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Vol 02 | Issue 04 | July 2015

Preparing Your Practice for Sale

Canine Urinary Tract Infections

Accredited CPD: Management of *Diabetes Mellitus*

Also in this issue:
The Coughing Dog | High Protein Diet in Feline Diabetics | Canine Scrotal Urethrostomy

An ear cleaner that has more to offer.

Editor's Note

I am very proud to see that the majority of the articles in this edition are from local authors. We all know how demanding the veterinary profession is and how hard it is to balance work and private life - so thank you to everyone who writes, comments on and reviews articles in this publication.

Whilst editing this edition I also noticed how nicely advertising dovetails with the articles - and thought it prudent to mention that we request advertising after we select articles - so the tail is not wagging the dog! I would also like to thank those companies who have been prepared to place advertising in such a new, unproven publication, your support has been invaluable.

Vet360 and Vetnews will be delivered together - so SAVA members are assured of receiving their copy of Vet360. Veterinarians not affiliated with SAVA can complete the subscription form contained in this edition - the fee covers postage fees and other CPD benefits. We can then post your magazine directly.

Regards

Liesel

We are currently distributing with the VetNews. A win-win situation for SAVA, Vetlink and the reader. Non-SAVA members are urged to subscribe to ensure individual postage. Some magazines are distributed by Virbac Animal Health on behalf of Vetlink. To ensure receipt by post, please subscribe on www.vet360.vetlink.co.za

Index

- Your Business or Practice May Not Be Sold Close To The Value It's Worth 4
- Canine Urinary Tract Infections 7
- Biofilms & Urinary Tract Infections...A Sticky Situation 10
- The Importance of a High Protein Diet in Managing Feline Diabetics 12
- Management of Diabetes Mellitus 16
- Journal Update 23
- The Coughing Dog 24
- Canine Peri-oral Dermatitis 27
- How to Perform a Scrotal Urethrostomy And Stop the Stone Cycle 32
- Fibre Responsive Diarrhoea 36

PREVIOUS EDITION: May 2015
- Transfusion Medicine (CPD)
- Diagnostic Approach to Vomiting Cat
- Hyperlipidaema & Schnauzers

NEXT EDITION: September 2015
- Planning for Retirement
- Chest Drain Placement and Thoracocentesis
- Pruritis in Dogs - CPD accredited

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Your Business or Practice May Not Be Sold Anywhere Close to it's Actual Value

This is the introduction to a series of articles by Mr Deon Nel * addressing retirement and estate planning for vets.

You will have to sell your business at some stage whether you are planning to or not and the chances are very good that your practice is not worth anything near what you think it is!

Sadly most sales are "forced". The unprepared sale normally happens under circumstances such as death, disability, illness or divorce. Somehow we believe that we are infallible and that "it will never happen to me". Not all business owners or practitioners think about preparing their business or practice for sale in case the unforeseen happens.

Even a planned sale will not fetch the true worth of the practice if the practice is not prepared for sale.

Statistically practices are not sold (disposed of) for the value they are worth, particularly when the practitioner is no longer around. Would a spouse, children or any dependant really know what the real value of the practice is?

A forced sale is the time when the buyer has the power to determine the price of the practice. You will have to accept the best offer that you can get at the time. This need not be the case as advance planning could determine your buyer well in advance of the sale.

In South Africa a veterinary practice must be sold within six months of the practitioners' death and it can only be sold to another qualified vet. This means that a veterinary assistant, friend, family member or a spouse cannot take over the practice. Not only are the chances of finding a qualified buyer within 6 months remote, but you will probably not be able to get a good price for the practice because the new buyer holds all the cards.

There is a need for the business owner or practitioner to safeguard their dependants' and family's futures by preparing the practice for a sale or disposal, so that in the event of a calamity the selling decisions can be made and acted upon by people not normally in control.

When you do make the sale – forced or otherwise – the proceeds or liquidity is your prime concern. But please, please, please do not forget your silent partner in all of this...

The tax man!

I have known many practices that have had to pay the lion's share of the proceeds to the tax man, often leaving the vet or his dependants unable to support themselves because the practice was not worth what the vet thought it was.

And the tax man has many tentacles. South Africa may be a third world country but we have one of the strictest and most complicated tax regimes in the world.

This means there are many different taxes that may eat into your proceeds, before you get to put anything in your pocket. Things like income tax, VAT, estate duty and perhaps the most diabolical of them all, Capital Gains Tax (CGT). We will talk about all of these issues in just a bit. It is therefor important to value the practice for the purpose of selling (disposal), whether it is for prepared and planned, or unprepared and unplanned circumstances.

Valuation plays a vital role in the determination of market value in terms of the disposal (sale) of the interests and assets of a business or professional practice. This will be dealt with in a future article.

ISSUES THAT MAY ARISE AT THE TIME OF A SALE?

When an agreement is prepared for the sale of a business or professional practice, there are a number of Income Tax (including CGT) and VAT related issues that should be considered before the sale agreement is finalised.

The key question is whether the business is sold as a going concern or not. Essentially that means that the practice once sold will continue to operate in the future as if the sale had not occurred. For example, all the original staff will be retained by the new owner.

Clearly sellers prefer to sell the practice as a going concern while buyers would prefer to just purchase the operating assets separate from the business instead of buying the entire business as a going concern because it gives them greater tax write-off's in the future.

1. Value–Added Tax

When the business is sold as a going concern and both the seller and purchaser are registered for VAT purposes essentially there will be no negative cash flow effects due to VAT.

However, if the assets are sold individually, VAT will certainly play a part. This can cost either the uninitiated buyer or seller money. You should always consult your accountant about these issues before buying or selling your business.

2. Income Tax and Capital Gains tax

The sale of the business will always be subject to tax! If it is sold as a going concern, Capital Gains Tax (CGT) will be applicable, otherwise income tax is applicable.

Essentially, the only difference is the amount of tax that will be paid. CGT is actually just a form of income tax – they are not two separate taxes! The CGT rate is lower than the normal income tax rate. This is a very complex area of the tax legislation and you definitely want to consult a good accountant about this before you sell your practice.

Where plant and machinery, office furniture and fittings or motor vehicles are disposed of, and have a very low book value (gross cost less depreciation deducted in the past) and are sold for an amount greater than the book value a recoupment will arise which will be taxed in the hands of the seller. This will be to the detriment of the dependants in the case of a sale due to death.

Most assets will be fully written off for tax purposes at the time of the sale of the practice. This means that SARS has already given the seller a tax benefit and should the seller now dispose of the asset for a profit, the full amount of this disposal will now again be taxed by SARS.

For example, if an asset with a book value of R 100 is sold for R 1000, then R 900 will be subject to tax. SARS is recouping the tax that it allowed the practitioner to previously claim, as a deduction when the asset depreciated from R 1000 to just R 100.

3. Liquidity to look after staff and dependants in event of death

Unfortunately, things move very slowly in the Master's office which means that it can often take up to 2 years before an estate is wound up. This often causes unintended liquidity hardships because funds are not readily available to pay staff or dependants.

This problem is further exacerbated by the fact that the veterinary practice has to be disposed of within 6 months of the death of the practitioner.

The best and easiest way to make provision for the liquidity in this instance is via a life insurance policy on the life of the vet, to cover running costs of the practice until disposal and to provide for dependants and family. This is because your solution is 100% assured. The one caveat is in the event of suicide - because an insurance policy will not pay out if the suicide occured within 24 months of taking out the policy.

When taking out the appropriate policy it is imperative to ensure that the proceeds will be adequate. The proceeds need to cover all of the "hidden" costs and taxes that arise. Make sure the proceeds from the disability or life insurance policy cover things like 3.5-3.99% executor fees, income tax, potential VAT liabilities and capital gains tax, as well as estate duty.

As you can see, the best solution is to plan the disposal of your business. Your plan will probably be years in the making!

When it comes to selling your practice, especially in the event of a "forced sale" it is ideal to already have an agreement in place with a pre-determined buyer. This is particularly important to South African vets because of the strict rules regarding the sale and ownership of the practice.

This sale or disposal at death to a predetermined buyer can be concluded by a "buy and sell" agreement, a binding contract between individuals.

Whether you are the buyer or the seller the next thing you would like to do is to ensure that the funds will be

available to buy the practice when the time comes.

HOW TO FINANCE THE PURCHASE OF THE PRACTICE

There are many good investment options available and this is where advanced planning will really pay off. Even if the investment will not cover 100% of the money required to purchase the business, it will certainly be the minimum required for the seller to provide, before a financier would even consider funding the shortfall.

In the event of death, a life insurance policy is actually the cheapest way to finance the arrangement – for example where you know without a shadow of a doubt who will be taking over your practice in the inevitable event of your death. Let's compare taking out a loan to finance the acquisition versus using a life insurance policy (Estate Duty, CGT and CPI ignored for example purposes):

Assumed price of buying a practice:
R1 800 000
Assumed premium of life cover: R3 000 per month
or R36 000 per annum
Cost of capital: = 2.00% per annum

Compare that to a prime interest rate of 9.50% per annum, where the interest only is equal to R171 000 per annum. The life cover will pay out to the policy owner as a cash lump sum without any other or further cost incurred. To pay the loan over a period of 10 years at an interest rate of 9,50% will cost R23 291,56 per month (monthly in arrear) or R279 498,72 per annum. The best option is obvious.

And should things not work out for any reason, you will still end up with a lump sum through a "forced savings" plan.

THE "QUICK FIX"

Because there are so many things that can go wrong when executing an exit plan from your business, whether by planned or forced sale, you should have a contingency plan in place. In the words of the iconic US president Benjamin Franklin "one ounce of prevention is worth a pound of cure"

Any good vet will make sure that he insures his livelihood in case of a sudden calamity. Ironically, this same contingency plan often becomes Plan "A" while the vet is preparing his business for sale. Clearly the starting point for all of this is knowing what your business is worth.

Additional information on this topic available through the Vet360 App and website. www.vetlink.co.za

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Please contact me for a no-obligation chat so that I can explain these issues to you in more detail and we can design an appropriate plan to ensure that liquidity issues are not a thing that should keep you awake at night. Register for a more detailed webinar on www.vetlink.co.za and fill in the Value Builder Questionnaire for a preliminary valuation of your practice.

vet360 Webinars

Vet360 Webinars series features an outstanding selection of practical and informative webinars, brought to you by our business authors and other experts. **UPCOMING - October:** THE BEST SYSTEM TO GET MORE OF YOUR IDEAL CLIENTS INTO YOUR PRACTICE ANY TIME YOU WANT! by James Molfetas. Author of book "Infinite Sales Funnels" http://infinitesalesfunnels.com/

SEPTEMBER 29 @12/20h00

SEPTEMBER WEBINAR TITLE: You will have to sell your practice at some point - make sure it is on your own terms and that you get what it is worth.

SPEAKER: Deon Nel

Recently a vet passed away… his dependents were "forced to sell" the practice within the period prescribed in the current Veterinary Council rules. This has had major implications. We will look at the issues that this case has highlighted because all vets potentially face these issues. Don't let this happen to you. Find out what your options are and the best way to ensure that you get to sell your practice on your terms for top dollar, even in the event of death, divorce or partnership disputes.

DATE: 29 September 2015.
TIME: Session 1: 12h00
Session 2: 20h00
COST: Absolutely Free
RSVP: Book your spot:
http://vetlink.co.za/webinars/

Please Note: We are running 2 sessions on the same day - please choose the time which best suits you. Only 50 spots per session.

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CB is the official publication of the NAVC. CB provides relevant diagnostic and treatment information for small animal practitioners.

Canine Urinary Tract Infections

Gregory F. Grauer, DVM, MS, DACVIM, Kansas State University

Georgia, an 8-year-old spayed black Labrador retriever, was presented with urinary incontinence of several months' duration.

History

Georgia's incontinence, most pronounced when she was sleeping or lying down, had been present for several months and preceded more recent signs of lower urinary tract inflammation (ie, pollakiuria, dysuria/stranguria, breaking normal housetraining behavior).

Examination

Georgia was slightly overweight and had excessive perivulvar skinfolds with some perivulvar inflammation and evidence of licking and pigmentation change (*Figure 1*). Physical examination, including rectal examination and palpation of the urinary bladder, was unremarkable.

Initial Laboratory Evaluation

CBC and biochemistry profile were within normal limits. Urinalysis obtained by cystocentesis revealed a cloudy appearance with pH of 7.5, urine specific gravity of 1.037, 2+ proteinuria, 25 to 30 RBCs/hpf, 10 to 15 WBCs/hpf, 25 struvite crystals/hpf, and gram-negative rods. Urine culture yielded *Escherichia coli* (>1000 cfu/mL) sensitive to amoxicillin–clavulanic acid.

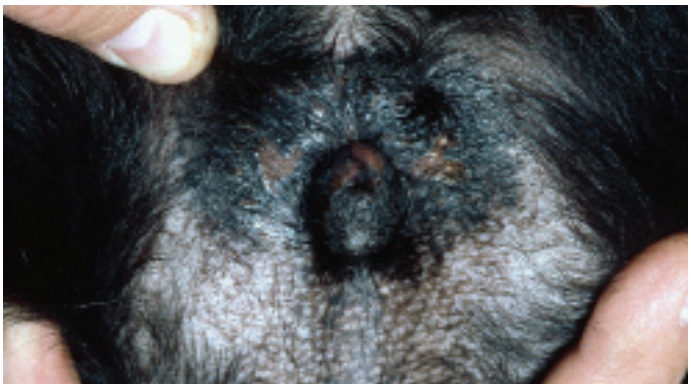


Figure 1. Radiograph of the vulvar region demonstrating recessed vulva and perivulvar dermatitis

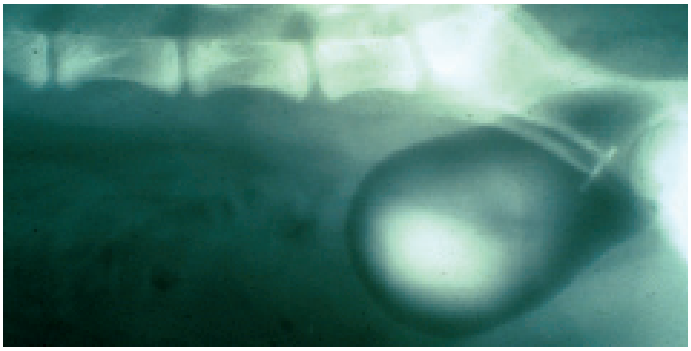


Figure 2. Double-contrast cystogram demonstrating the intra-pelvic bladder

Plain film radiographs of the abdomen followed by a doublecontrast cystogram demonstrated a intrapelvic bladder (*Figure 2*).

ASK YOURSELF?
Is this urinary tract infection simple or complicated?

