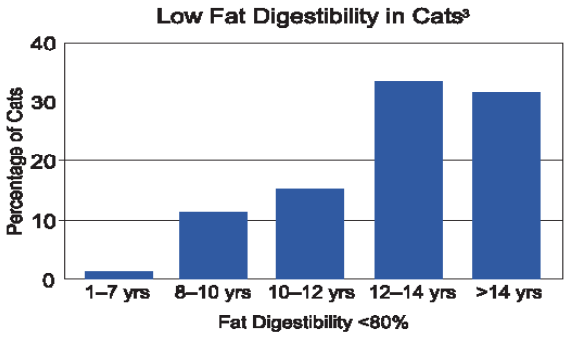

Figure 4. Percentage of cats with low fat digestibility by age (pooled data from four colonies).

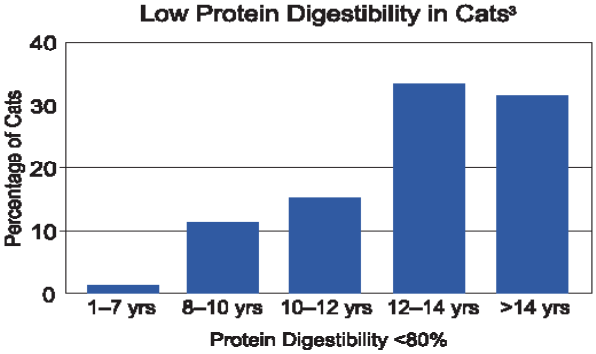

Figure 5. Percentage of cats with low protein digestibility by age (pooled data from four colonies).

correlated with several other measures of health or well-being, including serum vitamin E (tocopherol), vitamin B12 (cobalamin), skin thickness, body fat, and body condition score. Overall, while obesity tends to be the predominant body-mass concern in cats between 7 and 12 years of age, in those older than 12 years, obesity is rare and being underweight is a far greater life-threatening risk factor (Table 1 and Figure 6).[3]

Table 1. Incidence of Feline Obesity and Underweight by Age*

Age Group	Body Weight (kg)	ObesityIncidence	Percent Underweight
Adult (1–7 years)	3.7 ± 0.8	< 1%	< 1%
Mature (7–12 years)	4.4 ± 1.7	28%	< 1%
Geriatric (> 12 years)	2.9 ± 1.0	< 1%	23%

**Patil AR, Cupp CJ. Addressing age-related changes in feline digestion. Proc Nestle-Purina Compan Anim Nutr Summit, Focus Gastroenterol 2010:55-61.*

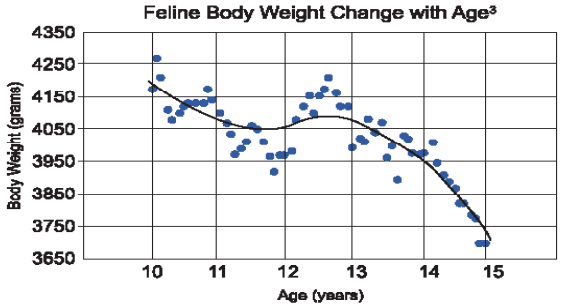

Figure 6. After 12 years of age, there is marked decline in body weight among cats, which supplants obesity as a common life-threatening condition.

Nutrient digestibility

The cause or causes of this decline in nutrient digestibility remain unknown but presumably reflect enteropathy of some type. In some cases, this intestinal dysfunction may overlap with what is loosely classified as (idiopathic) IBD. Some cats compensate for the loss in digestive function by eating more and, therefore, exhibit no weight loss. It is important to recognize that many cats show only subtle changes in stool characteristics (slightly larger volumes of stool with a more clay-like consistency) but not frank diarrhoea, even when steatorrhoea is marked.

Regardless of the precise cause or causes, weight loss in otherwise healthy older cats and changes in faecal characteristics should be investigated, as should malabsorption. Thorough physical examination, routine complete blood count, serum chemistry profile, urinalysis, and faecal examination are all indicated, as are radiographic and ultrasonographic evaluations as appropriate. If nothing specific is found to explain the weight loss, the next step is to measure serum thyroxine, feline pancreatic lipase, feline trypsin-like immunoreactivity, and cobalamin/folate levels. I recommend that these be determined concurrently because studies have indicated that about 50% of hyperthyroid cats have evidence of concurrent intestinal and/or pancreatic abnormalities, including sometimes severe hypocobalaminemia, when the endocrinopathy is initially diagnosed.[7,8] Furthermore, all abnormalities detected should be treated concurrently to optimize clinical response to treatment.

Many hyperthyroid cats are appropriately diagnosed and treated, but GI signs, especially weight loss, persist despite return to the euthyroid state. Subsequent evaluation of GI function as outlined above then reveals evidence of enteric disease and cobalamin deficiency. Only when these are also appropriately treated do the cats return to optimal health.

The diagnostic process

Determination of faecal fat (by percentage) would be desirable and may be the only way to confirm an intestinal problem in some patients. Faecal fat greater than 20% would indicate fat malabsorption. Unfortunately, such a test is not commercially available for pet cats. It has been reported that 100% of cats older than 7 years of age with serum tocopherol (vitamin E) less than 5 mg/L also have low fat digestibility, and that more than 90% of cats with serum cobalamin less than 100 g/L have low fat digestibility.[3] So finding such low serum concentrations of either cobalamin or tocopherol can be the basis for inferring that a cat has low fat (and probably protein) digestibility.[3]

A new test that often reveals abnormalities in intestinal function in affected cats is an assay of faecal α1-proteinase inhibitor by species-specific immunoassay.[9] This test is presently available only from the Gastrointestinal Laboratory at Texas A&M University.

Abnormal results indicate the presence of an enteropathy-associated increase in enteric loss of protein; this test can detect protein-losing enteropathy that is not sufficiently severe to lower serum albumin (the liver can compensate for enteric protein loss). Chronic enteric protein loss can contribute to gradual depletion of lean body mass. In a recent study more than 70% of affected cats had abnormal [α1-PI] test results (Figure 7), but interestingly, serum albumin was slightly subnormal in only two of the 11 cats with protein-losing enteropathy.[10]

Finally, it should be noted that it is very common to see increased serum concentrations of trypsinlike immunoreactivity (fTLI) and/or pancreatic lipase (fPL) in these cats with idiopathic chronic enteropathy (Figure 8). The significance of these pancreatic abnormalities is not clear. Many affected cats show few if any signs that may be considered suggestive of pancreatitis (e.g.anorexia or vomiting), but the increases can be striking in some individuals. In the future, assay of enteric inflammatory markers such as faecal calprotectin may prove useful in confirming the presence of enteric disease, but the relationship of inflamma-

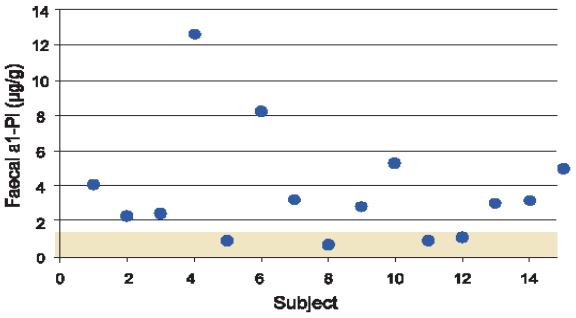

Figure 7. Faecal alpha 1-proteinase inhibitor concentration was increased in 11 of 15 geriatric cats with idiopathic chronic enteropathy, indicating active protein-losing enteropathy in addition to the previously recognized nutrient malabsorption. The tan line indicates normal α1-PI concentration.

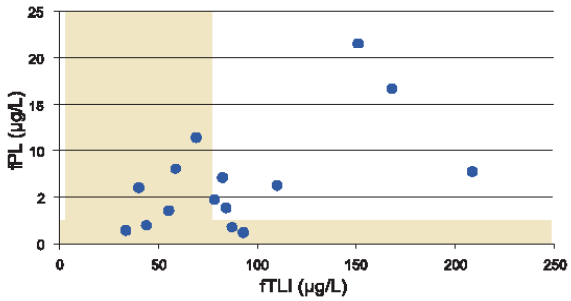

Figure 8. Pancreatic pathology is also present in a high proportion of geriatric cats with idiopathic chronic enteropathy, as reflected in increased serum concentrations of trypsin-like immunoreactivity (fTLI) and pancreatic lipase (fPL). The shaded areas represent normal reference ranges.

tion to this enteropathy is currently uncertain. Even histologic examination of intestinal biopsy specimens may not provide evidence of a conclusive diagnosis; lesions may be patchy and interpretation of biopsy findings is inherently subjective.

It is also likely that in cats, as in dogs, functional problems in the intestine may not be associated with either inflammation or villous atrophy but rather with intraluminal microbial changes and biochemical derangements in the enterocytes lining the small intestine that are not revealed by classic histologic evaluation.

Treatment

In some cats, despite a thorough investigation, it is not possible to confirm enteropathy. A presumptive diagnosis of idiopathic enteropathy is the best that can be achieved. The approach to management in these instances is essentially the same as for patients with histologically confirmed IBD — that is, a dietary change (low-carbohydrate alternative fiber source, novel antigen, or hydrolyzed diet), prebiotic or probiotic supplementation, correction of low serum cobalamin/folate concentrations, supplementation with vitamin E and perhaps other antioxidants, antibiotic treatment with metronidazole or tylosin, and perhaps glucocorticoid therapy or immunomodulation with chlorambucil or cyclosporine.[11] However, in the absence of specific laboratory abnormalities or any overt clinical signs to monitor, other than perhaps very slowly progressive weight loss, it is probably premature to recommend particularly aggressive treatment for these patients. A cautious, conservative approach is warranted.

