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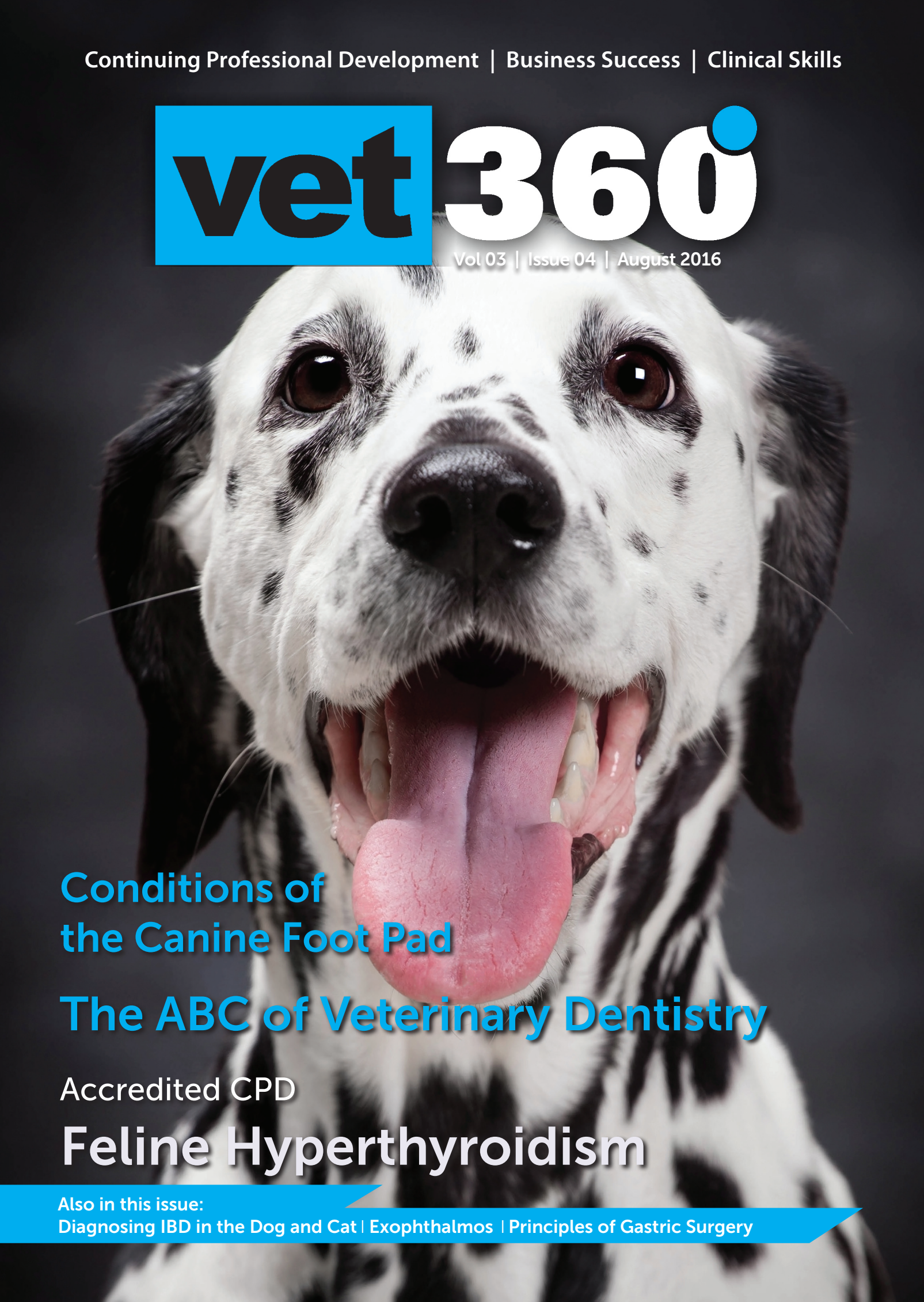

Conditions of the Canine Foot Pad

The ABC of Veterinary Dentistry

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Feline Hyperthyroidism

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Editor's Note

This edition is so full of material that we did not have space for a business article. Thank you to all our local specialists for their time, experience and advice.