CORRECT ANSWER Complicated

Simple vs Complicated Urinary Tract Infections

Simple or uncomplicated urinary tract infections (UTIs) lack structural or functional abnormalities in the host's defence mechanisms. This form of infection is easiest to treat and usually clears soon after appropriate antibiotic treatment. Simple, uncomplicated UTIs are the most common type to occur in female dogs.

Complicated UTIs are associated with one or more defects in the host's defence mechanisms: for example, interference with normal micturition, anatomic defects, damage to mucosal barriers, or alterations in urine volume or composition. Health of host defence mechanisms appears to be most important in influencing the pathogenesis of UTIs. Although antibiotic treatment is the cornerstone of UTI management, status of host defence mechanisms is thought to be the most important determinant of longterm treatment outcome. Antibiotic treatment should control the pathogenic bacterial growth for a period sufficient to allow host defence mechanisms to be corrected and prevent colonisation of the urinary tract without further antibiotic administration.

Diagnosis

Georgia had a UTI caused or complicated by probable urethral sphincter mechanism incompetence (USMI), abnormal vulvar anatomy, and subsequent perivulvar inflammation. The intra-pelvic bladder location results in a shortened urethra, which has been associated with USMI. Decreased urethral sphincter

tone allows bacteria to more easily ascend to the bladder. Abnormal vulvar anatomy and USMI resulted in a moist perivulvar dermatitis and increased the number of pathogenic bacteria at the vulvar opening. Excessive perivulvar skinfolds likely occurred secondary to weight gain.

Treatment

Georgia was treated with amoxicillin–clavulanic acid at 13.75 mg/kg PO q12h for 4 weeks, indefinite administration of phenylpropanolamine at 1.5 mg/kg PO q12h for sphincter incompetence, local treatment (astringents, hot-packing, topical antibiotics), and an Elizabethan collar to prevent licking of the perivulvar dermatitis.

Follow-up

The owner reported that the vulva appeared improved and the patient rarely leaked urine and showed none of the previous signs of lower urinary tract inflammation. Urine sediment was inactive. Urine culture 10 days after antibiotic withdrawal showed no growth. Approximately two months after antibiotic treatment was discontinued, Georgia again showed signs of lower urinary tract inflammation (ie, pollakiuria, breaking house training). Urine culture obtained by cystocentesis again yielded *E coli* but with a markedly different sensitivity profile (sensitive to fluoroquinolones)

ASK YOURSELF?
Is this recurrent urinary tract infection a relapse or re-infection?

CORRECT ANSWER Re-infection

Recurrent UTIs

Relapses are infections caused by the same species of bacteria, usually within several days of treatment cessation. With a relapsed UTI, previous antibacterial treatment failed to eliminate infection. Relapses may be caused by improper antibiotic or dose, emergence of drug-resistant pathogens, or failure to eliminate predisposing causes that alter normal host defence mechanisms and allow bacteria to persist (eg, viable bacteria sequestered within struvite uroliths).

In re-infections, previous antibacterial treatment can clear the first infection and the urinary tract subsequently becomes infected with another bacteria. The time between re-infections is usually greater than the time between relapses. Re-infections often indicate failure to eliminate predisposing causes that alter normal host defence mechanisms. Because of the length of time between the UTIs and markedly different sensitivity profile, Georgia's recurrent UTI is most likely a re-infection with a different uropathogenic *E coli*. Even though the phenylpropanolamine and local treatment of the perivulvar dermatitis helped improve host defence mechanisms, abnormalities were still present (USMI, abnormal vulvar anatomy). Subclinical urinary incontinence associated with USMI may persist despite phenylpropanolamine treatment. In addition, episioplasty may be considered if the perivulvar dermatitis persists or recurs.

Ancillary therapies designed to prevent recurrent UTIs are considered in cases where breaches in host defences are present but not correctable or in which an underlying cause for reinfection is not identified. Ancillary therapies include urinary antiseptics and acidifiers, CE (see Cranberry Extract & *E coli*), and prophylactic long-term, low-dose antibiotic treatment. Of note, these ancillary therapies have not been proven effective in controlled, prospective clinical trials.

Outcome

Once Georgia's recurrent UTI had been effectively treated (follow-up urine cultures had no growth), long-term CE treatment was initiated to help support her compromised host defence mechanisms. Recheck urinalyses were performed quarterly. Long-term prophylactic antibiotic treatment was not used because of the potential for creating an antimicrobial-resistant UTI.

AT A GLANCE

Complicated & Recurrent UTIs

- Long-term antibiotic treatment (4–6 weeks) based on culture and sensitivity
- Patient assessment for compromised host defense mechanisms
  - Chemistry profile and follow-up testing to rule out systemic immunocompromise (eg, chronic kidney disease, diabetes mellitus, hyperadrenocorticism)
  - Imaging to rule out local anatomic abnormalities
- Correction/control of compromised host defense mechanisms, if possible
- Prophylactic preventive measures if host defense mechanisms cannot be corrected (only after appropriate antibiotic treatment has sterilis-ed the urinary tract)
  - Urinary antiseptics/acidifiers
  - CE
  - Long-term, low-dose prophylactic antibiotic treatment

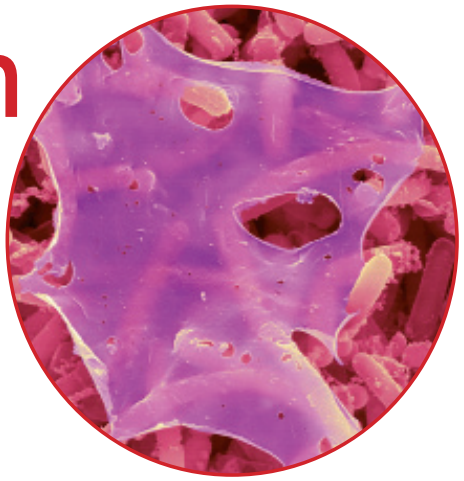
Cranberry Extract & *E coli*

Proanthocyanidins, specifically A-type isoforms found in cranberry extract (CE), can potentially inhibit *E coli* attachment by blocking interaction of bacterial fimbriae with the surface of uroepithelial cells. Canine studies evaluating the efficacy of CEs are limited to in vitro data. One study demonstrated that *E coli* in urine from dogs receiving oral CE (Crananidin, nutramaxlabs.com) had decreased ability to agglutinate to human RBCs. Similarly, a second study demonstrated that *E coli* in urine from dogs receiving oral CE (Paxon, vetoquinolusa.com) had decreased ability to adhere to Madin-Darby canine kidney cells. Based on these studies, oral administration of CE may help reduce *E coli* reinfections in patients with compromised host defence mechanisms.[1,2]

REFERENCES - available on www.vet360.vetlink.co.za

Biofilms & Urinary Tract Infections...

A Sticky Situation



Kelly E. O'Neill, DVM
Mary Anna Labato, DVM, DACVIM
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Tufts University

What is a biofilm and how does it affect the urinary tract of animals?

A biofilm is a structured community of bacterial, fungal, or other cells enclosed in a self-produced polymeric matrix adherent to an inert or living surface.[1] Biofilms are common in living organisms, including animals, but can also be found in other environments. Biofilms alter the function and pathogenicity of bacteria. Growing evidence suggests they may play a substantial role in infections, especially those that are recurrent or difficult to treat.[2]

From the perspective of bacteria, becoming part of a biofilm confers certain advantages. Within the biofilm, bacteria are able to pool their resources and receive protection from the immune system, antimicrobials, harsh environments and other stressors. However, being part of a community may also decrease access to water and oxygen, especially at depths further from the surface, and lead to an accumulation of waste products.[3] Adjusting to these disadvantages may ultimately enable bacteria to thrive in this environment and increase problems for veterinary patients.

Bacteria change from free-living (or planktonic) organisms to ones that can adhere to surfaces (ie, to establish a colony) in order to form a biofilm; in doing so their behavior and structure alter. As the community grows, autoinducers, chemical-signaling molecules that enable bacteria to sense one another and regulate one another's activities, accumulate. As they accumulate, the autoinducers induce changes in bacterial surface attachments, the extracellular polymeric matrix, and the amount and type of virulence factors that are expressed. Alterations in gene expression lead to phenotypic changes in flagella, the structure of cell walls, and the production of enzymes. Up to 40% of cell wall proteins appear to be different in bacteria found in a biofilm compared with their planktonic counterparts.[4]

Most of what we know about the interactions between antimicrobials and bacteria are based on planktonic bacteria. However, biofilm bacteria can be up to 1000 times more resistant to antimicrobials than bacteria that are free-living.[5]

Antimicrobials may not penetrate deeply into the extracellular matrix or may be thwarted by the anaerobic interior. Increased antibiotic resistance develops because the proximity of bacteria increases their ability to share resistance genes with each other. In addition, at any time 1% to 10% of cells within a biofilm slow down because of altered metabolism and expression.[5] Dormant cells exhibit a slower metabolism and are more resistant to antimicrobials. Dormancy is reversible, and when environmental changes occur, these cells can revert to a more active form.[5]

How Biofilms Affect the Urinary Tract

In a healthy animal, the lower urinary tract has many natural defenses that prevent the development of infections. Periodic emptying of the bladder, sloughing of epithelial cells that line the urinary system, and the mucous layer lining the epithelium prevent bacteria from staying in the urinary system. The body's natural immune response, low availability of iron (which is necessary for bacteria to function), and high concentration of urea also make the urinary system inhospitable to bacteria.[6]

Alterations in the normal structure of the lower urinary tract, however, can make it easier for bacteria to adhere, grow, and create a probiofilm environment. Any event that causes trauma to the mucosal layer of the bladder and erodes the glycosaminoglycan layer can result in a disruption in these natural defenses.

Such events include placement of a urinary catheter, the presence of stones, or masses within the bladder or urethra. In animal models of urinary tract infections (UTIs), bacteria instilled directly into the bladder of healthy animals were cleared in 2 to 3 days by natural mechanisms. However, the presence of a surgically placed foreign object in the bladder caused animals to develop chronic urinary tract infections with a con-

current biofilm on the foreign object in less than 24 hours.[7]

Recognising Biofilms

Biofilms tend to develop in the urinary tract—but how do we detect the presence of a biofilm?

Unfortunately, biofilms are difficult to detect. Visualisation techniques used in research include scanning electron microscopy or confocal laser microscopy, neither of which is readily available or useful to detect infection in a live animal. A polymerase chain reaction (PCR) test to search for a biofilm-specific gene is in development but is not yet commercially available.[8]

How Biofilms Affect the Urinary Tract

Biofilms tend to develop in the urinary tract—but how do we detect the presence of a biofilm? Unfortunately, biofilms are difficult to detect. Visualisation techniques used in research include scanning electron microscopy or confocal laser microscopy, neither of which is readily available or useful to detect infection in a live animal. A polymerase chain reaction (PCR) test to search for a biofilm-specific gene is in development but is not yet commercially available.[8]

Suspect the presence of a biofilm under the following conditions:

- Any chronic urinary tract infection, especially when the patient presents with a low bacterial cell count.
- Relapses occur after theoretically successful treatment.
- Antibiotic use fails to clear signs in culture-directed treatment.
- Any catheter-associated infection.[5,9] While a foreign object is not necessary, the presence greatly increases the likelihood of biofilm development.
- Urine culture is negative but the patient responds to antimicrobial treatment.

Treatment

If a biofilm infection is suspected, treatment strategies include prompt removal of implants or foreign material in combination with appropriate antimicrobial therapy. There is no perfect treatment strategy, but options include prolonged antibiotic use (≥6 weeks [this is a highly empirical therapy unsupported by clinical evidence]), higher antibiotic dosages, and using a combination of antibiotics.[6,9] Research suggests that β-lactams and aminoglycosides may help prevent the formation of a biofilm but are less useful once a colony has become established; fluoroquinolones, on the other hand, are better able to penetrate an "older" biofilm colony (ie, a well-established colony that may have secondary bacteria communities and decreased frequency of dividing and growing).[1]

Although there is no clinical evidence to support their effect on the biofilm, the instillation of commensal bacteria shows promise in the treatment of some infections (eg, commensal strains of *Staphyloccocus epidermidis* that secrete the EspA protease to prevent biofilm formation and nasal colonisation by *S aureus* in humans). These low-virulence bacteria cause passive interference with more pathogenic strains, and this strategy has shown success in human cases of recurrent UTI and vaginitis (eg, with Lactobacillus *spp*).[4,10] Research in human medicine is

exploring potential strategies that may prove useful in eliminating these infections, including strategies to induce the dissolution of biofilms and the use of low-energy surface acoustic waves to disrupt biofilm formation. Research is under way to investigate drugs that target biofilm-specific enzymatic activity and promote dispersion signals to break up biofilms to increase susceptibility to antimicrobial therapy.[1,9]

Prevention

One of the most successful strategies in the fight against biofilms is aggressive prevention. Utilising best practices when placing and maintaining indwelling urinary catheters can decrease infection rates. Indwelling catheters should be avoided when possible, and staff should be encouraged to consistently follow general recommendations to eliminate microorganism migration. These recommendations include washing hands with antiseptic soap for at least 30 seconds or using alcohol-based hand rubs between patients if water is not available.[11] In addition, it is critical to avoid contamination when manipulating catheter connection sites. Catheters that become infected or soiled should be replaced immediately.[5] Despite these strategies, rates of catheter- associated UTIs remain high.[12] Biofilms can contribute to chronic infections, and treatment of these infections can be challenging. In the future, new treatment modalities may offer options for preventing and eliminating biofilms.

REFERENCES
Available at www.vet360.vetlink.co.za

We will discuss bone implant infections and biofilms in the next edition. Editor.

Comments from the Laboratory on Biofilms

By Dr Marijke Henton, BVSc, MMedVet (Bact)
IDEXX Laboratories marijke@idexxsa.co.za

This informative article gives good information useful for clinicians struggling with infections caused by biofilms.

Bacteria are genetically complex, and possess many switched off genes, which are quickly activated under the right environmental conditions. They are easily able to acquire DNA from other bacteria, and can even incorporate free environmental DNA into their gene structure. An example of a switch gene is the one that codes for the protective matrix that pathogenic strains of *Staphylococcus* form in the udder, but not in the laboratory. To study such conditions, scientists have to mimic the udder environment, as the matrix is not formed on common culture media.

Bacteria have other synergistic relationships, such as those between members of the *Actinomyces* group and anaerobes. Some biofilms are made up of more than one bacterium, and these often show opposite antibiotic sensitivities.

The prevention of biofilm formation is crucial, and this informative article gives good guidelines.

Although the urinary tract is a common site, keep in mind that biofilms can form elsewhere. Antibiotics affect cellular processes, such as cell wall production (penicillins) or DNA formation (fluoroquinolones). Antibiotics can therefor only act whilst the bacterium is multiplying. Bacteria involved in chronic infections only multiply slowly, and so may escape the effect of a short course of antibiotics.

When submitting a sample in a possible biofilm case, it is crucial to inform the laboratory of this, so that additional culture techniques can be implemented, and culture can be prolonged. Select a laboratory which understands what is required to correctly culture such samples.