As many of the observations about digestive disturbances in elderly cats are relatively new, appropriate clinical studies evaluating treatment interventions have not been performed. Dietary changes and supplements would certainly be the safest and most easily administered interventions. When specific abnormalities such as hypocobalaminaemia are identified, they should be rectified. The effect of dietary changes has to be evaluated on an individual trial-and-error basis, which can be difficult if gradual weight loss is the only clinical sign to evaluate.

Observing improvements in the newer GI disease markers, such as faecal α1-proteinase inhibitor and serum fPL, may provide objective evidence of a positive response, but the value of this approach remains to be evaluated. Careful observation of stool characteristics may provide some evidence of improved digestibility, especially if grossly apparent abnormalities were present at the outset.

If there is no apparent response to dietary change after two to four weeks, an alternative diet should be tried. I prefer to select diet changes based on reduced carbohydrate content (generally associated with increased protein content) or different amounts or types of fermentable fiber. Adjusting the fat content of the diet does not appear to be particularly useful in treating feline enteropathies. Unfortunately, definitive studies in geriatric cats with malabsorption have not been done. Treatment needs to be individualized and evaluated on a trial-and-error basis.

With regard to older cats in general, there is some evidence that diet can play a role in maintaining body weight and fat mass — and prolonging life. A control diet (nutritionally complete and balanced adult cat food) supplemented with antioxidants (vitamin E and ss-carotene), a blend of n-3 and n-6 fatty acids, and a prebiotic (dried chicory root) was associated with reduced decline in body weight and increased longevity (by more than 1 year) compared with feeding either the control diet *alone* or the control diet supplemented with antioxidants alone.[6] These striking observations illustrate the potential benefit to be gained from dietary and other interventions to address the gastrointestinal changes that appear to be so common in aging cats.

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Current Understanding of Feline Epilepsy, Diagnostic Testing, And Treatment

By Jennifer L. Garcia, DVM, DACVIM

Pakozdy A, Halasz P, Klang A. Epilepsy in cats: theory and practice. J Vet Intern Med 2014;28:255-263.

An overview of our current understanding of feline epilepsy, including diagnostic testing and treatment.

Why they did it

There is a lack of scientific literature about epilepsy in cats, and much of the terminology as well as diagnostic and treatment recommendations have been extrapolated from experience with dogs and people. This article reviews the available literature and provides an overview of our current understanding of feline epilepsy, including diagnostic testing and treatment.

Overview

Epilepsy has become a catchall term used to describe any sudden neuronal event; however, in veterinary medicine, we lack the diagnostic testing (electroencephalogram) required to definitively diagnose epilepsy.

Etiologic classification of epilepsy in cats:

Idiopathic (primary)	No underlying brain lesion is present
Symptomatic (secondary)	Implies the presence of an underlying brain lesion or disorder
Probable symptomatic (cryptogenic)	Occurs as a result of brain lesions that cannot be identified (e.g. post-anaesthetic seizures)
Reactive	Occurs as a result of a toxic or metabolic cause

The characteristics of epilepsy in cats

The authors note that epileptic seizures in cats are often focal complex seizures with or without generalization, and clinical signs can include drooling, facial twitching, hypersalivation, mydriasis, tremors, running, urination, and defecation. In the past, cats that experienced focal seizures were thought to have secondary epilepsy due to an underlying brain lesion, but studies have shown that both focal and generalised seizures may occur in cats with primary, or idiopathic, epilepsy as well. Unlike idiopathic epilepsy in dogs, there is no evidence at this time of a genetic origin for idiopathic epilepsy in cats. The age of onset of idiopathic epilepsy appears to be more likely in younger versus older cats. However, because considerable overlap among age groups has been reported, age of onset is of little diagnostic utility. Seizures due to idiopathic epilepsy maybe more likely to occur during periods of rest because of "an increase in cortical neuronal synchronization during sleep," which decreases the seizure threshold. A definitive diagnosis of idiopathic epilepsy requires exclusion of all other possible causes of epilepsy. However, identification of a cause of secondary epilepsy can be frustrating, especially in cases of incomplete work-up, equivocal test results, or patients with more than one epileptogenic condition.

Making the diagnosis

In addition to the signalment, detailed history, and evaluation of the timing and frequency of the seizures, thorough physical and neurologic examinations will be required in these cases. The authors also point out that normal findings on a neurologic examination do not rule out the possibility of a brain lesion. Blood pressure testing, a complete blood count, a serum chemistry profile, a urinalysis, and thyroid testing will be required to look for metabolic or endocrine derangements, which may provide evidence of an underlying disease process. Interestingly, the authors note that feline leukaemia virus, feline immunodeficiency virus, feline infectious peritonitis, and toxoplasmosis testing are of limited utility as studies have demonstrated that these infections are rarely the cause of primary brain disorders. When a primary brain lesion is suspected or other noninvasive tests have proven inconclusive, magnetic resonance imaging and cerebrospinal fluid analysis should be considered.

Hippocampal necrosis as a cause of feline epilepsy

There is data to suggest that hippocampal lesions may cause seizures in cats. Cats with this type of lesion may develop acute cluster seizures with evidence of salivation and aggression. Necrosis in this area may be a result of an inflammatory, a neoplastic, a vascular, or a toxic event. Hippocampal necrosis is currently being studied as a form of feline temporal lobe epilepsy.

Epilepsy treatment in cats

There is currently no consensus as to when to begin antiepileptic drug therapy. Recent literature supports early intervention with phenobarbital as it may lead to better outcomes, but treatment should be considered on an individual basis. Risks of therapy, seizure severity and frequency, as well as owner compliance and monitoring will need to be considered. Phenobarbital is the antiepileptic drug used most often, but there are no high-evidence studies evaluating routine use of antiepileptic drugs in cats at this time.

Overall, the authors note that most studies suggest a good outcome for cats with idiopathic epilepsy treated with phenobarbital, although there are cases in which cats may be refractory to therapy.

Using Laboratory Analytes For Diagnosing Inflammation in Horses

By Emma Hooijberg BVSc DECVCP
*(Currently Employed at the Equine Section of the OVAH.)

Hooijberg, E.H., van den Hoven, R., Tichy, A. and Schwendenwein, I. (2014), Diagnostic and Predictive Capability of Routine Laboratory Tests for the Diagnosis and Staging of Equine Inflammatory Disease. Journal of Veterinary Internal Medicine, 28: 1587–1593

Why they did it

Laboratory tests available for diagnosing inflammatory disease in horses include the leucocyte count, fibrinogen, iron, serum amyloid A (SAA) and the myeloperoxidase index (MPXI). This study was performed in order to determine the diagnostic efficiency of these analyses for detecting various types of equine inflammatory disease.

SAA is a major, and fibrinogen a minor, positive acute phase protein in the horse. Inflammation causes a decrease in iron levels via increased IL-6 and hepcidin production. MPXI is a measure of the neutrophil myeloperoxidase content and is postulated to reflect the activation state of circulating neutrophils.

What they did

This was a retrospective study using clinical records from patients at the equine clinic of a university teaching hospital. Horses over one year of age who had undergone laboratory testing for all the above-mentioned analyses on a single blood sample were included. The animals were classified as having either systemic, local or no inflammatory disease based on previously defined clinical criteria and information from the clinic record.

A leukopaenia or leukocytosis was one of the criteria used to define horses as having systemic inflammation and thus the leukocyte count was not examined further in this study; it has however been shown in other studies to be insensitive for inflammation in horses, unlike in dogs and cats. The analytes SAA, fibrinogen, iron and MPXI were compared across groups.

Sensitivity and specificity were calculated for each analyte. Receiver-operator characteristic curves were used to determine the diagnostic accuracy of the analytes. Lastly, predictive modelling in order to try and develop diagnostic algorithms was performed. The effect of previous anti-inflammatory treatment was also considered.

What they found

The population consisted of 201 horses, 26 of which were classified with systemic, 114 with local and 61 with no inflammation. The MPXI was not different between groups and had no diagnostic value for diagnosing inflammatory disease. SAA had the highest accuracy (area under the curve 0.83) for diagnosing inflammation, with a high sensitivity of 82% (i.e. few false negatives), however there were 18 horses without clinical signs of inflammation with an increased SAA, which means it had only a moderate specificity in this study and that horses may have an acute phase response without showing clinical signs or requiring treatment. Iron and fibrinogen proved to have a high specificity (iron 90%, fibrinogen 97%) with low iron and high fibrinogen concentrations useful for diagnosing inflammation.

Values within the reference intervals for iron and fibrinogen did not however rule out inflammation and they had a very low sensitivity (iron 40%, fibrinogen 37%). It was not possible to predict the type of inflammation based on concentrations of iron, fibrinogen and SAA.

Using the analytes in combination had a good sensitivity which means that a high SAA and fibrinogen and low iron were highly predictive of an inflammatory state. There was no effect on transport or previous treatment on the results.