As the biofilm adheres tightly to surfaces, it is important to rub a swab firmly over the affected part, to dislodge the film, or submit the catheter tip.

Whilst removing bladder stones or masses, the surface of the bladder should be inspected and any damaged areas selected for sampling.

Always request aerobic and anaerobic culture, even if the affected area is one generally exposed to oxygen. The deeper layers of biofilms are functionally anaerobic, and anaerobic bacteria could be forming part of the biofilm. The best outcome for successful treatment depends on isolating the correct cause or causes of the biofilm, and treating accordingly [prolonged treatment at high doses].

Empirical antibiotic treatment is currently viewed as irresponsible, due to the massive increase in bacterial resistance found world-wide and also in South Africa, and so culture is indicated for such cases.

The Importance of a High Protein Diet in Managing Feline Diabetics

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Reviewed by Drs Marlies Böhm and L van der Merwe

The incidence of diabetes mellitus (DM) has been increasing dramatically in household cats over the past few decades[1,6] and currently affects between 1/100 and 1/500 cats[7,4] In cats, like in humans, obesity is the most important risk factor for, and cause of, diabetes. It has been experimentally shown that a 1 kg weight gain results in a 30% decrease in insulin sensitivity in cats[8].

Feeding style (*ad lib vs.* set meal times), a sedentary life style (esp. indoor confinement), increased age due to easier access to improved veterinary care (typical age is > 7 years at diagnosis) and neutering status (esp. neutered male cats) are also thought to play a role.[1,4-7] Other risk factors include diseases which destroy beta cells (pancreatitis or pancreatic neoplasia) and diseases which result in elevations of hormones that antagonise the effects of insulin (eg acromegaly, hyperthyroidism, hyperadrenocorticism).

Fat cats which are already insulin resistant may become symptomatic when the stress of concurrent disease causes the release on cortisol which further antagonises the effects on insulin.[3,5] Exogenous progestagens or corticosteroids may trigger clinical diabetes in a predisposed individual (the depot formulations are the riskiest because they cannot be withdrawn once side effects become obvious).[3, 4]

If cats are diagnosed and treated early enough many may go into remission i.e. no longer need insulin.[6] Studies have shown that use of long acting insulin, BID treatment with tight euglycaemic control and a low carbohydrate diet increase the likelihood of a cat going into remission. Cats in diabetic remission will continue to have decreased glucose tolerance and should be considered pre-diabetic.

About 25% may become clinically diabetic again at a later stage. Thus ongoing dietary and weight management is essential.

Studies have shown that use of long acting insulin, BID treatment with tight euglycaemic control and a low carbohydrate diet increase the likelihood of a cat going into remission.

Feline diabetes mellitus and management thereof varies from that of dogs due to the following:

1. The majority of cats are initially Type 2 diabetics

In humans, Type 1 diabetes is called insulin-dependent diabetes. It results from the destruction of beta cells and has to be treated with insulin injections in all cases, lifelong. Affected humans have low/non-detectable insulin levels. In humans, Type 2 diabetes is called non-insulin dependent. This means two things: insulin levels in affected humans are usually normal or increased and this type of diabetes can be treated with oral hypoglycaemic pills rather than insulin injections, at least initially.

Impaired insulin secretion, insulin resistance and amyloid deposition in the pancreas make 90% of diabetic cats Type 2 diabetics. Affected cats can have low, normal or high levels on insulin at diagnosis. Importantly, beta cells are not initially destroyed in in these cats, but may be exhausted from having to produce high levels of insulin for long periods. They can recover given time and correct management. Diabetic cats are 6 x LESS sensitive to insulin than normal cats. Weight gain exacerbates insulin resistance

2. Glucose Toxicity

The cat is an obligate carnivore and is thought to be naturally insulin resistant.[4] A carnivore uses mainly protein as energy source to maintain metabolic requirements and only a small amount of carbohydrates. Any excess carbohydrates are stored as fat.[7] Many commercial cat foods are high in carbohydrates (> 50% of calorie content) necessitating higher levels of insulin to be secreted in order to stabilise blood glucose level. Consumption of high carbohydrate foods is the primary cause of post prandial hyperglycaemia, promotes obesity and increases pancreatic beta cell's insulin secretion.[1, 4, 5] Cats are fundamentally carbohydrate intolerant. Persistent hyperglycaemia causes damage to the insulin secreting beta cells as well as damage to the insulin receptors on cell membrane - thus promoting both an insulin deficiency as well as peripheral insulin resistance. Glucose toxicity is dose dependent. The suppression of insulin by glucose toxicity is initially reversible, but will eventually result in loss of beta cells.

Cats which are poorly controlled for periods longer than 6 months have a decreased chance of remission. Normalising the glucose concentration and keeping it as close to euglycaemia as possible allows recovery of the beta cells from glucose toxicity in many cases. This requires insulin. Dietary management alone or oral hypoglycaemiac will not achieve this.

Longer acting insulins (glargine, PZI or detemir) should be injected twice a day for optimal diabetic control. Lente insulins should not be used as their duration of action is not long enough.[3] The starting dose for a non-ketotic diabetic cat is 0.25-0.5 IU/kg twice a day subcutaneously and dose adjustments should not occur more frequently than once every 5-7 days.[3]

Cats that receive adequate insulin therapy and appropriate dietary adjustment early on in the disease process can go into remission and become euglycaemic or have only minimal requirements of insulin.[5,6,8]

3. Stress hyperglycaemia affecting monitoring methods

Blood glucose curves can have significant day to day variations in cats with poor glycaemic control. Additionally the stress of hospitalisation can cause stress hyperglycaemia.[3]

Owner impression of control (PU/PD, appetite, weight gain) is a very valuable monitoring tool. Serum fructosamine reflects glucose levels over the preceding week. Using the change is fructosamine over time in individual cats is probably more reliable than once-off readings, as concentrations can vary considerably between individual cats.

4. Dietary management - changing to high protein diet for obligate carnivores.

The aim of dietary modification is to normalise the cats' body condition –be it obese or suffering from muscle loss, decrease post-prandial hyperglycaemia and minimise fluctuations in blood glucose.

Which diet? The ideal diet should have a high protein content > 40% of metabolisable energy (ME), (>10g/100kCal) and low carbohydrates <15% ME (<25% DM / < 3g/100kCal). Cats on a high protein weight loss formulation will lose weight more rapidly, maintain lean muscle mass and will remain weight stable after weight loss.

The carbohydrate should be a complex carbohydrate with a low glycaemic index (GI) for example barley. The "carbohydrate load" takes into account both the amount of carbohydrate as well as the GI factor. Carbohydrate restricted foods typically have a higher fat content for calorie provision and vice versa. Carbohydrates exacerbate the post-prandial glucose response - which can be attenuated by the addition of fibre - more research is however needed in this area.

Control of energy intake is the next step. The average indoor cat needs 40kcal/kg/day. The optimal feeding regimen for cats with diabetes has not been researched. It does seem however, that when feeding a low carbohydrate (low GI) diet, the timing of the meals does not need to be matched to the insulin injections as clinically relevant, post-prandial blood glucose increases are unlikely. Multiple small meals as well as ad lib feeding routines can be followed, as long as the amount is measured out in the case of overweight cats and is at least the amount required on a daily basis.[3]

Automatic feeders can be used in these situations. Feeding multiple small meals in the day is also considered a better approach to manage a feline diabetic on insulin treatment, as hypoglycaemia is avoided especially when beta cell function improves.[3,6,7]

The insulin dose should be adjusted downwards by 30-50% when switching from a high carbohydrate to a low carbohydrate diet in order to prevent hypoglycaemia.[8]

Conclusion

Insulin resistance and beta cell dysfunction are key factors that can result in the development of type 2 DM in cats. Obesity is a common cause of insulin resistance and together with other risk factors can eventually lead to the development of type 2 DM. By providing a high protein, reduced carbohydrate, reduced fat diet with soluble and insoluble fibre blend diabetic cats can be better managed and exogenous insulin requirements are decreased. Weight management is crucial for both obese and underweight feline diabetics.[1,7]

High protein diets are contra-indicated in diabetic cats with renal or hepatic dysfunction. Remission of the diabetic state in cats is possible with early and aggressive management due to reversal of glucose toxicity.[7] The prognosis for a well-managed uncomplicated diabetic cat with good owner compliance is good.[2, 3]

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ACANA

SINGLES

LIMITED INGREDIENT

LESS MEAT

Management of Diabetes Mellitus

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Diagnosis:

The animal should have a consistent history ie pu/pd, pp and weight loss. Dogs are usually straightforward. Cats are a little trickier. Stress can cause the blood glucose to go well above 20 mmol/l **AND** can spill over into the urine if you annoy the cat for long enough. Usually if the blood sample collection is the only stressor, there won't be enough glucose in the urine to confuse you. Occasionally dogs can do this too. The renal threshold for glucose in cats is 12 – 15 mmol/L and in dogs is about 10- 12 mmol/L. Remember that alpha 2 agonists (xylazine, medetomidine) cause a glucosuria.

If your cat has ketones **AND** glucose in the urine, you can probably believe the high serum glucose is due to diabetes and start insulin.

If in doubt, run a fructosamine.

Treatment

Before you start treating, determine if the patient has any complications of diabetes. Some of these can cause insulin resistance and make stabilisation a lot more difficult.

- Ketoacidosis – if also inappetant and vomiting, a ketoacidotic patient should be hospitalised, placed on IV fluids and initially treated with regular insulin. You also need to search for the trigger for the ketoacidosis (often a concurrent infection/inflammatory process.
- Hypertension – control with amlodipine and / or an Angiotensin converting enzyme inhibitor (ACEi).
- Proteinuria – if you've excluded a urinary tract infection and an ejaculate in the urine, your patient will benefit from ACEi therapy. Diabetic nephropathies are common in humans and there is a huge body of evidence supporting the benefits of ACEi for affected people.
- Cataracts – 80% of dogs with DM will develop these, often acutely. Diabetic cats will also eventually develop lens opacities but they don't usually affect sight noticeably.
- Uveitis – usually lens (cataract) induced. Initially treat with Maxidex, Maxitrol or similar. Remember that there will be some systemic absorption of the corticosteroid which will affect your patient's insulin requirements. Ideally remove lens once patient is stable.
- Peripheral neuropathy – most commonly observed in cats who develop a plantigrade stance.

Screen for common concurrent diseases/conditions:

- pancreatitis
- infections (abscesses in cats)
- urinary tract infection and bladder stones
- hyperthyroidism (cats)
- dioestrus – progesterone causes insulin resistance. Intact female dogs will be easier to stabilise if they are sterilised.
- chronic renal failure
  - NOTE - glucose is dissolved in the urine - and the SG measures dissolved solute. The SG of a diabetic cat with concurrent CRF may not be below 1.035 if there is a strong glucosuria. As a general rule of thumb a 2% or 4+ glucosuria will increase the urine SG by 0.008 to 0.010 when SG is measured using a the refractometer.
  - If your animal has another reason to be pu/pd (eg CRF, hyperthyroidism, Cushing's etc) you cannot use this sign to determine the efficacy of the insulin.

Initial stabilisation

Cats:

- Glargine: start on 0.25 IU/kg bid
- PZI (Protamine zinc insulin): 0.2-0.5 IU/kg bid, maximum total dose of 1 IU per cat per injection
- Caninsulin: start at 0.25-0.5 IU/kg bid with a maximum dose of 2 IU / cat per injection

Dogs:

- Caninsulin: go with instructions in box
- Lente insulin eg Protophane: start on 0.25 IU / kg bid

Aim: abolish lethargy, maintain weight, resolve polydipsia (< 60 ml/kg/d if dry food), no ketonuria. May still be slightly glucosuric at times

Get the owner to keep some sort of diary (example below) so that you both can pick up trends in the pa-

tient's progress. What exactly is recorded can be altered to suit each individual.

	Date	Date	Date
Insulin am – time and amount			
Insulin pm – time and amount			
Food - amount			
Appetite			
Water intake in ml			
Body weight, condition score			
Habitus			
Urine (accidents indoors, how often waking owner at night)			
Exercise			
Hypos / other observations			

Feeding:

Dogs should be fed the same amount ot the same food at the same time every day

- exactly the same amount of food for each insulin injection to "work on". While you're stabilising him he will probably need to eat more than the weight guide of the food suggests. That's OK. You don't want a skeleton, so feed more. But agree on the amount with the client and keep it the same every day till you both decide to change that amount)
- exactly the same food. I don't insist on a prescription diet for dogs unless they have concurrent problems or are proving difficult to stabilise, but do avoid tinned foods with simple carbohydrates.
- at the same time every day. Feed twice a day in dogs, at same times as insulin injections - as close to 12 hrs apart as practical/possible for the owners
- NO TREATS while stabilising. You know your client... if this is impossible, the treats need to be incorporated into the daily allowance and kept the same every day.

Cats won't usually eat at prescribed times. I agree on a daily amount of food in grams with the client and ask them to stick to that. It's important that the cat eats approximately the same amount of food after each insulin injection. If your patient eats 100g of food after the morning insulin injection and only 30g after the evening one, you cannot expect to stabilise the cat unless you also vary your insulin dose. This is tedious (because there's a good chance someone is going to need to check blood glucoses in the middle of the night) and it takes a lot longer to stabilise the cat. Feeding a prescription high protein low carbohydrate diet shows significant benefits in diabetic cats.

Glucose toxicity in cats: Chronic hyperglycaemia is toxic to beta cells and decreases insulin release. The pathogenesis is not clear. Once it resolves and endogenous insulin secretion increases, exogenous insulin requirements may fall drastically or even disappear. Insulin resistance may also improve dramatically as the cat loses weight (studies have shown that a 1kg weight gain decreases insulin sensitivity by 30% in cats!). This means you MUST monitor diabetic cats closely. Your clients may find this tedious but:

* if you get your treatment right, you may be able to wean the cat off insulin completely
* if you don't continue monitoring and the glucose toxicity resolves, the cat will have episodes of hypoglycaemia If you're lucky, signs will be mild (a bit of a wobble), but if you're not, you could have severe neurological symptoms developing: seizures, brain oedema and resultant blindness (thankfully often temporary).

Possible responses to insulin overdose:

- Somogyi overswing (figure 1) - the high insulin dose causes the glucose to drop below 3.3 mmol/l. This stimulates the release of counter regulatory hormones including glucagon, adrenalin, cortisol - which cause a rapid increase in blood glucose. This yoyo effect happens quickly and is one reason why glucose readings should be taken at least every 2 hrs during a curve. If I have a patient in hospital whose glucose readings are approaching 3 mmol/l I will often take more frequent readings for 2 reasons: so as not to miss a hypoglycaemic episode that needs treatment and also not to miss a Somogyi overswing. Animals that have had a Somogyi overswing may have persistently and uncharacteristically high glucose levels for 2-3 DAYS after the glucose dip. If you happen to do your glucose curve during this time, the patient will often appear insulin resistant. This illustrates why it important to take a good history and why you should never make a decision about changing a patient's insulin dose based on one test alone – not even if that test is a glucose curve.
- The patient adapts to low glucose (rather like an insulinoma dog). This becomes more likely the more frequently the patient is hypoglycaemic.