Take-home message

SAA, iron and fibrinogen are useful markers for diagnosing inflammatory disease in horses and should be used in combination in order to optimise diagnostic performance. Absolute concentrations of these analytes cannot be used to diagnose the type of inflammation.

Using the Vertebral Heart Scale to Categorize Dyspnoea in Cats

By Jennifer L. Garcia, DVM, DACVIM

Sleeper MM, Roland R, Drobatz KJ. Use of the vertebral heart scale for differentiation of cardiac and noncardiac causes of respiratory distress in cats: 67 cases (2002-2003). J Am Vet Med Assoc 2013; 242(3):366-371.

Why they did it

Dyspnoeic cats may have underlying congestive heart failure, but is there a way to determine the presence of this life-threatening cardiac condition without the need for performing echocardiography? These researchers sought to determine if calculating a cat's vertebral heart scale (VHS) might be a good indicator.

What they did

Researchers evaluated the medical records of 67 cats that were presented over a one-year period for evaluation of acute onset of respiratory distress. Inclusion in the study required thoracic radiography and echocardiography, and the VHS was calculated by two investigators for each cat. The VHS for normal cats is roughly < 7.3 on the lateral view of the thorax.

What they found

Researchers found that a VHS cutoff of > 8 vertebrae had a high sensitivity for screening for heart disease and that a cutoff of > 9.3 vertebrae had the highest specificity for a diagnosis of heart disease. For patients with a VHS between 8 and 9.3, researchers were unable to reliably predict whether heart disease or respiratory disease was the cause of the dyspnoea. Echocardiography would be required for these cases.

Take-home message

The presence or absence of a heart murmur does not reliably predict cardiac disease in cats. In addition, the variable appearance of cardiogenic pulmonary edema on thoracic radiographs may also make the diagnosis of cardiac disease difficult. For cats seen on an emergency basis in which echocardiographic evaluation is not possible, VHS may aid in the differentiation of dyspnoea due to cardiac disease vs. noncardiac causes.

Human Medication Intoxications

By: Andrew Linklater, DVM, DACVECC, Lakeshore Veterinary Specialists Glendale, Wisconsin

Because veterinary patients commonly present for human medication ingestion, veterinarians should have treatment plans for patients that have ingested these medications, including over-the-counter and prescription medications (eg, NSAIDs), new classes of anti-depressant medications, amphetamines (used in the treatment of attention deficit-hyperactivity disorder [ADHD]), marijuana, and vitamin D.

General Approach

As with any emergent patient, it is imperative to stabilize airway, breathing, and circulation first. Carefully questioning the owner often reveals the ingested toxin. Asymptomatic patients that present <4 hours after ingestion may benefit from emesis induction with apomorphine (dogs) or xylazine (cats). If the patient is stable, administration of activated charcoal may be warranted *(for treatment agents see Table 1)*

In addition, contraindications for emesis should be considered before induction. When needed, gastric lavage should be performed in an anaesthetised, intubated patient to provide comfort and limit risk for aspiration. General supportive care measures depend on clinical signs and may include anti-emetic therapy, IV fluid therapy, blood pressure monitoring, oxygen therapy, and/or symptomatic supportive care.

NSAIDs

NSAIDs, which interrupt prostaglandin production by inhibiting cyclo-oxygenase, can result in decreased blood flow to renal and GI systems. Liver and platelet function may also be affected with large-quantity or chronic ingestion. Toxicosis severity and treatment duration may be impacted by dehydration; time until decontamination and/or treatment; and pre-existing hepatic, renal, or GI disease. Vomiting and diarrhoea, secondary to GI irritation, are the most common signs, but they may not be evident initially.

Table 1: Agents Used for Toxic Exposures

Agent	*Dose/Schedule*	*Notes/Adverse Events*
20% IV lipid emulsion	1.5 mL/kg bolus, then 0.25 mg/kg/min - for 30–60 min	Discontinue if lipemia develops or if treatment is ineffective
Acepromazine	0.05–0.2 mg/kg IV or IM	Titrate dose; hypotension
Activated charcoal	1–2 g/kg PO q8h	Enterohepatic recirculation or extended release warrants additional doses
Apomorphine	0.03 mg/kg IV (dogs)	Sedation
Chlorpromazine	0.5 mg/kg IV	Sedation
Cyproheptadine	1.1 mg/kg (dogs) q4–6h - 2–4 mg total dose (cats) q4–6h	Oral or rectal dosing
Diazepam	0.25–0.5 mg/kg IV q1h; repeat up to 3 doses	Continuous infusion option
Maropitant	1 mg/kg SC	
Methocarbamol	50–100 mg/kg slow IV	Sedation
Midazolam	0.2–0.4 mg/kg IV or IM	Sedation
Misoprostol	2–5 µg/kg PO q8h	Wear gloves to handle (GI/uterine contractions, abortifacient possibility)
Ondansetron	0.6–1 mg/kg SC	
Pamidronate	1.3–2 mg/kg IV diluted over 2 hours	May repeat in 4–7 days
Pantoprazole	0.5–1 mg/kg slow IV q24h	Give diluted
Phenobarbital	4 mg/kg IV, 4 doses as needed	Loading dose; titrate based on level of sedation
Propofol	4–8 mg/kg induction IV	0.1–0.6 mg/kg/min infusion for continuous sedation; requires intubation
Propranolol	0.02–0.06 mg/kg slow IV - 0.1–0.2 mg/kg PO q8h	Titrate dose to effect
Xylazine	0.4–1.1 mg/kg IM (cats)	Yohimbine reversal

Accidental NSAID ingestion untreated for >8 hours can cause risk for acute renal failure and GI ulceration. When large doses are ingested, decontamination and ≥48 hours of twice-maintenance IV fluids are recommended to prevent renal injury. GI protectants (eg, misoprostol, H2-blockers, proton pump inhibitors, sucralfate) may be beneficial.

Misoprostol can help maintain GI blood flow. Proton pump inhibitors and H2-blockers help reduce gastric acid while sucralfate coats the ulcerated region; both should be administered if a GI ulcer is suspected. Simple renal injury may respond to fluids and medications; oliguria and anuria require intensive monitoring and/or dialysis. Liver injury has been reported with some NSAID ingestion and may warrant further therapy. Baseline serum biochemistry and daily monitoring for at least 48 to 72 hours are recommended.

Anti-depressant Medications

Most anti-depressant medications work via reuptake inhibition or altered transport of serotonin, norepinephrine, or dopamine *(See Table 2)*. Many are rapidly absorbed and come in extended-release or long-acting formulas. Clinical signs of toxicosis can vary, depending on the medication; selective serotonin reuptake inhibitors (SSRIs) can result in serotonin syndrome, affecting the cardiovascular (eg, hyper- or hypotension, tachycardia), GI (eg, vomiting, diarrhoea), and neurologic (eg, sedation, agitation, ataxia, tremors, seizures) systems.[1]

Standard decontamination is recommended but should be avoided in symptomatic patients. Supportive and symptomatic care are the mainstay of therapy.

Table 2 Common Anti-depressant Medications

Generic Name	*Common Brand Names*	*Mechanism*
Amitriptyline	Elavil	SNRI (TCA)
Atomoxetine	Strattera	NRI
Bupropion	Wellbutrin	NDRI
Clomipramine	Anafranil	SNRI (TCA)
Duloxetine	Cymbalta	SNRI
Fluoxetine	Prozac	SSRI
Mirtazapine	Remeron	NaSSA
Paroxetine	Paxil	SSRI
Selegiline	Anipryl, l-depreny	MAOI
Sertraline	Zoloft	SSRI
Venlafaxine	Effexor	SNRI
Viloxazine	Vivalan	NRI

Key: MAOI = monoamine oxidase inhibitor, NaSSA = noradrenergic and specific serotonergic antidepressant, NDRI = norepinephrine-dopamine reuptake inhibitor, NRI = norepinephrine reuptake inhibitor, SNRI = serotonin-norepinephrine reuptake inhibitor, SSRI = selective serotonin reuptake inhibitor, TCA = tricyclic anti-depressant

Fluid therapy does not enhance elimination but may help correct dehydration and acidosis; sodium bicarbonate may be given to patients with severe acidosis. Passive and/or active cooling may be necessary for hyperthermic patients. Propranolol or esmolol may be used for supraventricular tachyarrhythmias, norepinephrine or epinephrine for hypotension, and benzodiazepines for sedation or treatment of tremors or seizures.[1-3] Methocarbamol has been recommended as an alternative therapy for tremors. Phenothiazine use remains controversial.

Seizures not responsive to benzodiazepines may be treated with barbiturates. Although it is not specifically studied in toxicologic overdoses, status epilepticus not responsive to benzodiazepines and barbiturates may respond to propofol or inhalant (isoflurane) anaesthesia.[3-8] Specific therapy includes serotonin antagonism with cyproheptadine.

ADHD Medications

ADHD amphetamines *(See Table 3)* are stimulants that inhibit norepinephrine and dopamine reuptake in the brain. Clinical signs are neurologic (eg, agitation, shaking/trembling, circling, seizures, disorientation, coma, death), cardiovascular (eg, tachycardia, hypertension, potential reflex bradycardia), and GI (eg, vomiting, abdominal pain).

Table 3 Common ADHD Medications

Generic Name	*Common Brand Names*
Amphetamine–dextroamphetamine	Adderall
Dexmethylphenidate	Focalin
Dextroamphetamine	Dexedrine, Dextrostat
Lisdexamfetamine	Vyvanse
Methylphenidate	Ritalin, Concerta, Daytrana, Metadate

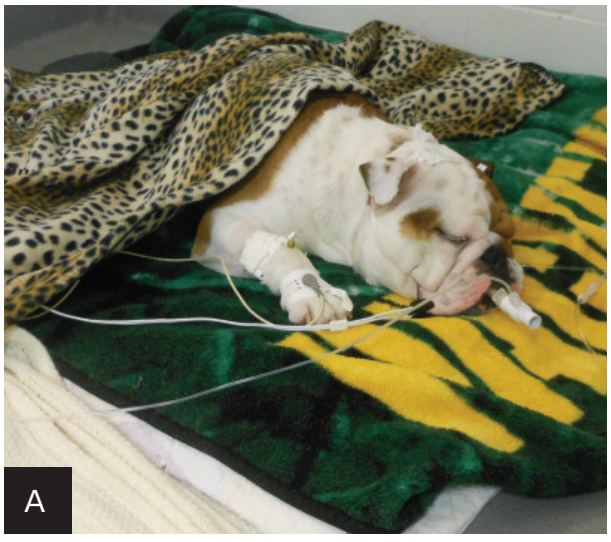

Figure 1A. One-year-old intact male bulldog presented after ingestion of the owner's Adderall (amphetamine–dextroamphetamine). The dog presented hyperthermic and tachycardic and developed seizures. It required sedation, antiepileptic medications, and beta-blockers to control signs.

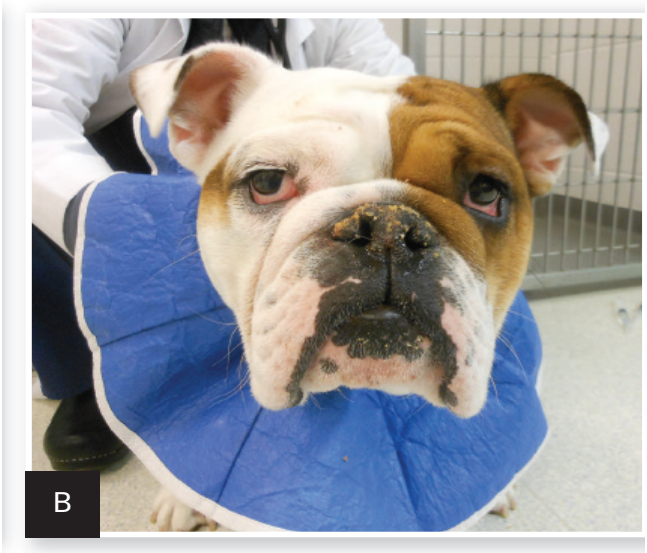

Figure 1B. Endotracheal intubation and intermittent oxygen supplementation were required because of heavy sedation. Thirty-six hours later, the dog was normothermic, had a normal heart rate, and no longer had seizures. Several days after presentation, it was reportedly doing well.

Standard decontamination should be used judiciously because of possible inability to protect the airway. Additional treatment *(See Figure 1)* is symptomatic and supportive; phenothiazines (eg, acepromazine) are the first-line agents for excessive stimulation and may also be used to treat hypertension and hyperthermia. Keeping the patient in a quiet, dark room to minimize stimulation may help. Use of benzodiazepines in patients with stimulatory signs is controversial; however, they may be used for seizures. Seizures not responsive to benzodiazepines may respond to barbiturates or propofol.[3-9] Excessive muscle fasciculations may also be treated with methocarbamol. IV fluids and cooling may be necessary for patients with elevated body temperatures. Because some ADHD medications are lipophilic, IV lipid solutions may be considered; however, there are little data available on their use with these medications.

Marijuana

Marijuana (*Cannabis sativa*), legally prescribed for humans in select states, contains Δ9-tetrahydrocannabinol (THC), which alters neurotransmitter activity. Patients typically present with altered mental status, ataxia, and dilated pupils; potential agitation, seizures, and/or coma may result in death. Additional signs can include vomiting, diarrhoea, arrhythmias, tachypnea, and incontinence. In dogs, over-the-counter urinary tests are unreliable for diagnosis.

Decontamination should be avoided when altered mental status is present, and emesis is often unrewarding. Supportive care is the mainstay of treatment. Because renal elimination of THC is minimal, the benefit of diuresis is questionable; fluids may be used to treat dehydration. Benzodiazepines can be used safely for agitation, seizures, and tremors; monitoring temperature, respirations, and heart rate is essential. Most patients recover uneventfully within 24 to 96 hours; however, ingestion of large quantities may require aggressive supportive care. IV lipid infusions have been advocated anecdotally and used successfully by the author.

Vitamin D

Vitamin D_3 is available as a sole supplement, in multivitamins (which may also contain toxic levels of iron, xylitol, and vitamin A), or in rodenticides. Initial clinical signs (8–12 hours after ingestion) are vague and include lethargy, vomiting, diarrhoea, and inappetence resulting in hypercalcemia and renal failure (occurring 36–48 hours after ingestion), which cause polyuria/polydipsia, weakness, hematemesis, and arrhythmias. Decontamination should be initiated immediately, as vitamin D_3 has a prolonged action and treatment can be challenging and costly if hypercalcemia develops. When decontamination is not possible or is incomplete, IV lipid emulsion may also help prevent hypercalcemia, as vitamin D is fat soluble. Ionized calcium levels should be monitored q24h for 5 to 7 days. When ionized hypercalcemia is present, therapies (eg, saline diuresis, furosemide, steroids, salmon calcitonin) may need to be combined. Pamidronate inhibits osteoclast function and has prolonged activity, making it an attractive alternative if other therapy is unavailable or ineffective.

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All bunged up: Unclogging the Constipated Cat

By Margie Scherk, DVM, DABVP (Feline practice)

Straining in the litter box—possibly even crying out or leaving unwelcome hard pellets around the home—constipated felines are uncomfortable. And constipation can interfere with a cat's appetite and even result in vomiting. Traditional approaches to this hard problem include administering enemas, laxatives to soften the stool or increase contractions, dietary fiber, and promotility agents. Could we be missing something really basic? And when should we be concerned about long-term effects of constipation?

CAUSES OF CONSTIPATION

Constipation is a clinical sign that is not pathognomonic for any particular cause. Most commonly, constipation is a result and sign of dehydration. The body is 65% to 75% water, depending on a cat's age and percent body fat. Homeostasis attempts to maintain a consistent cellular and extracellular environment. When cells become dehydrated, the body takes steps to correct the fluid deficit. Drinking more and concentrating urine are helpful, but once those capabilities have been maximised, water is reabsorbed in the colon, resulting in drier stool that is harder to pass. Bearing this in mind, medical therapy might not be the best initial therapeutic approach.

Other causes of constipation include problems that result in obstruction (either mechanical or functional), painful defecation, stress within the home environment (social or a dirty toilet), and possibly metabolic disease (Table 1).

Table 1: Processes resulting in constipation

Mechanism	Examples
Increased water loss	• Diuretic drugs • Polyuria from chronic kidney disease, diabetes mellitus, hyperthyroidism • Vomiting
Inadequate water intake	• Inadequate amount or quality of water available or lack of access to water (social stress or limited mobility) • Diet excessively dry or high in insoluble fiber • Painful drinking from orodental disease or difficulties swallowing
Painful defecation	• Degenerative joint disease; diseases of anal glands, prostate, rectum
Reluctance to defecate	• Social competition or fear of being ambushed • Unpleasant litter box (e.g. dirty, negative association with painful urination or defecation, inadequate size, covered box, dislike of litter type, suboptimal location) • Hospitalization
Obstruction—mechanical	• Intraluminal foreign body, neoplasia, stricture, polyp • Mural thickening (neoplasia, inflammation), intussusception, diverticulum, or hernia • Extra-intestinal compression by neoplastic or other mass, pelvic fracture, prostatic disease
Obstruction—functional	• Drugs resulting in decreased motility (e.g. opioids, barium, atropine) • Idiopathic megacolon • Ileus due to inflammatory disease • Spinal neoplasia • Electrolyte imbalance (hypercalcemia, hypokalemia, hypomagnesemia)
Metabolic disease	• Obesity • Hypothyroidism

EVALUATING THE PATIENT

History

Given the myriad possible causes as well as concurrent problems, getting an appropriate history is very important. Clients may misinterpret stranguria as tenesmus. Not only is asking about the cat's current diet (type, frequency, appetite) important, but also be sure to ask questions to determine whether the patient might be dehydrated (due to decreased intake or increased water loss), may have orthopaedic pain, or may be disinclined to use the litter box because of social or toileting factors (fear, unpleasant litter box).

Mild constipation does not require a great deal of work-up or treatment, but identifying its causes is relevant for management to reduce the chance for progression. Chronic, recurrent constipation results in dilation of the colon and obstipation, which in some cats becomes irreversible, idiopathic megacolon that is refractory to cure due to loss of normal neuromuscular function

Physical examination

On examination, hydration is assessed by assessing skin elasticity plus coat luster, mucous membrane moisture, and eye position (Table 2). Skin elasticity can be misleading in older patients (as well as young kittens) because of age-related changes in body water distribution, elastin, and collagen. Body weight, weight change relative to previous evaluation, body condition score (indicating percentage body fat), and muscle condition score (indicating protein adequacy) help determine progression of dehydration as well as amounts needed to rehydrate the individual.