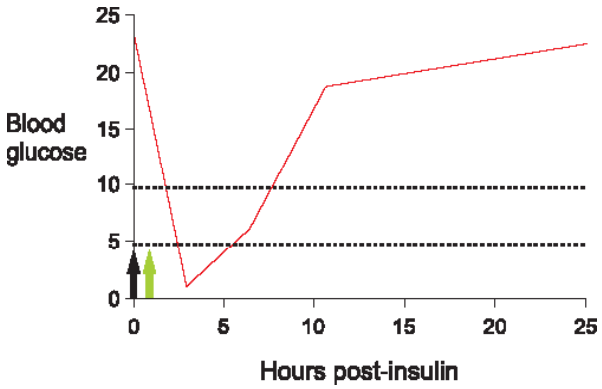

Figure 1: Somogyi overswing: An insulin dose which is too high will cause the nadir to drop very low which may stimulate counter-regulatory hormones which act to rapidly increase blood glucose again. This results in a high blood glucose reading at the end of the 12 or 24 hour period and may lead one to think the dose is actually too low. It is thus vital to sample every two hours at least when performing a glucose curve so that you don't miss the nadir.

Important points

- **Start with a lower dose low and work up**
- **Use insulin bid if at all possible**
- **Don't change dose more frequently than every 5 days (7d for glargine)**
- **NEVER base a decision to change treatment on one observation/result on its own**

These patients only show clinical signs when the blood glucose goes really low.

- Clinical signs of a hypoglycaemic episode (hypo) : Tachycardia, nervousness, tremor, strange behaviour, hunger, seizures, coma.

Assessing response to treatment:

There are various methods than can be used.

Clinical signs: As per the monitoring table. Urine glucose is included amongst these but should NEVER be used on its own to make treatment decisions.

Example: A patient that is experiencing a Somogyi overswing may be persistently glucosuric. If the glucosuria is used as the only measure to determine insulin dose this patient would receive an even higher dose, whereas in fact it needs a dose reduction . Persistent glucosuria is an indication that the patient is not as stable as one would like but further information/testing is needed before you can decide whether a dose increase or decrease would be most appropriate.

It is possible to stabilise a straight forward diabetic using just the clinical signs in the table above.

Fructosamine: This serum protein reflects blood glucose over last 1-2 weeks and requires a single blood sample. It is not affected by stress hyperglycaemia. Serial fructosamine measurements are of more use to monitor improvement or control in the individual patient as the normal range is highly variable in veterinary patients. Fructosamine will not show that there are very low blood glucose nadirs (hypoglycaemia) and will also be falsely elevated by haemolysis and dehydration and falsely decreased by hypoproteinaemia, hypoalbuminaemia, hyperthyroidism, hyperlipidaemia, azotaemia and prolonged sample storage at room temperature

Normal range	225-365 µmol/l
Excellent control	350-400 µmol/l
Fair control	400-450 µmol/l
Poor control	> 500 µmol/l

Glucose curves

How:

- Ideally done at home: spend the time to train the owner. This first 2-3 curves are training for owner and patient
- Measure blood glucose every 2 hours –
  - for 12 hours if bid insulin
  - for 24 hours if *sid* insulin or appears to be doing strange things – (can cut a corner and stop once glucose starts increasing again after the last insulin dose)sampling:
- Use an insulin needle to collect sample if taking from a vein. Put a dressing on the vein – you'll be wanting to sample from here many 10s of times, try and keep it healthy!
- Sample from the edge of the ear to limit ear "squeezing", or if the dog has a tail - clip the hair on the tail tip and use that. This saves the poor ear from being squeezed to death.
- You can get false low results if the blood drop is obtained with difficulty and mainly tissue fluid or serum is being sampled or the sample size is too small!
- Avoid using catheters for sampling if at all possible. Samples are easily diluted by the flush needed to keep the catheters patent resulting in false low readings. This issue means that you need to draw much larger volumes of blood for each sample which can become a problem when your patient is small.

Interpretation of a glucose curve

- Does glucose decrease at all? Is the insulin effective?
- How low does the blood glucose go – is it safe? What is the nadir?
- What is the duration of the insulin's effect?
- How high does it start out at?

Aim: try to maintain a dog's glucose between 3.3 - 10 mmol/l for most of the day and between 3.3 – 15 mmol/l for the cat.

1. If nadir is < 3 mmol/l then decrease dose by 50%
2. If nadir is 3-5 mmol/l or pre-insulin blood glucose is < 10 mmol/l then decrease dose by 20%
3. If pre-insulin glucose is < 5 mmol/l, leave out next dose of insulin and then decrease dose by 20%
4. If pre-insulin glucose is < 3 mmol/l, leave out next dose of insulin and then decrease dose by 50%
5. If nadir is > 8 mmol/l and pre-insulin glucose is > 10 mmol/l – then increase insulin dose by 20%... but if there are no clinical signs of DM, increase by 1 unit only

INTERPRETATION OF A GLUCOSE CURVE

• Does glucose decrease at all? Is the insulin effective?
- How low does the blood glucose go – is it safe? What is the nadir?
- What is the duration of the insulin's effect?
- How high does it start out at

Glucose curves have limitations

- Curves on sequential days are rarely the same. There are lots of variables - the food, exercise, hormone levels...
- Time to nadir can vary from day to day in THE SAME ANIMAL
    - NB Don't try to save money and cherry pick what times you want to sample. A lot can happen in a 3-4 hr gap and you could be missing a very low nadir. Several studies have shown that glucose curves and the timing of the nadir will vary from day to day in the same dog/cat, even when they are stable. So you cannot assume that a dog that had its nadir at 11 am on a previous curve will do the same again tomorrow even with a very good owner who sticks to EXACTLY the same feeding and exercise regime.
    - The only thing a random blood glucose is useful for is to confirm that a hypoglycaemic event is currently happening.

Stress plays havoc with curves – so try and have the clients do them at home

- Only start a curve after the animal has been stable and nothing has changed in the management for 5-7 days
- It is important to only change the dose if your glucose curve agrees with the clinical signs and history. If not, try another curve

Problem cases

These are usually animals whose clinical signs persist despite high insulin doses (> 2 IU/kg/dose), animals who have repeated unexplained hypoglycaemic episodes or animals whose insulin requirements appear to fluctuate. To solve these, consider the following steps:

Visit 1: (a long consultation!)

Status quo: client should have a diary and should be recording. Look at diary and determine trends

Food: take a careful history to determine how closely client is sticking to the prescribed schedule. Ask about treats, rewards, what happens during braai's and parties, how are they coping with small kids dropping food, how are they pilling dog. The carbohydrate load may be important esp in cats: amount of carbohydrate in diet as well as the glycaemic index of the source.

Insulin storage and administration

Storage: in fridge, not next to the freezer compartment, in the dark

Ideally replace every 2 months – if in doubt, use a new bottle and see whether problem resolves

Administration: Insulin should be rolled not shaken to re-suspend it otherwise you break the crystalline structure of the protein.

- Check that the client is drawing up exactly the correct amount - without bubbles.
- Check that the syringes are appropriate to the amount of insulin. Get 0.3 ml or 0.5ml syringes if giving small doses.
- Check insulin is injected s/c not into the skin.
- Check that client is rotating the site and is aspirating. Palpate the injection areas for s/c thickening or injection site reactions
- Use the proper insulin syringes with swaged on needles initially to get the client used to injecting: the needles are shorter, the bubbles are easier to see and fewer bubbles form - so the whole process is less stressful. Once the client is comfortable you could progress to 1ml syringes and 25 G needles.
- Elderly client's often have poor near vision and have difficulty seeing the numbers on an insulin syringe. If you're injecting whole units of insulin, an insulin pen that allows the client to dial in the correct number may be a better be

Insulin absorption: absorption of insulin injected s/c can be variable depending on site, scarring, local blood supply and hydration status. Exclude absorption problems causing apparent lack of response by administering a dose of the normal insulin i/m or a dose of soluble insulin i/v and monitoring glucose every 2 hours.

Look for complications of diabetes that may have developed since induction of therapy. Apart from uveitis, these don't usually cause significant insulin resistance, but you'd want to manage these problems

- ketoacidosis
- hypertension
- proteinuria
- infections
- cataracts, (diabetic retinopathy), uveitis, dry eye

Resolve these issues and then (after nothing has changed for 5-7 days).

DO A GLUCOSE CURVE, ideally at home. Consider the following:

- is the insulin having any effect at all. If no, see 1 - 5
- is there a Somogyi overswing. If yes, decrease dose by 50%
- is the effect lasting long enough. If no, change from sid to bid treatment or change to a longer acting insulin does the insulin drop the glucose levels but just not low enough. If yes, exclude 6 - 8 and increase the dose

Concurrent conditions which may cause insulin resistance:

Insulin resistance is defined as an insulin requirement of = > 2 IU /kg/ injection

- Medications: eg prednisolone, progestagens, propranolol
- Season/pregnancy/dioestrus – spay bitch Common concurrent diseases: pancreatitis, urinary tract infection, dental disease, Cardiac disease, exocrine pancreatic insufficiency and in cats also hyperthyroidism and chronic renal failure, Cush-

ings, acromegaly – more common than you think in cats, any other concurrent infection/illness.
- Somogyi overswing: which is defined as a blood glucose < 3.6 mmol/l and > 17 mmol/l in the same 24 hr period. An overswing may be difficult to actually document because the counter-regulatory hormones released after a hypoglyceamic episode may make the animal appear insulin resistant for several DAYS after the hypoglycaemic event. Thus a Somogyi overswing should be suspected if the patient has shown convincing clinical signs of a hypo historically BUT is persistently glucosuric/hyperglycaemic. It is most often documented in animals that given insulin once daily and who have their insulin doses adjusted according to urine glucose.
- Rapid metabolism of insulin or anti-insulin antibodies - uncommon.
- Glucagonoma, phaeochromocytoma, hypothyroidism – (all very rare in cats)

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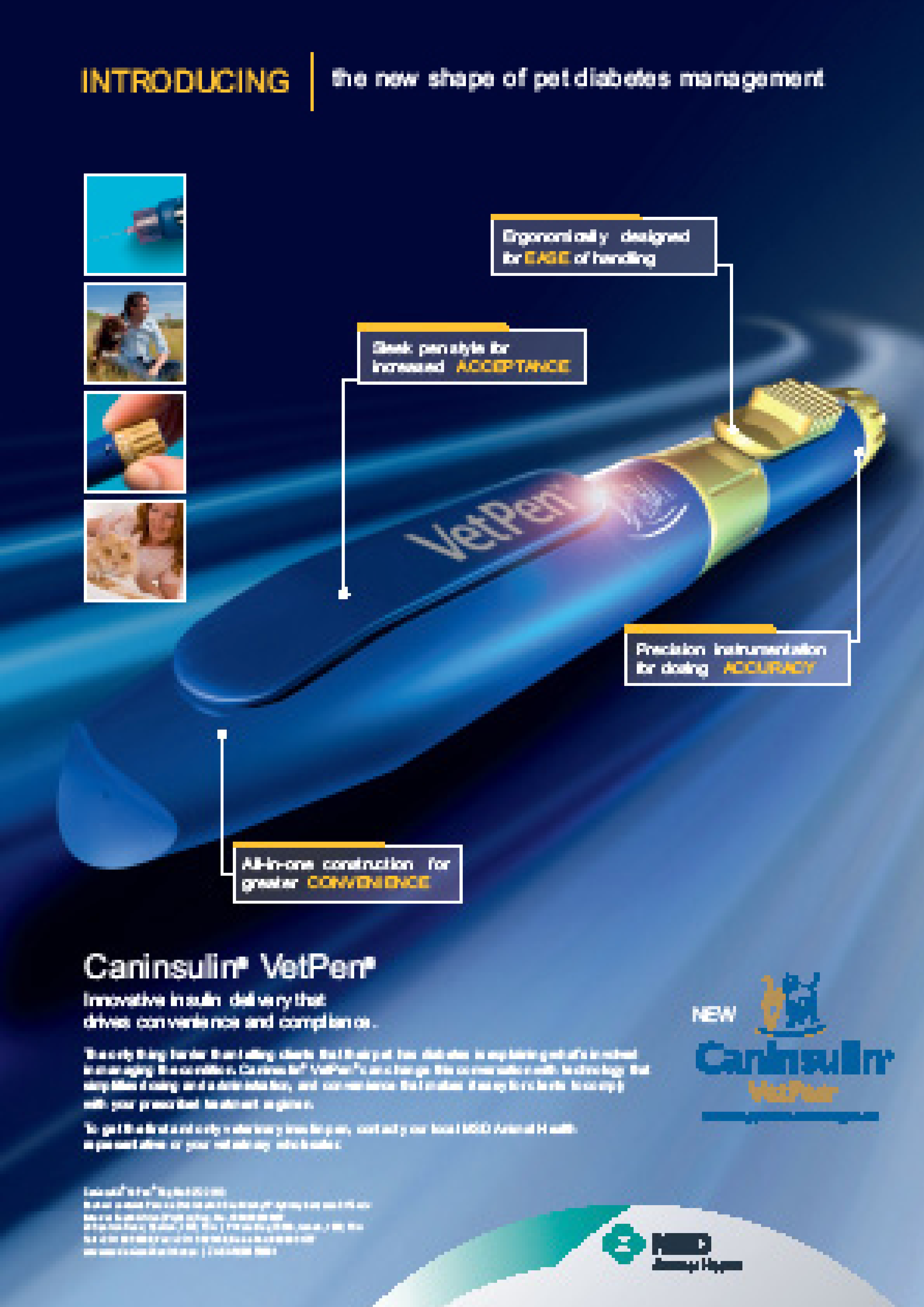
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CPD Questions

AC/1370/15

1. Which one of the following statements is CORRECT?
   a. The renal threshold for glucose in dogs in 3.3 - 10mmol/L.
   b. The renal threshold for glucose in dogs is 10 – 12 mmol/L.
   c. The renal threshold for glucose in cats is 3.3 – 15mmol/L.
   d. The renal threshold for glucose in cats is 12 – 20mmol/L.
   e. Acute stress hyperglycaemia will cause glucosuria.

2. Which one of the statements is INCORRECT?
   a. SG is a measure of dissolved solute.
   b. SG is a measure of urine concentration.
   c. SG is affected by glucosuria.
   d. SG is measured be reftractometer.
   e. SG can always be used to measure efficacy of insulin treatment.