Diagnostic testing

If a cat is experiencing its first episode of uncomplicated constipation, further testing may not be needed and therapeutic rehydration will likely be adequate.

*Table 2: Estimate of dehydration**

Degree of deficit relative to euhydrated state	Physical examination findings
Mild: About 5%	Slightly tacky mucous membranes or saliva, minimal loss of skin turgor, normal eye position
Moderate: About 8%	Dry mucous membranes, moderate loss of skin turgor, mildly sunken eyes
Severe: 10% or more	Extremely dry mucous membranes, skin not returning to original position when tented, severely sunken eyes, weak and thready pulses, tachycardia, hypotension, altered level of consciousness

*Source: Davis H, Jensen T, Johnson A, *et al*. 2013 AAHA/AAFP Fluid therapy guidelines for dogs and cats. J Am Anim Hosp Assoc 2013;49:149-159.

For recurrent constipation or when complications such as trauma or degenerative joint disease (DJD) or neurologic signs are present, additional steps are recommended. A minimum database consisting of a complete blood count (CBC), serum chemistry profile, total thyroxine (T_4) concentration measurement, and urinalysis should be performed to assess overall metabolic status and to get more information regarding the degree of dehydration.

Abdominal palpation reveals the presence of firm feces in the colon unless the faeces is hidden in the pelvic rectum. Radiographs are required to confirm that the firm mass is intraluminal as well as to identify possible extraluminal problems such as obstructive masses or orthopaedic or skeletal problems. Spondylosis deformans of the lumbosacral vertebral column as well as pain from degenerative changes in the shoulders, elbows, hips, stifles, or hocks may limit mobility, making it harder to get to the litter box or to squat comfortably. Evidence of pelvic fracture or other poorly aligned fractures may be observed.

Sedation may be helpful, allowing gentle manipulation of joints to assess whether range of motion is restricted or if pain is present. All cats with recurrent constipation should have a digital rectal examination. This helps to assess abnormalities of the anal glands, prostate, and pelvic inlet and the presence of rectal diverticulum, polyps, or other obstructive masses. Chronic tenesmus can result in perineal herniation.

Abdominal ultrasonography is useful to assess motility, to further examine abdominal structures, and to collect fine-needle biopsy samples of suspicious lesions. Colonoscopy may be required to biopsy mural or intraluminal masses. Computed tomography or magnetic resonance imaging may be used if an intrapelvic lesion is present or if neurologic deficits are present.

Cats with evidence of neurologic problems (*e.g.* paraparesis, hyporeflexia, urinary retention, regurgitation) should have a complete neurologic examination to rule out sacrocaudal dysgenesis (*e.g.* Manx breed), spinal neoplasia, or dysautonomia.

TREATING A CONSTIPATED CAT

There are five steps involved in relieving constipation in cats

Step 1: Rehydration

The cornerstone of therapy for constipation is rehydration and maintenance of a hydrated state. Fluid therapy for rehydration may consist of intravenous fluids, but subcutaneous therapy is generally adequate. The volume of fluid needed to correct the fluid deficit is based on the patient's previous hydrated weight. If not known, the total protein concentration in conjunction with packed cell volume may be helpful. An isotonic polyionic fluid *(e.g.* lactated Ringer's solution) is appropriate for rehydration subcutaneously. A replacement solution such as Normosol-R (Hospira) or Plasma-Lyte 148 (Baxter) would be better choices should the intravenous route be used. A maintenance solution is preferable for ongoing maintenance therapy to prevent hypernatremia and hypokalaemia, but if subcutaneous use results in discomfort, lactated Ringer's solution may be considered. The volume required for maintaining hydration is 60 ml/kg normal, hydrated weight/day

Step 2: Faeces removal

Removal of the faeces with enemas or manual extraction may be done while the patient is being rehydrated. But do not start dietary therapy, prokinetic agents, and laxatives until the patient has been rehydrated. This is because dietary fiber and medical therapy increase faecal water or interfere with the colon's attempts to resorb water needed for cellular hydration.[3] Administering smaller volumes (e.g. 35 ml) of warm water (or saline solution) mixed with 5 ml of mineral oil, glycerin, polyethylene glycol (PEG or PEG 3350), lactulose, or docusate sodium several times throughout a 24-hour period is safer and more effective than administering the entire volume as a bolus.[1] Because docusate sodium increases absorption of intraluminal contents into the bloodstream, it should not be administered concurrently with mineral oil.

Pediatric rectal suppositories can also be used (*e.g.* bisacodyl, docusate sodium). If the patient is anesthetized or sedated for rectal manipulation (digital examination, manual faecal extraction, or enema administration), use a cuffed endotracheal tube to prevent aspiration from vomiting.

Step 3: Dietary therapy

Soluble fibers (*e.g.* pectin, oligosaccharides) are capable of adsorbing (binding) water and forming a gel. Insoluble fibers increase faecal bulk, resulting in distention and reflex contraction. Both interfere with water reabsorption into the body and should only be considered when a patient is well-hydrated. Different fiber sources have different soluble:insoluble proportions.

Fibers can also be characterized by differences in fermentability. This refers to the ability of intestinal bacteria to produce short-chain fatty acids (SCFA) and gas from the fiber. Moderately fermentable fibers such as beet pulp are preferable to a highly fermentable, high-gas-forming fiber source.[4-6] SCFAs are vital as an energy source for colonocytes and are key in motility. While a psyllium-enhanced dry diet has been shown to be effective in treating constipation,[7] increasing water intake by including wet foods and increasing desirable water stations in the home is beneficial. As with all things in cats, individualization is critical. Regardless of which diet is chosen, reassess the patient to ensure that the diet is having the desired effect.

Step 4: Laxative administration

Cathartics are agents that increase colonic motility. They include hyperosmotic laxatives such as polysaccharides *(e.g.*lactulose) and PEGs or those that irritate and stimulate the mucosa *(e.g.* vegetable oils, sennoside, glycerin).

True laxatives act by other mechanisms. Lubricating laxatives (*e.g.* mineral oil, hairball remedies) impair water absorption from the colon into the body; emollient laxatives (*e.g.* anionic detergent such as docusate sodium) enhance absorption of lipid into the body, but impede water absorption into the body; bulk-forming laxatives (*e.g.* cellulose or poorly digestible polysaccharides such as cereal grain) increase faecal bulk, fermentation, and viscosity.

The steps in treating constipation in cats

Rehydrate the cat and maintain hydration.

↓

Remove the faeces by enema or manual extraction.

↓

Modify the cat's diet (canned low residue diet, psyllium-enhanced dry diet).

↓

Institute laxative therapy.

↓

Administer promotility drugs.

Step 5: Promotility drug administration

Consider promotility drugs after other therapies have been instituted and shown to be insufficient. Cholinergic agents (*e.g.* bethanechol) have undesirable side effects and cannot be recommended.[8] Drugs affecting serotonin 5-HT4 receptors (*e.g.* cisapride, mosapride, prucalopride, tegaserod) have been used to effect.[9-11] These should be given orally, as the transdermal route fails to deliver therapeutic levels.[11] Experimentally, nizatidine and ranitidine inhibit anticholinesterase activity, acting synergistically with cisapride.[12]

If the patient has concurrent medical problems, it may be receiving other medications that might exacerbate constipation. These include those that increase dehydration, such as diuretics, and those that interfere with intestinal motility, such as anticholinesterase and sympathomimetic agents, barium, opioids, tricyclic antidepressants, and some H_1-antihistamines.

WHAT ROLE DOES THE ENVIRONMENT PLAY?

A basic environmental need is to have multiple but separated resources.[13] These include duplicates each of water, food, litter boxes or outside latrines, perches, resting areas, and toy stations. By having multiple sites, separate from each other, the chance of intercat aggression or threat (perceived or real) from other individuals is minimized. Having unhooded litter boxes is important to eliminate the risk of ambush.

Litter boxes need to be large (at least 1.5 times the length of the cat) and very clean. The litter boxes, and all resource stations, need to be easy to access, especially for a cat that is mobility-restricted (*e.g.* due to DJD).

Water stations must also be kept clean and freshened regularly. Feeding small amounts of food frequently results in cats drinking a greater volume of water.[14] Wet food increases water intake significantly, favouring a positive hydration status.

TO CUT IS TO CURE?

Colectomy should be considered a "last resort" for a cat with megacolon that is refractory to medical management and has been struggling with obstipation for more than six months. If pelvic trauma resulting in malunion occurred more than six months earlier, colectomy is likewise justified.

Should pelvic trauma have occurred less than six months ago, however, pelvic osteotomy may be all that is required to prevent megacolon from developing in cats.

Colectomy is a procedure with significant potential complications and should be referred to a surgeon with advanced soft tissue and anastomosis skills whenever it's possible.

SUMMARY

- Early correction and management of constipation will help prevent irreversible problems from developing. The effects of all drugs and dietary manipulations depend on the patient being adequately hydrated. Behavioral and environmental aspects should not be overlooked.
- Clean, attractive litter boxes that are safe and easy to access not only enhance a positive quality of life but also prevent retention of feces or inappropriate elimination.
- Regular follow-up is very important. Assessing the effect of the recommendations on the individual and making adjustments as warranted will provide the best outcome.