3. Which one of the following statements is INCORRECT?
   a. Glucose toxicity occurs in cats.
   b. Glucose toxicity causes damage to the beta cells.
   c. Insulin resistance can cause glucose toxicity.
   d. Glucose toxicity is irreversible.
   e. Glucose toxicity results from chronic hyperglycaemia.

4. Which of the following statements relating to the Somogyi overswing (SO) is INCORRECT?
   a. The SO may occur when the glucose nadir drops below 3.3mmol/L.
   b. The SO results in a very low pre- insulin injection glucose reading.
   c. The SO is due to the effects of the hormones glucagon , adrenalin and cortisol.
   d. A SO can have a residual effect on the glucose concentrations for up to 3 days after the event.
   e. The presence of SO can cause persistent glucosuria.

5. A diabetic dog receiving insulin treatment is persistently glucosuric. Which one of the following explanations **is not likely to be responsible** for this.
   a. The insulin dose is not high enough
   b. The effect of the insulin is not long enough
   c. The insulin dose is too high
   d. The insulin is being injected S/C
   e. The insulin may be inactivated by heat

6. Which one of the following statements relating to serum fructosamine is INCORRECT?
   a. Fructosamine measures blood glucose over 1-2 weeks
   b. Fructosamine measures blood glucose over 2-3 weeks
   c. Fructosamine has a very variable normal range in veterinary patients
   d. Fructosamine is not affected by stress hyperglycaemia
   e. Fructosamine cannot indicate presence of hypoglycaemia

7. Which one of the following statements relating to glucose curves is INCORRECT?
   a. Glucose curves are affected by stress
   b. Glucose curve samples should be taken every 2 hrs at least
   c. Glucose curves are best performed at home
   d. Glucose curve results are highly repeatable
   e. A glucose curve should only be done 5-7 days after a dose adjustment

8. Which one of the conditions listed is not a complication of *diabetes mellitus*?
   a. Urinary tract infections
   b. Cataracts
   c. Pancreatitis
   d. Calcium oxalate uroliths
   e. Proteinuria

9. Which one of the following statements regarding DM in cats is CORRECT?
   a. Glucose control in cats is improved by a high protein low carbohydrate diet
   b. Cats must be made to eat 2 meals a day to coincide with the insulin injections.
   c. Cats require a medium acting insulin formulation
   d. Glucose curves are effective monitoring tools in cats
   e. Insulin resistance in cats is caused by weight loss

10. Which one of the conditions listed below DOESN'T cause insulin resistance ?
    a. Progesterone
    b. Uveitis
    c. Pancreatitis
    d. Obesity
    e. Proteinuria

ANSWER the questions on the Vet360 App. Available from the Itunes/Play store! Alternative system is available through webbrower - visit www.vet360.vetlink.co.za and follow the link to CPD Solutions

Do you want to complete your CPD on a webplatform or SMS system? Use the following codes for sms or follow the link from www.vet360.vetlink.co.za

- Transfusion Medicine in Small Animal Practice Dr Liesel van der Merwe SMS code = a90559
- Diagnosis of Demodicosis in Dogs & Cats Karen A. Moriello SMS code = a22801
- A review of sterilisation practices and impact on the individual animal, in dogs and cats AC/1225/14 Dr K de Cramer and K May SMS code = a26581
- Rehabilitation of neurological patients By Dr Megan Kelly, DVN, BVSc, Holisticvet SMS code = a97907

Influence of Disease Process and Duration on Acute Phase Proteins in Serum and Peritoneal Fluid of Horses with Colic

Pihl T.H., et al (2015) J Vet Intern Med 29:651–658
Summarised by Patrick Page BVSc MMedVet(Med)Eq

Why they did it

Monitoring of inflammatory biomarkers in blood and peritoneal fluid may assist in differentiation of colic cases requiring medical and surgical treatment. The acute phase proteins, serum amyloid A (SAA), haptoglobin and fibrinogen, have been identified as valuable biomarkers and increase in response to equine inflammatory disorders.

What they did

A prospective, multicentre, observational study of clinical data, as well as blood and peritoneal fluid biomarkers was conducted on horses admitted to two referral hospitals, the Onderstepoort Veterinary Academic Hospital, University of Pretoria, and the University Hospital for Large Animals, University of Copenhagen.

The effect of disease process (colic due to simple obstruction, strangulating obstruction, or inflammatory condition), demographics (age, sex, breed), anatomical disease location, colic duration (<5 hours, 5–12 hours, 13–24 hours, >24 hours, unknown), hypovolaemia, and admission hospital on concentrations of SAA, haptoglobin, and fibrinogen, as well as lactate and white blood cell counts in horses with colic was investigated.

Horses underwent routine clinical examination, rectal examination, nasogastric intubation, abdominocentesis, venous blood gas analysis, faecal analysis for sand or parasite eggs, as well as haematology and serum biochemistry. The final colic classification was based on repeated clinical and clinicopathologic data evaluation and surgical or post-mortem findings, where available. The inflammatory disease classification consisted of cases diagnosed with duodenitis-proximal jejunitis (anterior enteritis), acute typhlocolitis and peritonitis. Data were analysed with multivariate linear regression to identify associations between biomarker concentrations and clinical variables.

What they found

A total of 367 horses diagnosed with colic due to simple obstruction (216), strangulating obstruction (88) and inflammatory disease (63) were included. No significant differences were identified between the 2 admitting hospitals for duration of colic, disease process or anatomical location, while breed distribution varied significantly, as expected.

Increasing duration of colic before admission was associated with increased concentrations of acute phase proteins in blood as well as peritoneal fluid. Overall, acute phase protein concentrations in blood and peritoneal fluid were highest in horses with colic due to inflammatory diseases, followed by those with strangulating obstructions and simple obstructions.

Blood concentrations of SAA and fibrinogen were significantly ($p<0.05$) associated with disease process in more colic duration groups (at 5–12 hours and >24 hours) than the other biomarkers. In general, if a horse had colic for less than 5 hours, none of the biomarkers were significantly different between disease processes. After 5–12 hours of colic, blood SAA and fibrinogen were significantly higher with inflammatory diseases. Similarly, after 24 hours duration horses with inflammatory diseases had significantly higher SAA in both blood and peritoneal fluid, and higher blood fibrinogen.

The marked acute phase protein response in horses with colic due to inflammatory disorders could be explained by the amount of tissue affected and the severity of tissue damage; i.e. tissue damage may be more widespread, for example, with enterocolitis compared to localised strangulating obstruction. Also, strangulating lesions are generally associated with severe pain that results in shorter duration of disease prior to admission.

Take home message

Duration of colic before presentation is an important factor to take into consideration when interpreting concentrations of inflammatory biomarkers and serial measurements are recommended. Serum amyloid A in blood seemed to be the most promising biomarker to aid in differentiating between colic disease process. However, owing to considerable overlap in biomarker concentrations among the disease processes, further evaluation is needed before their diagnostic potential in management of horses with colic can be established.

Coughing Small Breed Dogs

by Dr Russel Leadsom BVSc,Cert VC (Veterinary Cardiology)
Reviewed by Dr Alain Carter BVSc, MMedVet(Med) Small Animals

The small breed dog that presents with both a heart murmur and a chronic cough is often a diagnostic challenge. Is the coughing due to a respiratory condition, a cardiac condition, or both?

RESPIRATORY

Dogs with a cough due to respiratory conditions often have a history of "kennel cough" that never fully resolved. Certain breeds such as Yorkshire Terriers and Chihuahuas may present with a typical "goose-honking" cough suggesting a collapsing trachea that is expiratory if thoracic, and inspiratory if cervical. Living in a household with cigarette smokers is frequently associated with pulmonary problems.

Chronic coughing (when the cough is present for more than 8 weeks) may be associated with chronic tracheobronchitis, chronic obstructive airway/pulmonary disease, bronchiectasis, idiopathic pulmonary fibrosis and pulmonary hypertension. Inhaled foreign bodies, tumours, allergies, enlarged lymph nodes and infections should also be considered.

These dogs are often overweight, and have a normal to slow heart rate. Accentuated respiratory sinus arrhythmia due to excessive vagal tone is frequently detected on ECG, together with tall P waves (P pulmonale) and a right shift in the electrical axis.

Auscultation may reveal inspiratory crackles that are easily misinterpreted as being due to pulmonary oedema and left side congestive heart failure. Expiratory wheezes may also be evident. If crackles have been present long term, they are probably due to respiratory disease.

Radiography may reveal a bronchial pattern (`tramlines and doughnuts`), sometimes with an interstitial pattern obscuring the normal vasculature. Right ventricular enlargement is signified on right lateral radiographs (Figure 1) by increased sternal contact and upward tilting of the cardiac apex (apex tipping), and by a `reverse D` shape on a dorsoventral view (Figure 2)

Pulmonary hypertension (PH) is now recognised as a sequel to chronic mitral valve disease with pulmonary congestion in approximately 30% of dogs. It should be considered when dogs that have been well controlled on standard medications develop an intractable cough, become increasingly breathless with or without cyanosis, and have persistent inspiratory crackles despite increasing frusemide doses. The development of a murmur over the tricuspid valve, together with an increased, or split, second heart sound, and the detection of ascites would also be suggestive of PH.

Confirmation of PH required Doppler investigations.

CARDIAC

It is important to appreciate that most dogs that cough due to a cardiac condition do so in response to an enlarged left atrium - and not necessarily due to congestive heart failure.

Conditions that result in left atrial enlargement include mitral valve disease, patent ductus arteriosus and ventricular septal defects - all of which produce characteristic murmurs.

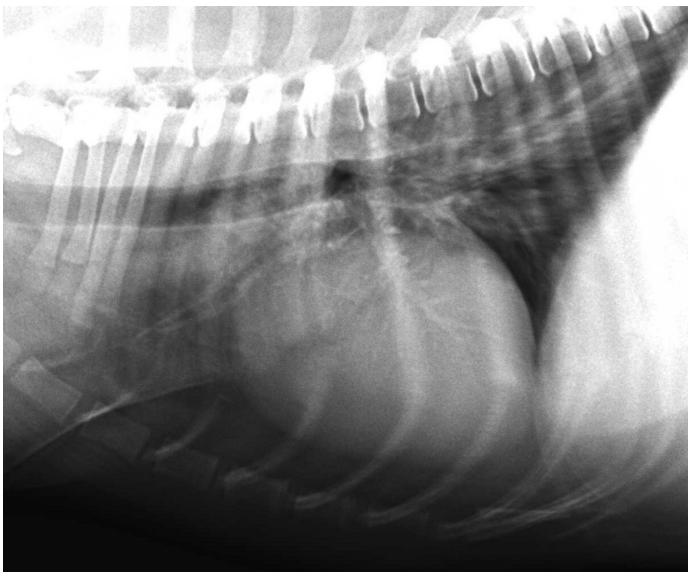

Figure 1. Chronic respiratory disease. Thoracic radiograph, right lateral view

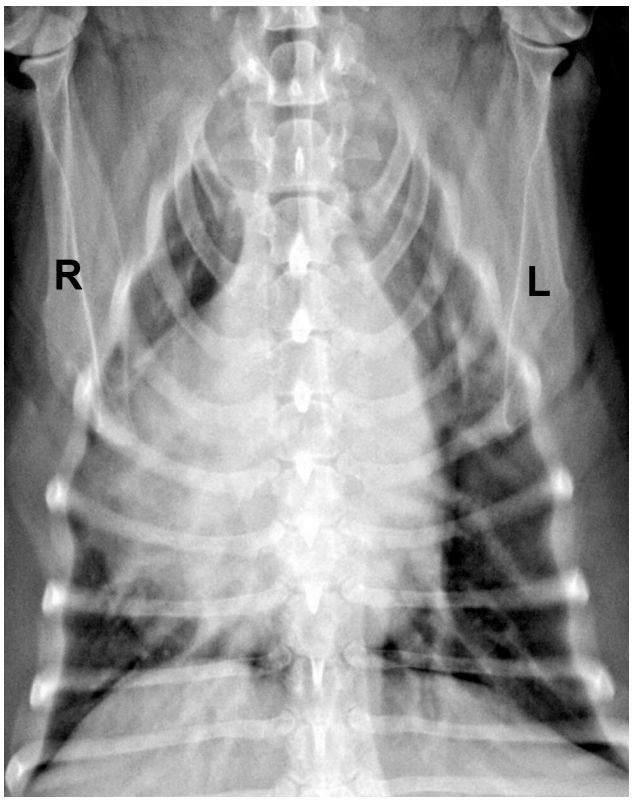

Figure 2. Chronic respiratory disease. Thoracic radiograph, dorsoventral view

The enlarged left atrium causes compression and irritation of the distal trachea and left mainstem bronchus. This does not occur in cats as their left atrium is positioned more anteriorly, and explains why they do not normally cough with cardiac disease.

Dogs with cardiomyopthy that are in left side congestive heart failure (CHF) often have no cough history, this may be explained by the fact that the left atrium is frequently not as enlarged as occurs with mitral valve endocardiosis.

CHF with pulmonary oedema may not produce a cough because there are only minimal cough receptors at the alveolar level. Cough receptors are only more numerous higher up the respiratory tract and are stimulated by irritation, pressure and airway distortion. This explains the presentation of a dog in acute cardiac decompensation with frothy fluid from its nose and mouth - but has no cough!

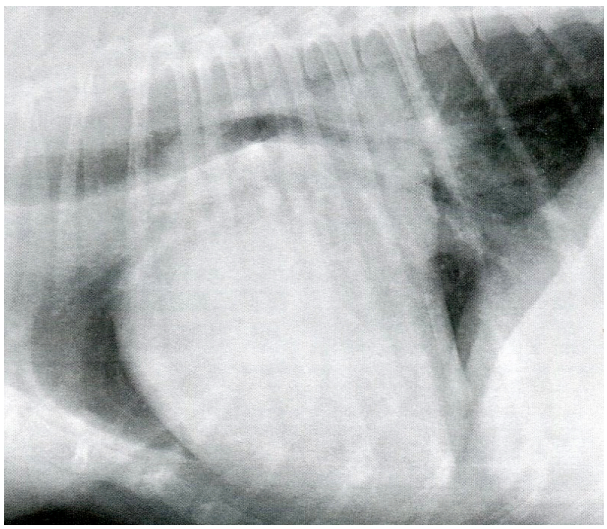

Figure 3. Advanced mitral valve disease. Thoracic radiograph, right lateral view.

Cardiac cachexia (body wasting) is a frequent and serious complication of CHF.

ECG findings include sinus tachycardia, tall and wide QRS complexes, wide P waves (P mitrale), and supraventricular premature beats. Vagal tone is lost as the sympathetic nervous system is activated by CHF resulting in the loss of any bradycardia and sinus arrhythmia.

Radiographs reveal variable left atrial enlargement. On a right lateral radiograph the normal curvature in the dorso-caudal 1 to 3 O'clock position is lost and replaced by a wedge like density of the enlarged left atrium. (Figure 3) The distal tracheal will be elevated dorsally and narrowing (compression) of the left bronchus is often observed. A pulmonary interstial pattern may be identified; with further congestion being initially indicated by an alveolar pattern in the peri-hilar locality.

Vertebral heart scores are useful – but do not confuse right ventricular enlargement.

	CARDIAC	RESPIRATORY
Physical	Often lean due to cardiac cachexia Murmur over mitral valve area	Often overweight
Heart rate	Tachycardic	Normal to bradycardic
ECG	Sinus tachycardia, atrial originating premature contractions P mitrale	 Respiratory sinus arrhythmia, P pulmonale
Radiography	Enlarged left atrium with loss of caudal waist (lateral view) Pulmonary veins congested (larger than arteries) Elevated distal trachea Mild interstitial to severe alveolar patterns	Normal cardiac size or right side enlargement with Increased sternal contact and `apex tipping` (lateral view) Reverse `D` cardiac outline (dorsoventral view) Bronchial thickening produces `tramlines & doughnuts`

CARDIAC AND RESPIRATORY

Dogs with concurrent cardiac and respiratory conditions are frequently encountered. Careful interpretation of the clinical, radiographic, and electrocardiographic findings will generally point to the correct diagnosis.

Measurement of the biomarker pro brain natriuretic peptide (proBNP) serum concentrations may be useful to help confirm or exclude CHF; consideration must be given to the fact that proBNP levels can be elevated in renal dysfunction and in severe pulmonary hypertension.

A trial dose of frusemide for 48 hours may help identify the cardiac patient, whereas prednisolone may alleviate the respiratory condition.

But confusion can occur when a respiratory patient appears to improve on a diuretic; this may be attributed to

1. A reduction in the respiratory tract secretions, this is counterproductive in the long term by increasing viscosity and hindering removal of the secretions and mucus, with resultant `plugging` of the airways.
2. Upper airway obstructive conditions (eg collapsing trachea and laryngeal paralysis) may produce non-cardiac oedema that responds, and benefits from short-term intermittent frusemide therapy.

REFERENCES - available on www.vet360.vetlink.co.za

Comments on the "Coughing Dog"

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As noted in the article the differentiation between the patient with mitral valve disease and congestive heart failure and respiratory disease can be difficult. The age old dogma that a cough and a murmur equals congestive heart failure needs to be broken.

A first step is to look at the heart rate. As noted in the article if the cough is due to respiratory disease then the heart rate tends to be normal to reduced which is the opposite to heart failure where it tends to be increased. The age breed and body condition also help to differentiate between the two conditions. Once a clinical suspicion has been determined chest radiographs will help in identifying the underlying problem. Use of the biomarker proBNP can help as a screening test to include or exclude congestive heart failure.

As with any biochemical test it has to be read with the clinical picture in mind. It is not uncommon in the smaller breeds of dogs to have concurrent chronic respiratory disease and congestive heart failure and these patients can be challenging to diagnose accurately and to treat effectively. It must also be remembered that an echocardiogram can be helpful in diagnosing and managing congestive heart failure but it does not replace chest radiographs which should be performed prior to an echocardiogram.

Canine Peri-oral Dermatitis

Jennifer Schissler Pendergraft, DVM, MS, DACVD
Colorado State University

Profile

Definition

- Peri-oral dermatitis (PD) is inflammation of the maxillary or mandibular cutaneous or mucocutaneous tissues.
- PD has diverse clinical presentations and causes and may be noted as a singular clinical entity or among generalized dermatologic or systemic signs.

Systems

- PD is not limited to lip fold intertrigo (ie, bacterial and Malassezia *spp* overgrowth; (Figure 1); rather, it is a potential manifestation of focal or generalised cutaneous conditions.
- Conditions include hypersensitivities, immune-mediated dermatopathy, infection, hepatopathy, periodontal disease, and neoplasia.

Signalment & Causes

Some causes have known breed and age associations.

Risk Factors

- Redundant lip folds can predispose patients to intertrigo.
- Any primary cause of PD poses risk for secondary bacterial or Malassezia *spp* infection.
- Chronic use of topical or systemic glucocorticoids poses risk for demodicosis.
- Sun exposure can pose a risk for pemphigus foliaceus and discoid lupus erythematosus (Figure 2).

Pathophysiology

- Cutaneous or mucocutaneous inflammation can occur from causes that prompt erythema, pruritus, and primary lesions (eg, papules, pustules, vesicles, bullae), followed by secondary lesions (eg, erosions, ulcerations, crusts, alopecia).
- The resulting skin barrier disruption predisposes patients to secondary bacterial and Malassezia *spp* overgrowth.
- The micro-environment of a deep, redundant lip fold predisposes patients to intertrigo.
- Severe periodontal disease with ptyalism may predispose to secondary perioral infection, particularly with deep lip folds.
- Pruritus and malodour are common.

Figure 1: Lip fold intertrigo in a dog

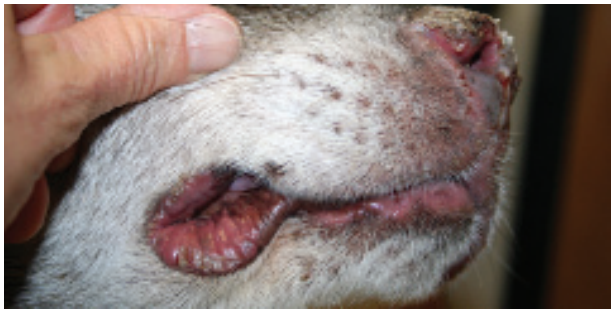


Figure 2: Discoid lupus erythematosus in a dog

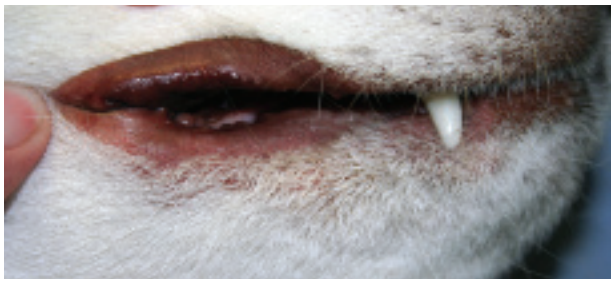


Figure 3: Zinc-responsive dermatitis in a dog

History

Signalment and clinical signs should be noted and history recorded:

- Degree, location, and seasonality of pruritus
- Duration and progression of lesions
- Previous treatments and response
- Dietary history

Physical Examination

- Cutaneous examination (eg, of the footpads, interdigital spaces, and nasal planum) should be completed.
- Otoscopic and ophthalmic examinations should be performed.
- The oral cavity, mucous membranes, and mucocutaneous junctions should be examined.
- Lymphadenopathy should be assessed and lymph nodes palpated.

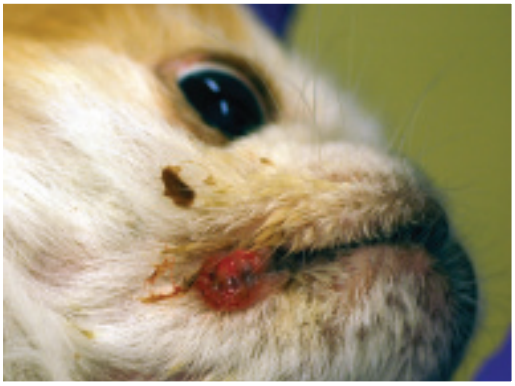


Figure 4: Cutaneous epitheliotropic lymphoma in various canine patients

Diagnosis

Definitive Diagnosis

- Definitive diagnosis is achieved via history, examination, and appropriate diagnostics
- Secondary infections should be resolved, as they can confound clinical and histopathologic features of the primary cause.
- Histopathology is required for diagnosis of immune-mediated disease, superficial necrolytic dermatitis, zinc-responsive dermatitis (Figure 3) and cutaneous epitheliotropic lymphoma (Figure 4)
- Patients with nonseasonal perioral pruritus may require an 8-week prescription or home-cooked elimination diet to differentiate atopic dermatitis (Figure 5) from cutaneous adverse food reaction (CAFR).

Cytology

- Acetate tape preparation (only for dry lesions) and impression smear of exudates should be performed to assess for bacteria, Malassezia *spp*, and presence of acantholytic keratinocytes.

Fine-needle Aspiration

Nodules and enlarged lymph nodes should be aspirated.

Deep skin scrape

Deep skin scrape or pluck for Demodex *spp* (Figure 6) should be performed in all cases.

- If patient compliance impedes the performance of a deep skin scrape, several representative areas (~100 hairs per sample) can be plucked and examined with mineral oil and a coverslip.[1]

Cultures

- Dermatophyte culture is indicated if lesions are consistent (see Causes of Perioral Dermatitis) and secondary infection and Demodex *spp* have been ruled out.
- Bacterial culture is indicated if clinical and cytologic response to antimicrobial therapy is lacking.
- Culture nodular or ulcerative lesions if bacteria are found on cytology; culture superficial lesions if intracellular rods are found.

Elimination Diet Trial

- A strict novel or hydrolyzed diet or home-cooked novel diet should be prescribed for a minimum of 8 weeks to differentiate CAFR from nonseasonal atopic dermatitis.
- Diet should be rechallenged to confirm the diagnosis.

Histopathology

- Vesiculobullous presentations (Figure 7) and lesions that remain after resolution of secondary infection should undergo biopsy.
- Multiple lesions representing all stages of disease should be sampled.

Additional Diagnostics

- Serum biochemistry profile and abdominal ultrasonography are recommended to support diagnosis of superficial necrolytic dermatitis.
- CBC, serum biochemistry profile, faecal flotation, and urinalysis are recommended in cases of adult-onset generalised demodicosis to screen for underlying systemic disease.

Table: Conditions causing peri-oral dermatitis in the dog

Causes of peri-oral dermatitis include but are not limited to the diseases listed. Except for intertrigo, these diseases may manifest without peri-oral involvement

Disease	Clinical signs	Locations, other signs	Age	Breed predisposition
Cutaneous epitheliotropic lymphoma	Alopecia, crusts, depigmentation, erosions, ulcers, erythema, plaques, scales	Any cutaneous, footpads, lymphadenopathy, mucous membrane	Adult	Any
Demodex spp infection Focal (<5 locations) Generalized (>5 locations / pedal / regional)	Erythema, alopecia, comedones, crusts, nodules (advanced), papules, pustules, Scales, ulcers (advanced)	Any, often facial Any cutaneous Ear canal	<1 year Any	Shar-pei Staffordshire bull terrier
Dermatophytosis	Crusts, erythema, papules, pustules, scales	Any cutaneous	Any	Any
Hypersensitivity Atopic dermatitis and/or cutaneous adverse food reaction (CAFR)	Erythema*, pruritus*, scales	Axillae, ear canals, facial, periocular groin, interdigital, perianal, ventral, neck	Any	Any
Immune-mediated cutaneous drug eruption	Any	Any	Any	
Immune-mediated discoid lupus erythematosus (DLE)	Crusts, depigmentation, erosion, erythema, scales	Facial, pinnal, footpads (rare), nasal planum*, periocular	Adult	
Immune-mediated juvenile cellulitis	Alopecia, crusts, nodules, plaques, pustules	Lameness, muzzle, pain, periocular pinnae, purulent otitis, pyrexia submandibular lymphadenopathy*	1–3 months	Dachshund Golden retriever Gordon setter
Immune-mediated pemphigus foliaceus or erythematosus (PF, PE)	Crusts, erythema, pustules	Facial, pinna, footpads, generalized, nasal planum	Adult	Akita Chow chow Cocker spaniel English bulldog Labrador retriever
Immune-mediated uveodermatologic syndrome	Crusts, depigmentation, erythema	Footpad, nasal planum, periocular, uveitis*	Adult	Akita Malamute Samoyed
Lip fold intertrigo	Crusts, deep, redundant lip fold*, depigmentation, erosions, ulcers, moist erythema, purulent exudate		Adult	Basset hound Saint Bernard Spaniels
Mucocutaneous pyoderma	Cheilitis, crusts, depigmentation, erosions, ulcers, erythema, fissures	Anus, nares, periocular, vulva/prepuce	Any	German shepherd dog
Superficial necrolytic dermatitis	Crusts, erosions, ulceration, erythema, hyperkeratosis, scales	Footpad*, hepatopathy or glucagonoma*, hocks, perianal, pinna, Lateral elbow	Adult	Any
Vesiculobullous dermatosis Bullous pemphigoid Epidermolysis bullosa acquisita Mucous membrane pemphigoid Pemphigus foliaceus	Ulcers Vesicles Crusts & pain Bullae	Mucocutaneous Oral cavity Footpad, facial, pinnal Generalized	Adult	Any
Vitiligo	Depigmentation leukotrichia	Any cutaneous Mucous membrane	Adult	Doberman pinscher Rottweiler
Zinc-responsive dermatosis	Crusts, erythema, hyperkeratosis, scales	Facial, footpads, nasal planum, periocular	Any	Alaskan husky Malamute

*Feature occurs in nearly all cases

Note: Other lesions may occur with secondary bacterial or Malassezia *spp* overgrowth.

Figure 5: Atopic lip fold dermatitis with cheilitis and secondary Staphylococcus spp infection

Figure 6: Perioral and facial demodicosis in a dog.

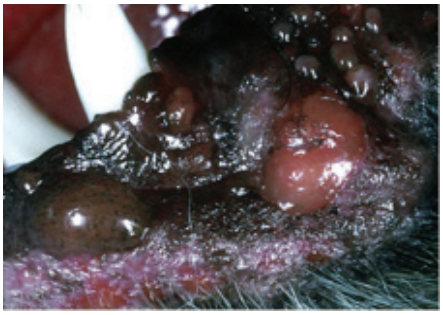


Figure 7: Mucous membrane pemphigoid in a dog

Treatment

Lip Fold Intertrigo

- Daily use of topical antiseptic and drying agents should be initiated.
  - 2% acetic acid, 2% boric acid, and antimicrobial-based wipes and solutions are appropriate for maintenance therapy.
- Acid- and alcohol-based topical medications are not recommended for erosive or ulcerative lesions.
  - If erosions, ulcers, crusts, nodules, or depigmentation are present, systemic antimicrobial therapy is indicated (see

Bacterial Infection & Mucocutaneous Pyoderma).

Cheiloplasty is curative in patients that have lip fold intertrigo (with no other underlying cause) and are refractory to maintenance therapy.