View the References for this article on Vet360 App or http://vet360.vetlink.co.za/

COMMENTS ON FELINE CONSTIPATION

By: Dr Liesel van der Merwe. BVSc MMedVet (Med), Lieselvdm.sam@telkomsa.net

- The importance of hydration cannot be overstated. Contrary to the article I will keep the constipated patient on fluids for 2-3 days and only start faecal manipulation after that time. If fluid is to move into the colon you need to administer slightly more than maintenance - being careful to avoid fluid overload. After 24 hours, I will start to instill lactulose into the colon (10 - 15 ml) using a feeding tube. This will be repeated every 12 hours. In many cases, the cat will pass faeces without manipulation after 2-3 days.
- Phosphate/fleet enemas are toxic to cats. The high phosphate causes severe hypocalcaemia and severe electrolyte and mineral derangements which are generally non-responsive to medical management.
- In my experience, in cats with severe recurrent constipation and obstipation, subtotal colectomy can be recommended and has good long-term results. Cats, unlike dogs, do not develop chronic diarrhoea if their colons are removed - and faecal consistency normalises within weeks after surgery. The surgery is best performed by an experienced surgeon.

Replacement of the Prolapsed Third Eyelid Gland

By Amy J. Rankin, DVM, MS, DACVO
VETERINARY MEDICINE

This modified Morgan pocket technique allows normal movement of the third eyelid post-operatively. Just follow these steps and achieve an optimal outcome.

A prolapsed gland is the most common disorder of the third eyelid in dogs. It is commonly referred to as a cherry eye because the prolapsed gland appears as a red mass that protrudes from behind the third eyelid. Prolapsed gland of the third eyelid is more common in young dogs and is overrepresented in some breeds, including American cocker spaniels and English bulldogs.

The exact cause of this condition is unknown, but it is thought to be due to a weakness of the tissues that normally anchor the gland to the periorbita. The condition may be unilateral but is often bilateral, with the glands prolapsing at different times.

Treatment pointers

The gland of the third eyelid produces a large portion of the aqueous tear film, about 30% to 40%. It is important not to excise or partially amputate the prolapsed gland because this may predispose the dog to the development of keratoconjunctivitis sicca later in life. The breeds that are more likely to develop a prolapsed gland of the third eyelid are also the same breeds that are more likely to develop keratoconjunctivitis sicca. Therefore, even with successful replacement of the gland, the Schirmer tear test should be monitored for life in all affected dogs.

Several surgical procedures have been described to reposition the gland and are generally divided into anchor and pocket techniques. The technique described here is a modified Morgan pocket technique. In the original conjunctival pocket technique, a conjunctivectomy is performed and the suture knots are buried, but in this procedure the conjunctivectomy is eliminated and the knots are placed on the anterior surface of the third eyelid to prevent possible trauma to the cornea.

The advantage of the modified Morgan pocket technique is that it allows the third eyelid to move normally post-operatively, whereas some of the tacking procedures restrict the movement of the third eyelid because it is anchored to a rectus muscle or the periorbital tissue of the orbital rim. If the third eyelid is unable to move normally, it may decrease its ability to protect the cornea and spread the precorneal tear film.

Recurrence of the condition is possible even when the surgery has been performed properly, especially in large- or giant-breed dogs.

Note that some veterinarians recommend using a topical corticosteroid preoperatively to reduce the inflammation and swelling of the gland. In general, I do not recommend the use of a topical corticosteroid either preoperatively or postoperatively because I am concerned that it may delay healing of the incision site.

Supplies

- Two Graefe fixation forceps or mosquito hemostats
- Eyelid speculum
- Bard-Parker blade handle and No. 15 blade
- Bishop-Harmon forceps
- Stevens tenotomy scissors
- Castroviejo needle holders
- 5-0 or 6-0 polyglactin 910 suture

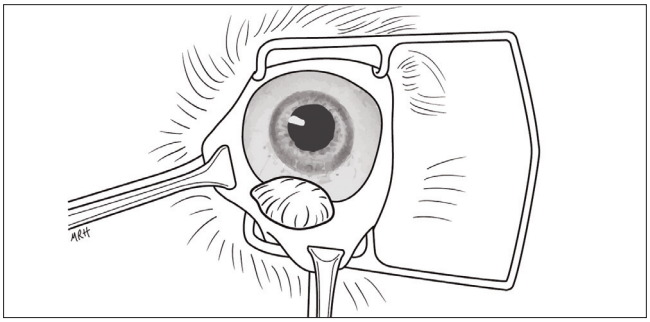

Figure 1

Step 1

Place the dog in sternal recumbency. After preparing the eye with a dilute povidone-iodine solution (1:50 dilution) and rinsing the eye with sterile saline or eye wash solution, place an eyelid speculum to retract the eyelids. Place Graefe fixation forceps or nonabsorbable stay sutures at the medial and lateral edges of the third eyelid, and evert the third eyelid so the bulbar conjunctival surface is exposed. (fig 1)

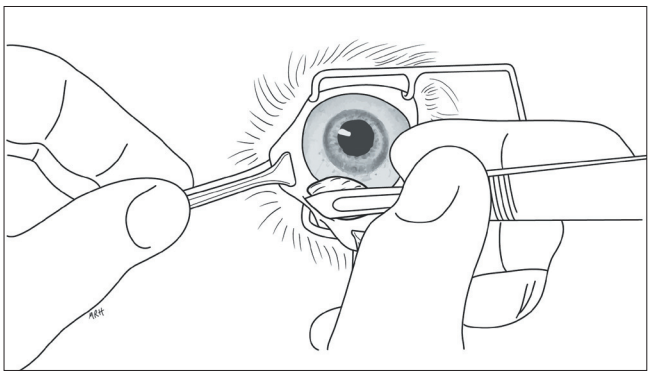

Figure 2

Step 2

Make a curvilinear incision distal to (fig 2) and proximal to (fig 3) the prolapsed gland with a No. 15 blade, but do not connect the incisions. There should be a small opening medially and laterally to allow for tears to flow out of the pocket. The smaller illustration below shows where the incisions should be.

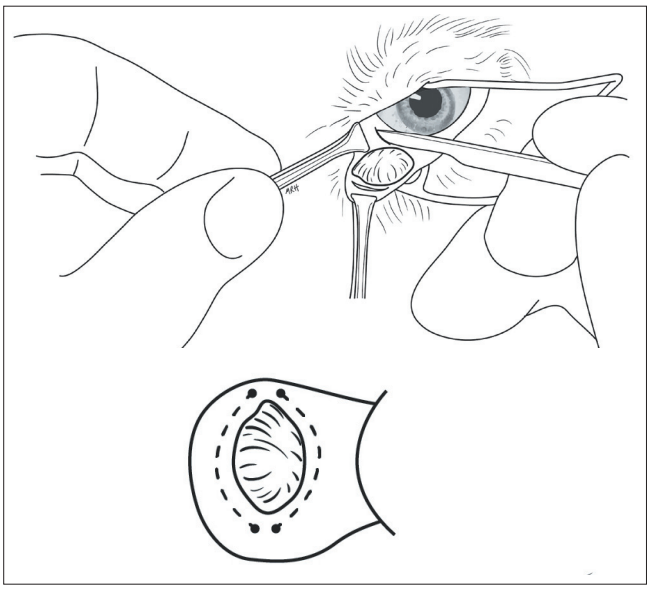

Figure 3

Step 3

Using Bishop Harmon forceps, grasp the edge of the distal conjunctival incision and, with tenotomy scissors, carefully elevate the bulbar conjunctiva from the underlying tissue toward the leading margin of the third eyelid to facilitate suturing the incision. (fig 5)

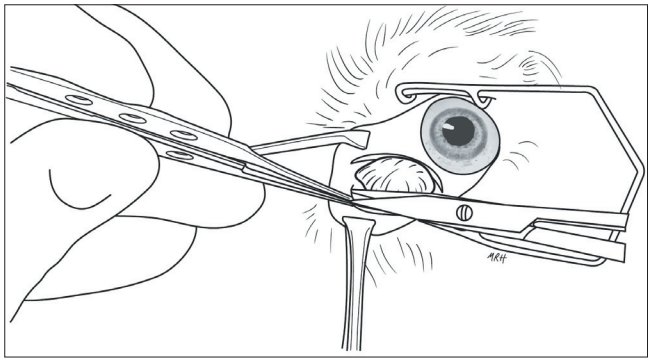

Figure 4

Step 4

Create a pocket in the proximal portion of the third eyelid with tenotomy scissors. The pocket should be large enough that the gland can be easily placed in it before closing the incisions. (fig 5)

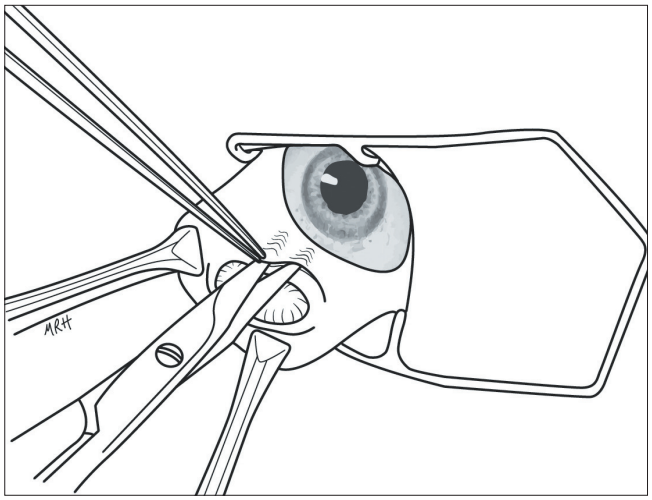

Figure 5

Step 5

Tie an anchoring knot (5-0 or 6-0 absorbable suture such as polyglactin 910) on the anterior surface of the third eyelid (fig 6), and pass the suture through to the posterior surface (fig 7).

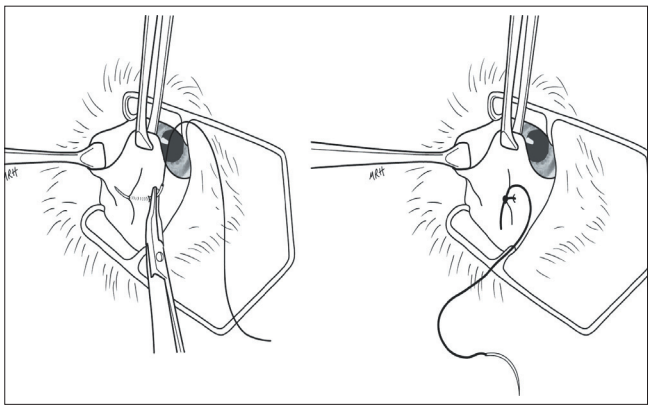

Figure 6

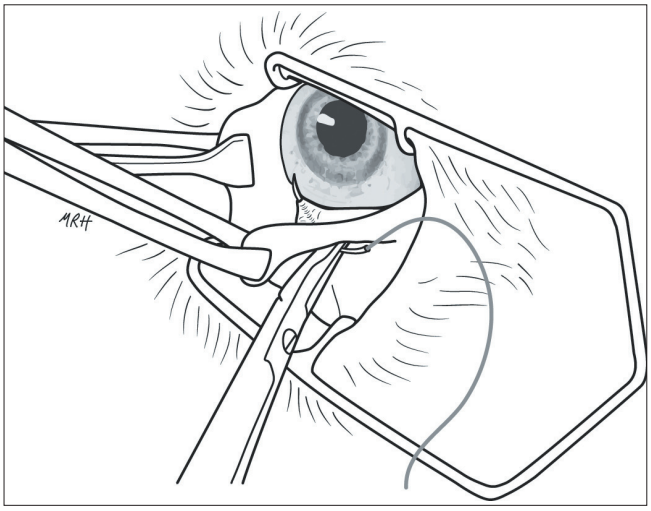

Figure 7

Step 6

Suture the outer incisions together in a simple continuous pattern starting at either the medial or lateral extent of the bulbar conjunctival incision. Leave an opening both medially and laterally to prevent cyst formation and allow for tear drainage (fig 8). (Some surgeons advocate an inverting pattern be placed, such as a Cushing pattern, after the initial simple continuous pattern.)

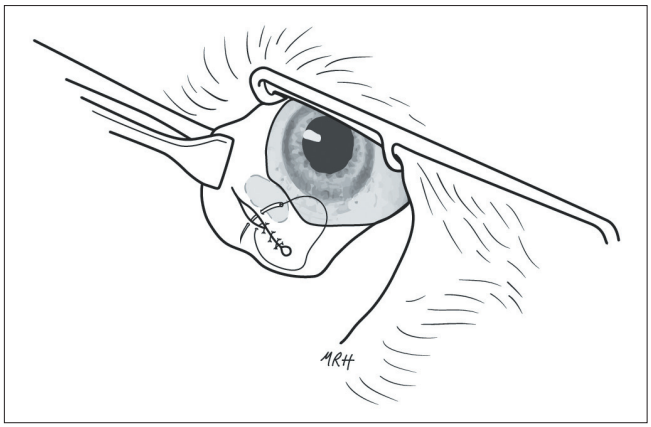

Figure 8

Step 7

After reaching the opposite end of the incision, pass the needle through the third eyelid, and place an anchoring knot on the anterior surface of the third eyelid to prevent the suture from contacting the cornea. If an inverting suture pattern is also placed, the knots should be secured on the anterior surface of the third eyelid (fig 9)

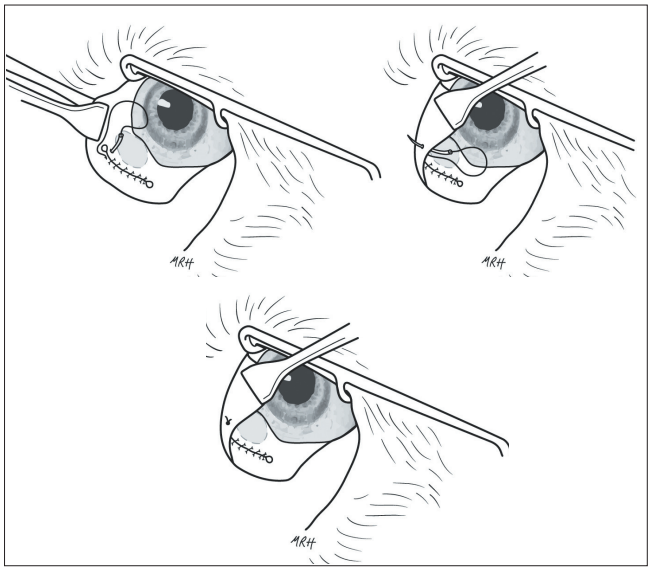

Figure 9

Postoperative care

After surgery, administer a broad-spectrum topical ophthalmic antibiotic medication (e.g. neomycin, polymyxin B, and bacitracin ophthalmic ointment or neomycin, polymyxin B, and gramicidin ophthalmic solution) three to four times a day for seven to 14 days and an oral nonsteroidal anti-inflammatory medication for a few days.

Most dogs do not need an Elizabethan collar postoperatively, but if the patient rubs at the eye or appears uncomfortable, an Elizabethan collar should be worn for seven to 14 days. The sutures do not need to be removed, and the incision usually heals within two weeks.

Additional Comments

Replacement of the Prolapsed Third Eyelid Gland

By Dr Izak Venter BVSc MMedVet (Opthal) JAEHA

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Prolapsed gland of the third eyelid is a condition that can be corrected with a reasonably easy surgical procedure, but proper magnification and the correct small surgical instruments are essential for a good surgical outcome.

This procedure described in the article by Rankin is almost identical to that performed at the AEH. The important issues are:

- The first conjunctival incision next to the leading border of the third eyelid should involve conjunctiva only. If the surgeon cuts too deeply, cartilage will be damaged.
- The second conjunctival incision at the base of the gland should also be superficial. In other words, just conjunctiva. Do not cut into the gland itself.
- Use 6/0 Vicryl for suturing, as thicker material may lead to corneal irritation.
- Placement of sutures needs to be precise. Magnification very important to ensure that this is happening.
- Post-operative treatment using ofloxacin eye drops *qid* for 10 days.

The three most common complications seen in our referral practice in cases done incorrectly are re-prolapse of the gland, ulcerative keratitis and accumulation of tears under the conjunctival pocket.

- Re-prolapse can occur, but with correct identification of the conjunctival wound edges and proper suture placement, this is a very unlikely complication.
- Corneal ulcers are the result of incorrect suture material being used and improper suture placement. The ideal suture material is 6/0 to 7/0 Vicryl. Any thicker material will lead to corneal irritation and secondary keratitis. Due to the size of this material proper small needle holders and magnification are essential.
- The accumulation of tears can occur if the terminal ends of the conjunctival wound are not left open and if the initial conjunctival incision is deeper than just the conjunctiva and involves the actual glandular tissue.

We find that the surgical success rate, if the procedure is done correctly, is very close to 100 %. The exception to this is long standing cases where the glands are inflamed, hypertrophic or ulcerated cases, where previous surgery was done with improper technique and the procedure is repeated and also specifically in Neopolitan Bullmastiffs. Neopolitan Bullmastiffs are renowned for re-prolapse after the surgery and the technique needs to be modified in this breed.

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Part 2 of 3

Doxycycline

So Much More Than Ehrlichia Treatment

Dr OJ Botha (BVSc), VetsBrands

Continued – From Feb 2015. Part 2 of 3 parts.
Full article available on Vet360 App or http://vet360.vetlink.co.za/

Antimicrobial Activity

Doxycycline may be described as an ultra- broad spectrum antimicrobial agent. It exhibits an extremely wide range of activity against aerobic and anaerobic bacteria in both the gram-positive and gram-negative spectrum. Over and above its broad spectrum of activity against the classical bacteria. Doxycycline is also indicated in the treatment of Ehrlichia, Hepatozoon, *Mycoplasma haemofelis,* other Mycoplasma species, Chlamydophila and Borrelia amongst others. (Cunha BA *et al*). Doxycycline has also recently been reported to be effective in treating early infections with Dirofilaria immitis in dogs. (McCall JW *et al*). Initial reports of the anti-viral effect of doxycycline in vivo against Dengue virus was published recently by Rothan *et al*

Anti-inflammatory and immunomodulatory Activity

Doxycycline shows strong anti-inflammatory actions by down-regulating proteolysis, angiogenesis and apoptosis. (Sapadin A *et al,* Shehwaro N *et al*). Danny Scott describes doxycycline as being anti-inflammatory and immunomodulatory in its action by way of the following actions on the immune system:

1. Decrease T-lymphocyte blastogenic response to mitogens.
2. Decrease antibody production.
3. Decrease complement activation.
4. Decrease chemotactic responses of neutrophils.
5. Decrease chemotactic responses of eosinophils.
6. Decrease prostaglandin synthesis.
7. Inhibit collagenase.
8. Inhibit lipase.