Bacterial Infection & Mucocutaneous Pyoderma

- Topical therapy is recommended in all cases.
- Systemic antimicrobial therapy is recommended for erosive or ulcerative, crusting, and depigmentation presentations.
- If cocci are found on cytology, appropriate empirical choices include:
  - Clindamycin at 11 mg/kg PO q12–24h
  - Cephalexin at 22–30 mg/kg PO q12h
  - Cefpodoxime at 5–10 mg/kg PO q24h
  - Cefovecin at 8 mg/kg SC q14d up to 2–3 times
  - Amoxicillin–clavulanate at 13.75 mg/kg PO q12h
- Solutions and wipes containing 2% acetic acid, 2% boric acid, or 2%–4% chlorhexidine may be used daily and maintained q3–7d after resolution.
- Avoid acid or alcohol-containing products if erosions are present.
- Shampoos containing 2.5% benzoyl peroxide or 2%–4% chlorhexidine may be used 2–3 times weekly.
- For mixed infections and ulcerative lesions, silver sulfadiazine cream should be considered.
- Mupirocin ointment is most appropriate for methicillin-resistant Staphylococcus *spp* infections.

Malassezia *spp* Infection

- These infections should be treated topically.
- Solutions and wipes containing 2% acetic acid, 2% boric acid, 2%–4% chlorhexidine, 2% miconazole, or 1%–2% ketoconazole may be used q24–48h until resolved, then maintained 1–2 times weekly.
- Focal, dry presentations should be treated with daily applications of ointments with 1% clotrimazole, 1%– 2% miconazole, 1% terbinafine, 4% thiabendazole, or nystatin.
- Shampoos with 1%–2% ketoconazole, 2% miconazole, 2%–4% chlorhexidine, or 2.5% benzoyl peroxide may be used 2–3 times weekly.
- Adjunct systemic therapy for generalized or severe multifocal presentations should be initiated:
  - Ketoconazole at 5–10 mg/kg PO q24h
  - Itraconazole at 5–10 mg/kg PO q24h
  - Fluconazole at 10 mg/kg PO q24h
  - Terbinafine at 30 mg/kg PO q24h

Dermatophytosis

- These infections should be treated systemically and topically (see Malassezia *spp* Infection).
- Lime sulfur may be used for generalized presentations.

Immune-Mediated Conditions*

- Mild focal presentations may be managed with 0.1% topical tacrolimus (Protopic®, protopic.com) and/or doxycycline and niacinamide.
- Typically severe presentations initially require prednisolone at 2 mg/kg q24h.
- A secondary immunomodulator may be administered as a steroid-sparing agent or to achieve remission.
- Immunomodulating agents include azathioprine, cyclosporine A, mycophenolate, and chlorambucil.
- Diagnosis, condition severity, and potential adverse effects will influence treatment choices.
- Juvenile cellulitis (Figure 8) is treated with prednisolone as sole therapy.
- For mild cutaneous drug eruptions, discontinuation of the drug alone may be sufficient.

Generalized Demodicosis

Treatment options include:

- Amitraz dips q14d
- Ivermectin at 0.3–0.6 mg/kg PO q24h
- Milbemycin oxime at 1–2 mg/kg PO q24h
- Doramectin at 0.6 mg/kg PO or SC q7d2
- Topical 10% imidacloprid and 2.5% moxidectin q7–14d3 (off label)

Epitheliotropic Lymphoma*

- Therapies include prednisolone as well as oral, inject-

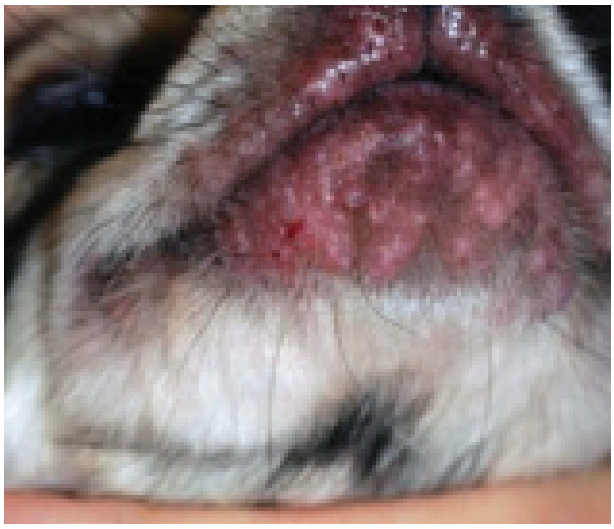


Figure 8. Juvenile cellulitis in a dog

able, and topical chemotherapeutic agents.
- Consultation with a veterinary oncologist is recommended.

Superficial necrolytic Dermatitis

- Treatment includes topical antimicrobial therapy and IV and/or oral amino acid supplementation (Aminosyn, hospira.com) to manage dermatologic lesions.
- Hepatopathy is treated symptomatically, and surgery or octreotide may be considered for pancreatic glucagonoma.[4]
- Prognosis for survival is poor to grave.

Zinc-Responsive Dermatosis

- Lifelong supplementation with zinc gluconate at 5 mg/kg PO q24h, zinc sulfate at 10–15 mg/kg PO q24h, or zinc methionine at 1.7 mg/kg PO q24h is required; additional topical or oral glucocorticoid therapy may be needed.

Hypersensitivity

- CAFR is controlled via restrictive diet.
- Treatment options for atopic dermatitis include but are not limited to:
  - Immunotherapy
  - Cyclosporine A
  - Antihistamines
  - Fatty acids
  - Adjunct topical anti-inflammatory and/or antimicrobial therapy

Follow-up

- Requirements depend on cause.
- For infections, recheck clinical and cytologic response in 2–3 weeks to ensure topical and/or oral therapy is effective.

REFERENCES - available on www.vet360.vetlink.co.za

How to Perform a Scrotal Urethrostomy And Stop the Stone Cycle

By Don R. Waldron, DVM, DACVS
VETERINARY MEDICINE

Male dogs suffering from recurrent urethral blockage due to urinary calculi may benefit from this procedure that permanently diverts urine flow.

Scrotal urethrostomy is a permanent urinary diversion procedure performed in dogs that have chronic or recurrent urethral blockage due to urinary calculi. Urethral calculi in male dogs may cause partial or total urethral obstruction by lodging in the urethra, usually at the base of the os penis. Less common indications for urethrostomy include urethral stricture from previous calculus injury or surgery, penile or preputial neoplasia, or severe trauma that necessitates penile and preputial amputation.

Scrotal urethrostomy requires scrotal ablation and neutering of intact male dogs. Complications of urethrostomy in dogs include haemorrhage from the urethra during the immediate postoperative period and an increased risk of urinary tract infection long term. Stricture of the surgical site is uncommon because of the width of the urethra at this level. Haemorrhage and stricture are both minimized by accurate apposition of the urethral mucosa to the skin.

Preoperative assessment and preparation

Before the surgery, perform appropriate blood work to ensure the patient's stability, giving special attention to renal and electrolyte values. Perform abdominal and perineal radiography or ultrasonography to assess the entire urinary tract for calculi. After anaesthesia is induced, position the dog in dorsal recumbency and prepare the caudal abdomen, including the prepuce and scrotum, for surgery by clipping and performing appropriate aseptic preparation.

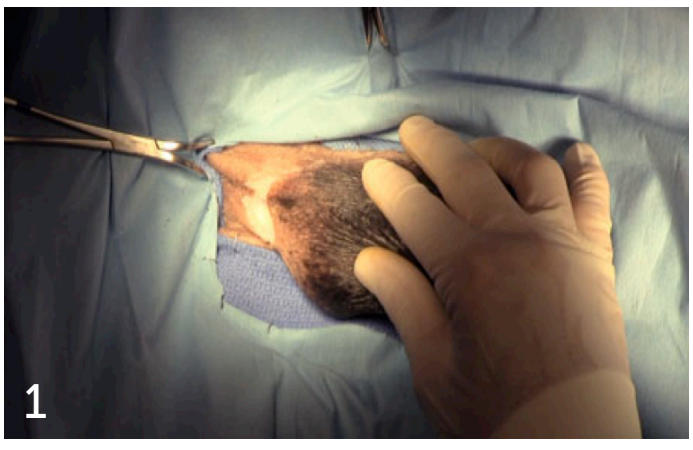
If the patient is urethrally obstructed, calculi can be flushed back into the urinary bladder via retropulsion involving careful catheterization and fluid lavage while the patient is anaesthetised but before starting the urethrostomy procedure. If cystic calculi are present, perform a cystotomy before starting the urethrostomy procedure to remove all bladder calculi. If possible, place an indwelling urethral catheter, which is to be maintained during the urethrostomy. Perform the urethrostomy before the bladder and abdominal walls are closed to allow for normograde and retrograde urethral catheter passage and lavage to ensure urethral patency.

How to perform a scrotal urethrostomy: A step-by-step guide

NOTE: The dog's head is to the left.

Step 1:

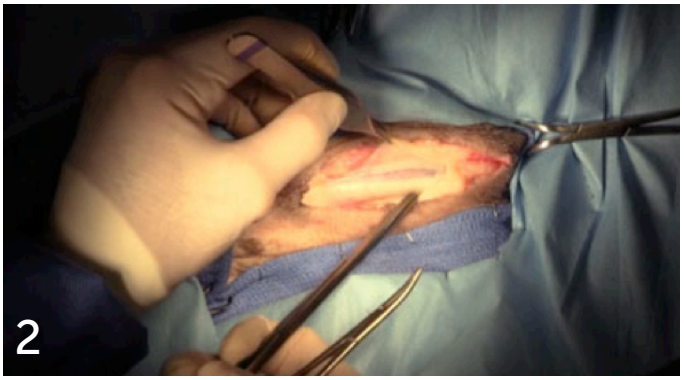
In intact dogs, make an elliptical skin incision near the base of the scrotum, preserving about 1 cm of scrotal skin on



each side. Excise the scrotal skin, and neuter the dog with an open or closed technique. In neutered dogs, excise the scrotal remnant or make a midline skin incision over the caudal penile body, extending caudally until the urethra starts to curve dorsally.

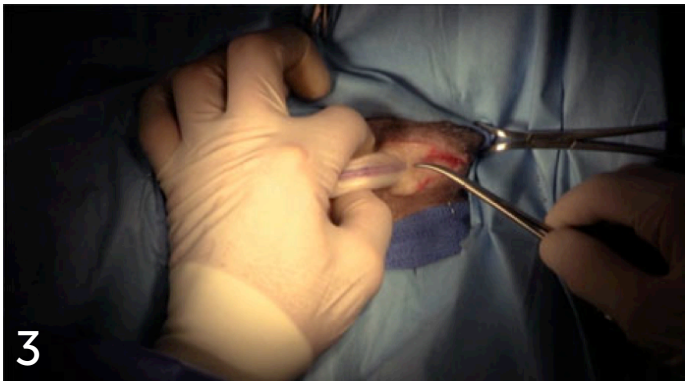
Step 2:

Incise subcutaneous fat to expose the ventral penile body and the retractor penis muscle attached to the penis. Bluntly elevate the retractor penis muscle, and displace it laterally to either side, exposing the urethra. The urethra is purple and located on the ventral midline of the penis.



Step 3:

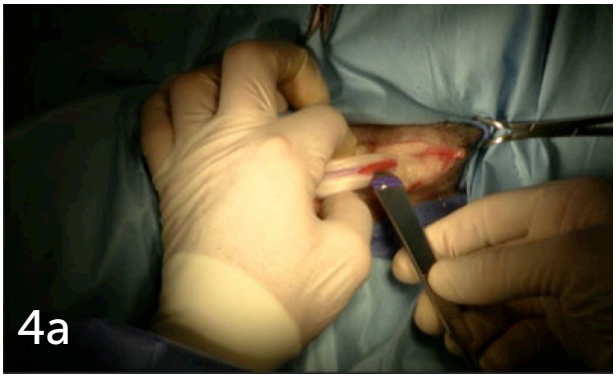
Use the thumb and index finger of your non-dominant hand to elevate the penis from the incision before incising the urethra, which results in decreased haemorrhage from the incised corpus spongiosum tissue surrounding the urethra

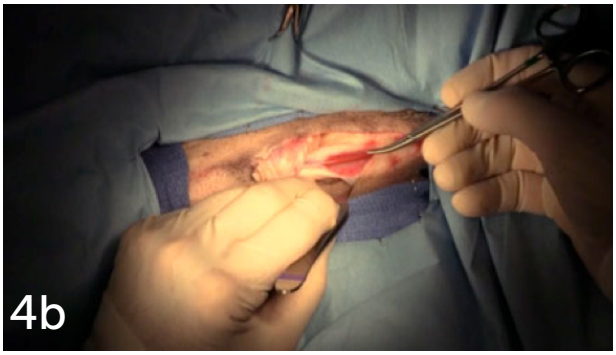


Step 4:

Use a No. 11 or No. 15 scalpel blade to incise the urethra on the midline over the previously placed urethral catheter. After you enter the urethral lumen, use tenotomy or iris scissors to extend the mucosal incision to a total length of 3 or 4 cm

Digitally elevate the urethra to decrease haemorrhage. Suctioning may improve the visibility of the urethral lumen and, especially, the urethral mucosal edge, which tends to retract away from the penile body.





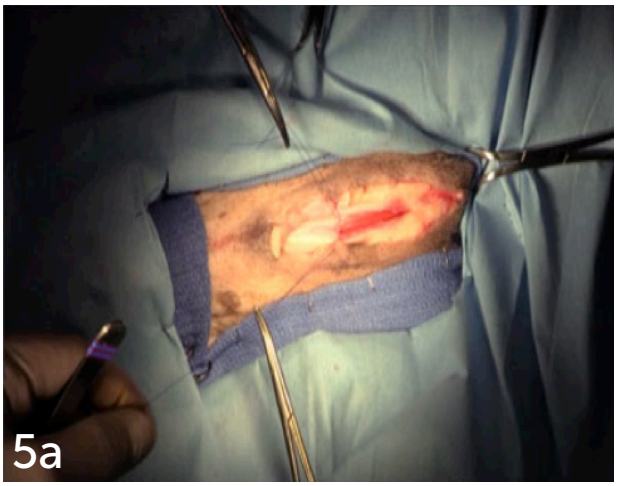
Step 5:

Using 4-0 polypropylene suture on a tapered needle, suture the cranial and caudal aspects of the urethral incision to the skin to establish the boundary of the urethrostomy. Careful suturing of the urethral mucosa to the skin is emphasized. Initially, stay sutures are placed to provide tension on the ends of the incision and will be tied later (see photos above).

The sequence of urethrostomy suturing is as follows: First incorporate a 2- or 3-mm purchase of urethral mucosa only, then a small purchase of the tunica albuginea (white fibrous-appearing tissue of the penis), and, finally, a small, angled purchase of the scrotal skin with the needle. The skin purchase engages the dermis and epidermis only. Taking the purchases separately minimizes mucosal trauma. This sequence of needle passage produces compression of the corpus spongiosum tissue and reduces postoperative haemorrhage from incised tissue.

After the corners have been sutured, place a simple continuous suture pattern between the caudal and cranial sutures on each side. End the continuous pattern by tying it to the tags of previously placed interrupted or stay sutures.

Modifications of this technique include the use of absorbable suture, such as poliglecaprone 25 or glycomer 631. Interrupted sutures may be placed to construct the urethrostomy stoma, with the medial suture tag being cut short.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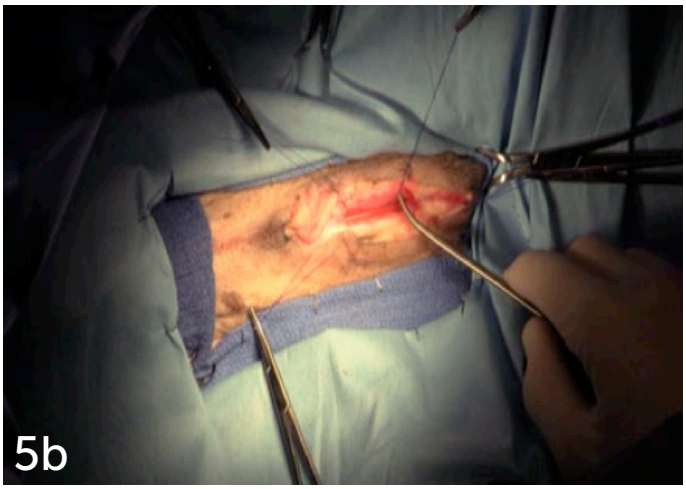
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Step 6:

At the conclusion of surgery, an 8- to 10-F red rubber catheter (not pictured here) should be able to be easily passed from the urethral stoma to the urinary bladder.

Postoperative care

Urethral catheterization is not maintained after surgery. Place a self-restraint collar, and advise owners to keep it on the patient for one week. Occasional dripping of blood concurrent with or at the end of urination is common for the first three to five days after surgery. Nonabsorbable sutures are removed two weeks after surgery with the patient sedated, and absorbable sutures will fall out spontaneously.

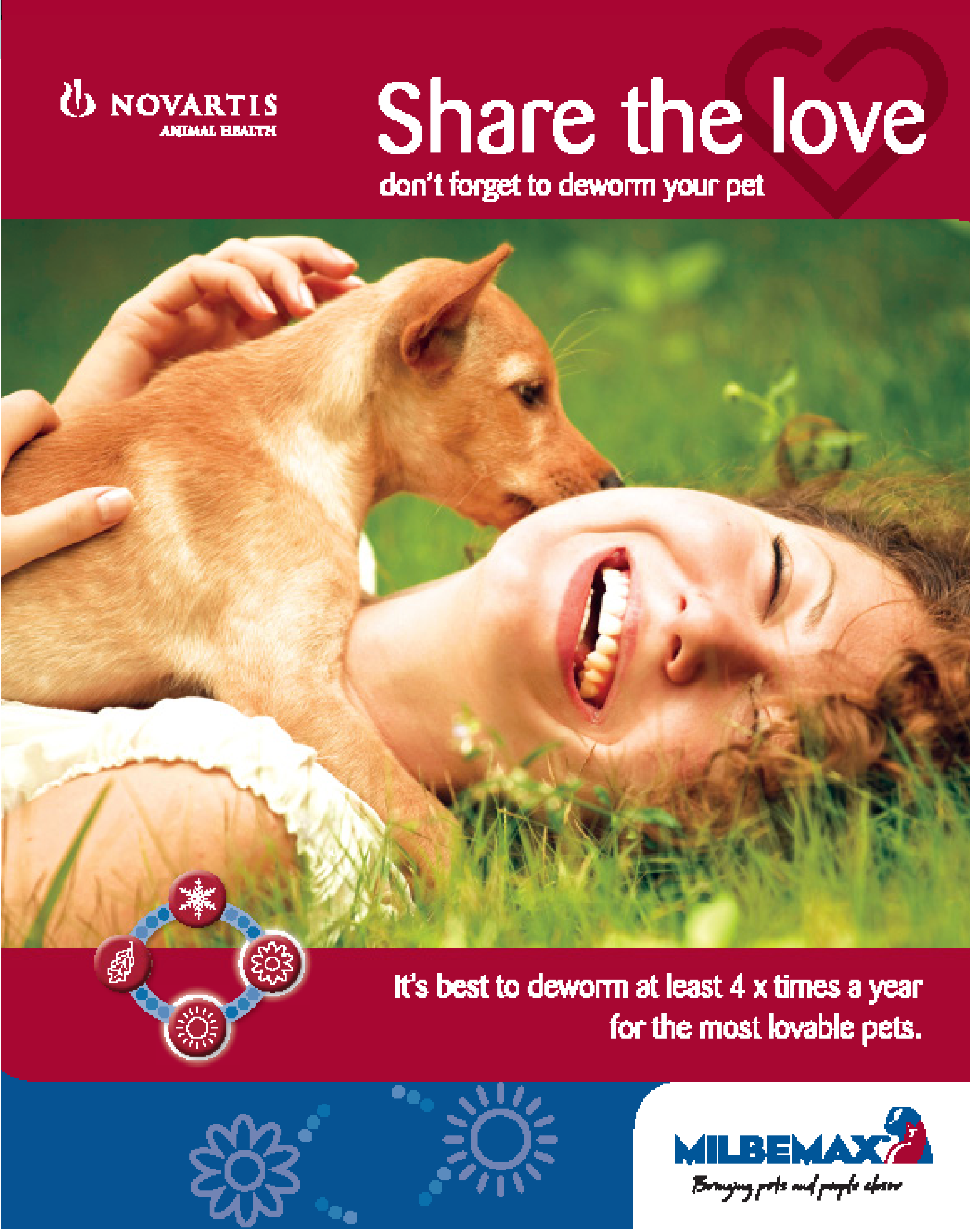
REFERENCES - available on www.vet360.vetlink.co.za

Comments on Scrotal Urethrostomy

Prof Louis Coetzee (BVSc, MMedVet (Surg)
Department of Companion Animal Clinical Studies, Faculty of Veterinary Sciences

All that I can ad to this article are the following:

1. We are currently using 5/0 Vicryl to attach the urethral mucosa to the scrotal skin with a continuous suture pattern - this method helps to prevent postoperative haemorrhage every time the dog is urinating.
2. It is recommended to apply white petroleum jelly (Vaseline) on the exposed urethra twice daily for the first 4 to 5 postoperative days to prevent drying out of the urethral mucosa and this also discourages the dog from licking the area.
3. It is imperative to keep the animal sedated with a phenothiazine derivative such as chlorpromaxine (Largactil) at 3mg/kg (PO) q12 for the first 10 to 12 days after surgery to prevent hypertension and scrotal skin haemorrhage as well.
4. The dog has to wear an Elizabethan collar (Buster plastic collar) during the first 14 days to prevent self mutilation of the scrotal urethrostomy.
5. The owner has to clean the skin on the medial thigh areas every day with Hibitane in water if there is any blood stains on the skin.
6. The dog must be kept away from any bitches in oestrus during the rehabilitation period (6 to 8 weeks after the operation).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Fibre Responsive Diarrhoea

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

Some patients presenting with diarrhoea can be managed with diets containing specific types and levels of fibre. Determining which diet is most effective is often trial and error.

The colon helps maintain fluid and electrolyte balance and absorb nutrients; it is also the major site of faecal storage until expulsion and provides an environment for microorganisms. Disruptions to normal colonic function lead to changes in both absorption and motility; clinically, this often manifests as large-bowel diarrhoea. The patients history will include clinical signs such as increased frequency, increased urgency, tenesmus, small volumes, frank blood (haematochezia) and mucous in the stool. Approximately one-third of dogs with a history of chronic diarrhoea have colitis.[5]

Chronic colitis is defined as inflammation of the colon that is present for ≥2 weeks. Inflammation of the colon reduces the amount of water and electrolytes absorbed and changes colonic motility by suppressing the normal colonic contractions that mix and knead and by stimulating giant migrating contractions (i.e. more powerful contractions that rapidly propel intestinal contents). Weight loss is uncommon. Clinical signs may wax and wane. Initially, the clinical signs may be sporadic, but progression usually occurs. Physical examination is unremarkable in most cases. A thorough rectal examination may reveal rectal polyps or malignant neoplasms that can mimic signs of chronic colitis.

The initial approach should include a complete history and physical examination, including rectal palpation and evaluation of faeces. Faecal smears for Giardia, faecal flotation for parasite identification (*Trichuris vulpis* in dogs,*Tritrichomonas foetus* in cats), and culture for bacteria (*Campylobacter*, *Salmonella, Clostridium*) are suggested in cases of chronic colitis. Rectal cytology is an important tool to exclude other causes of large-bowel diarrhoea. It can reveal inflammatory cells, neoplastic cells, and certain infectious organisms. A dietary trial is recommended before pursuing more advanced diagnostics.[5]

The most common fibre sources in pet foods contain a mixture of pectins, hemicellulose and cellulose and this mix is classed as moderately fermentable. Types of these fibre mixtures include rice bran, oat bran, wheat bran, soy fibres, soy hulls and beet pulp.

Fibre is generally categorised according to its solubility (ability to disperse in water) or fermentability (rate at which fibre can produce short chain fatty acids (SCFA)). Insoluble fibres (e.g. cellulose) increase faecal bulk and thus stimulate intestinal motility and decrease intestinal transit time. The addition of insoluble fibre may cause a decrease in nutrient digestibility and diets must be formulated to account for this.

Soluble fibres (e.g. pectins, gums) have a greater ability to absorb water and are more fermentable. Fibres that are rapidly fermented by the gastrointestinal bacteria can lead to borborygmus and flatulence. The important end products of fibre fermentation are the SCFAs, including acetic, butyric and propionic acids. Butyrate specifically, is the preferred energy source of the coloncytes, which derive more than 70% of their energy requirements from the luminally derived SCFAs. They also help maintain intestinal motility and ameliorate intestinal inflammation.[1]

There is a rapid turnover of the epithelial cells within the gastrointestinal tract and, therefore, high energy requirements. Dogs fed diets containing fermentable fibre have an increased colon weight, increased mucosal surface area and mucosal hypertrophy when compared to dogs fed diets containing non-fermentable fibre.[2] These changes indicate an increased absorptive potential, enhancing sodium absorption and maintaining normal intestinal electrolyte and fluid balance. Other beneficial effects of the production of SCFA include promoting the growth of indigenous microflora and inhibiting the proliferation of pathogenic microbes, acting as a prebiotic. Oligosaccharides are fermentable carbohydrates (often called prebiotics) which foster beneficial bacteria in the gastrointestinal tract.

Small intestinal bacterial overgrowth/antibiotic-responsive diarrhoea: The use of a fermentable fibre source is vital in cases of small intestinal bacterial overgrowth/antibiotic responsive diarrhoea to act as a prebiotic for the normal commensal bacterial populations. A healthy and balanced microbiota helps prevent the overgrowth of the pathogenic bacteria.

Fibre responsive large bowel diarrhoea (FRLBD): is presumed to be a stress-related disorder. Nervousness, abnormal personality factors, and stressors have been identified in approximately 40% of cases. Abdominal pain and vomiting can occur in some dogs. The condition may be concomitantly influenced by


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A range specifically designed to help manage fibre responsive conditions in cats and dogs.

other factors, including inflammatory disease, dietary indiscretions, pathogen overgrowth, parasitic infection and neoplasia. Some sufferers of CILBD can be fibre responsive, with fibre aiding in promoting colonic health. Dietary management is recommended in all mild cases, although medical management may be required alongside dietary management in chronic or severe cases.

There are several potential mechanisms by which dietary fibre supplementation may result in clinical improvement in dogs with FRLBD. Soluble fibre adsorbs a large quantity of water, improving faecal consistency. Colonic bacteria, which make up approximately 40-55% of the dry stool mass, ferment soluble fibre, which results in a vast increase in the numbers (but not types) of colonic bacteria and quantity of bacterial by-products. Insoluble fibre greatly adds to faecal volume. Thus, dietary fibre can increase faecal bulk which increases colonic distension, the major stimulus for normal colonic motility. With increased colonic distension, an improved motility pattern in dogs with FRLBD may result in resolution of clinical signs. In fact, dietary fibre has been shown to normalise colonic myo-electrical activity and colonic motility in people.

When the initiating cause of the colitis is unknown, dietary modifications can be very much a case of trial and error.[3]

Modifications to the diet include:

- high-fibre diets that normalise the transit time and bind faecal water and acts as a prebiotic.
- low-fibre, highly digestible diets that aid in reducing the quantity of undigested nutrients entering the colon.
- hypoallergenic diets, which can also be used when an intolerance or hypersensitivity is present.

Soluble fiber, psyllium hydrophilic mucilloid (Metamucil®, Procter & Gamble), added to a highly digestible diet has resulted in excellent or very good results in approximately 80% of dogs with chronic idiopathic large bowel diarrhoea. The median amount of Metamucil® added to the diet was two TBSP/day which was approximately 1.3 g psyllium/kg/day, starting lower and gradually increased until the stool consistency is normal.[4]

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Intensive Care Unit Seminar Clinical Techniques

24 & 25th October 2015
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24 OCTOBER 2015

07:00 Registration
08:00 Welcome **Kenneth Joubert**

Identifying & Treatment of Critical Illness

08:30 Looking for that dying patient in your hospital. **Kenneth Joubert**
09:15 Pathology of Sepsis, SIRS, MARS and CHAOS. **Samantha Currie**
10:00 Questions
10:15 Tea

Fluid therapy - how do we know if we are giving enough

10:45 Theorectical Considerations of Fluid in Critically Ill Patients **Kenneth Joubert**
11:30 Assessment of cardiovascular function and fluid load with ultrasound **Christelle Le Roux**
12:15 Questions
12:30 Lunch

Pulmonary Critical Care

13:30 Pulmonary Physiotherapy – How to mobilise secretions **Phillipa Riley**
14:15 Dealing with patients with respiratory distress **Joanne McLean**
15:00 Questions
15:15 Tea

Pain Control & Nutrition in Critical Illness

15:45 Opioids in critical care - when, what and where. **Roxanne Buck**
16:30 Nutrition for ICU patients **Chanelle Retief**
17:15 Questions

25 OCTOBER 2015

Abdominal Injury

08:00 Fast/T - Fast **Christelle Le Roux**
08:45 GDV's and other abdominal surgery **Ross Elliott**
09:30 Questions
09:45 Tea
10:15 Damage Control surgery – how to do it **Ross Elliott**
11:00 Renal Failure & Dialysis **Kenneth Joubert**
11:45 Questions
12:00 Lunch

Pathology & Toxicity

13:00 Foresensic Pathology - what to take and what to look for. **Samantha Currie**
13:45 Common toxicities and how to manage them **Joanne McLean**
15:30 Questions & Closing
15:45 Closing Tea

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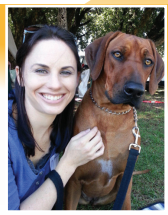
Ross Elliot BVSc, MMed Vet Surgery Small Animals

Chanelle Retief BSc Dietetics

Dr Joanne McLean BSc (Vet Biol) BVSc (Hons)



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