Anti-neoplastic Activivty

It has also been suggested that doxycycline may play a role in the treatment of certain neoplastic conditions such as osteosarcoma in dogs. (Cakir Y, Hahn KA). Further research proved that doxycycline reduced the growth of human squamous cell carcinoma in nude mice models. (Shen LC *et al*)

Matrix metalloproteases (MMPs) inhibition

MMPs are a group of zinc dependent proteases that are capable of degrading a wide variety of extracellular matrix proteins. The MMPs also process a number of bioactive molecules that cleave cell surface receptors and release cell surface apoptic ligands. They are also involved in the inactivation of cytokines. They are also thought to play a major role in cell behavior such as proliferation, migration, differentiation, angiogenesis, apoptosis (programmed cell death) and the host defense mechanism. (Van Lint P *et al*). The MMPs are increased in many pathological conditions such as periodontitis, hepatitis, glomerulonephritis, atherosclerosis, emphysema, asthma, autoimmune disorders of skin rheumatoid arthritis, osteoarthritis, chronic ulcerations (including corneal ulcers), bone resorption and tumor progression and metastasis. (Acharya M R *et al*).

Doxycycline exhibits marked MMPs inhibitory activity. They achieve this by chelating Zn^{2+} ion, thereby inhibiting MMPS activity. It is believed that doxycycline also effect MMPs expression and proteolytic activity.(Zakeri, B *et al*). Doxycycline has been proven to exhibit mainly the activity of MMPs2 and MMPs9. (Acharya M R *et al*). Due to the marked inhibition of doxycycline on MMPs it has of late been the focus on extensive research in the treatment of diseases associated with excessive MMPs levels.

Antimicrobial indications:

Ehrlichiosis

In 1979 van Heerden and Immelman published the first report of treatment of canine ehrlichiosis with doxycycline. They recommended a 10 day treatment of 10 mg/kg oid. (van Heerden J, Immelman A). However in 1994 Iqbal published data that claimed 3 of the 5 infected dogs reverted and as such the 10 day treatment period was questioned. (Iqbal *et al*). A study published in 1998 claimed that 14 days treatment was successful in ensuring a PCR and culture negative test in 8 out of 8 dogs tested. However the follow up period in this trial was too short. (Breitschwerdt E *et al*). Following on from this reports of relapses in ehrlichiosis after the recommended 10 days treatment resulted in the company changing the dosage schedule to 20 days.

Eddlestone *et al* reported that they had managed to clear ehrlichiosis with a regime of 5 mg/kg bid. (Eddlestone SM *et al*). However in 2010 it was reported that although a 28 day treatment with doxycycline cured dogs that were in the acute phase of the disease even this extended period of treatment was not sufficient to clear dogs in the chronic phase of the disease. (McClure JC *et al*). In a landmark study performed by Fourie and co-workers, they followed the dogs for 184 days post treatment. During this study it was proven that a 20 day treatment with a generic doxycycline tablet alleviated the clinical symptoms and achieved negative real time PCR tests in 6 out of 6 dogs treated. However 5 of the 6 dogs reverted to clinical symptoms and became PCR positive again 50 days post treatment. When these dogs were treated for 28 days 5 of the 6 dogs remained clinically and real time PCR test negative for 184 days and were truly considered as cured from clinical ehrlichiosis. (Fourie *et al*). the above trial proved conclusively that a 28 day with

doxycycline in acute cases of ehrlichia should be the minimum time for treatment. In cases were dogs are suffering from chronic ehrlichiosis it is recommended that dogs be treated at 10 mg/kg oid for 90 days and then be placed on lifelong treatment of 2 mg/kg oid.

Bordetella brochiseptica in dogs

Bordetella brochiseptica, Pateurella and Staphylococcus were the primary pathogens isolated from cases of lower respiratory infections in dogs. In a study performed in 2000 it was found that all the Bordetella organisms were susceptible to doxycycline as well as amoxcycillin clavulanic acid (Speakman AJ *et al*). However during a recent survey performed in 2012 it was found that the above mentioned organisms were still susceptible to doxycycline but that the incidence of resistance against amoxycillin clavulanic acid had increased significantly. Doxycycline attains exceptionally high tissue levels in the sinus, pharynx, tonsils, lower respiratory tract and lung tissue (Cunha BA *et al*). From the above it may thus be concluded that doxycycline should be one of the first line of defence antibiotics considered when treating upper and lower respiratory diseases in dogs. The effects of matrix metalloproteases on chronic lung conditions in humans and the benefits with long term doxycycline treatment is a topic of extensive research. (Pimenta SP *et al*). Further work in this regard on the possible role of doxycycline in the treatment of chronic airway disease in smaller breeds of dogs is justified.

Bartonella henselae

Bartonella henselae has been described as the cause of pyogranulomatous lymphadenitis in dogs. Treatment with doxycycline was successful. (Morales SC *et al*)

Mycoplasma haemofelis

Doxycycline at 10 mg/kg oid for 20 – 28 days is the treatment of choice against *Mycoplasma haemofelis (*formerly *Haemobartonella felis*). (Sykes JE) It has been reported that as is the case with ehrlichia in dogs relapses after treatment with doxycycline may ensue. As is the case in ehrlichia extended treatments of more chronic cases may be justified. (Messick JB).

Upper respiratory syndrome in cats

Doxycycline is the treatment of choice in cats suffering from *Chlamydophilla felis*. The recommended dose is 10 mg/kg oid for 10 to 20 days. (Gruffydd-Jones T *et al*). Doxycycline at 10 mg/kg for 10 to 20 days is also the treatment of choice for the treatment of *bordetella brochiseptica* in cats. (Egberink H *et al*). Concomitant infections with both of the above mentioned pathogens has also been described. (Di Martino B *et al*) In a recent article published by Schultz and co- workers they found that many cats affected with feline asthma or chronic bronchitis mycoplasma species could be detected with PCR or culture. (Schultz *et al*). In a study where the efficacy of amoxycillin clavulanic acid, Cefovecin and doxycycline were compared against upper respiratory infections in cats, it was conclusively proven that doxycycline was the most effective in resolving the clinical symptoms as well as the reduction of the above mentioned pathogens. (Litster AL *et al*) From the above it is clear that doxycycline should be the first line treatment for any upper respiratory infection in cats. The use of amoxycycillin clavulanic acid and potentiated sulphonamides as first line treatment should be strongly discouraged. In the light of newer research on the matrix metalloproteinase role in the pathogenesis of corneal ulcers and the beneficial effects of doxycycline hereon it makes further sense to use doxycycline as the first line treatment in upper respiratory syndrome of cats.

Chronic *Staphylococcus intermedius* pyoderma

Staphylococcus intermedius pyoderma is a multifactorial disease that in most cases has an underlying allergic component or an immune deficiency component as underlying factor. (Ackerman L) It is often frustrating to treat with relapses being extremely common. The underlying reasons for this syndrome must firstly be addressed adequately before the treatment with antibiotics is taken into account. It was reported that resistance to doxycycline was overestimated when the Kirby Bauer method was used (Blunt CA *et al*). Maaland suggested different breakpoints and zone inhibition for doxycycline so it is critically important that in vitro clinical results be used as the estimation for efficacy of doxycycline treatment in staphylococcal pyoderma cases in dogs. (Maaland MG *et al*). In a study performed by Ganiere JP *et al* it was found that only 6% of *Staphylococcus intermedius* organisms were resistant to doxycycline.

It was also proven that doxycycline was active against methicillin resistant as well as clindamycin resistant staphylococcal organisms. (La Plante La *et al*). Methicillin resistant Staphylococcus *spp* have been successfully treated with doxycycline. (Weese JS *et al*). Although doxycycline is classified as a bacteriostatic antibiotic it reaches extremely high levels in the cytoplasm of staphylococcus due to the fact that the cell wall is highly lipophilic and doxycycline will passively diffuse through and accumulate. Extended periods of treatment with doxycycline have been shown to be extremely safe with minimal side effects noted. (Walker C *et al*).

It is the recommendation of the author that doxycycline be the antibiotic of choice in the long term management of staphylococcal pyoderma. The author recommends that chronic staphylococcal pyoderma be treated initially with the antibiotic of choice as determined by culture and antibiogram (in most cases by a first generation cephalosporin such as Cephalexin) for 14 days. Follow up treatment with doxycycline at 10 mg/kg oid for 3 to 6 months thereafter is recommended. As mentioned before the underlying cause needs to be addressed before antibiotic treatment is considered. (Ackerman L)

To be continued – Next issue will continue Antimicrobial indications. Anti-Inflammatory indications will also be discussed. References will be published in the last section. Full article available on Vet360 App or http://vet360.vetlink.co.za/

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