The CPD article on Feline Hyperthyroidism is important as we are seeing more and more affected cats. Unfortunately we seem to have caught up with the rest of the world in this respect. Once again the devil is in the detail - not to over or underdiagnose cases which are marginal, based on laboratory testing.

We have started a series on dentistry and look forward to continuing it all the way through A to Z. Dentistry is really a burgeoning aspect of practice - both income-wise and in the provision of preventative animal health care. Practices would be remiss in not promoting it. Although not exhaustive - the author does seem to touch on the important aspects and pique ones' interest.

Lastly - thanks once again to out stalwart Dr Heidi Scroeder for her summary on footpad diseases in dogs - an often frustrating condition for us non-dermatologists.

I hope you enjoy the edition.

Regards

Liesel

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PREVIOUS EDITION: June 2016

- Breed Specific Hepatopathy in Scottish Terriers
- Gall Bladder Mucocoeles in the Dog
- Diagnosis of FIP in Cats

NEXT EDITION: October 2016

- Interdigital Cysts in Dogs
- Neck Pain in Dogs.
- What is PCR?

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The ABC's of Veterinary Dentistry: A to C

Mar 14, 2016
By Jan Bellows, DVM, DAVDC, DABVP, FAVD
DVM360 MAGAZINE

Our alphabet of 26 letters forms the foundation for billions of words. I've narrowed the list down to a mere 26 related to dental care in pets. I hope you enjoy this series of articles starting with A for dental anaesthesia and ending with Z for dental zebras (I'm still searching for a good Y dental concept if you have one please email me at dentalvet@aol.com). Each letter holds major importance in our quest to do the best for our veterinary patients.

A is for Anaesthesia

Most people recline in the dental chair, open their mouths and are OK with a suction tube placed under their tongues, probes placed into their sulci and radiograph sensors placed next to their teeth. But dogs and cats will not tolerate this without a fight — and who wants to fight? General anaesthesia is needed to thoroughly clean, polish and examine the teeth and oral cavities in our patients *(Fig 1)*.

Figure 1. A peek inside All Pets Dental, Dr. Bellow's veterinary practice in Weston, Florida. (Photos courtesy of Dr. Jan Bellows)

Unfortunately many of our clients are so worried about anaesthetising their dogs and cats that proper care is declined. How can you allay your clients' fears? Share with them the efforts you take to make anaesthesia as safe as possible by choosing the right patient for anaesthesia through preoperative testing, tailoring the anaesthetic protocol, and monitoring the effects of anaesthesia before and after the procedure.

Monitoring. Close patient monitoring is paramount during and after anaesthesia. (All photos courtesy of Dr. Jan Bellows)

The American College of Veterinary Anesthesiologists (ACVA) recommends monitoring for:

1. Circulation — Ensure that blood flow to tissues is adequate; measured via blood pressure.
2. Oxygenation — Ensure adequate oxygen concentration in the patient's arterial blood; measured via pulse oximetry.
3. Ventilation — Ensure that the patient's ventilation is adequately maintained; measured via capnography *(Fig 2A)*.
4. Temperature — Avoid hypothermia, which is common in anaesthetised dental patients and a source of trouble for perfusion and ventilation.

Figure 2A. An in-line capnograph.

The ACVA recommends having a trained veterinary technician at the patient's side. The technician can respond to feedback from the electronic monitoring system, use his or her hands-on clinical expertise to manage the patient's proper anaesthetic depth, and maintain an anaesthetic record of significant events and trends in monitored parameters. In our procedures, the technician relays information to the veterinarian, who is appraised of the patient's condition at least every five minutes throughout the procedure (Figure 2B).

How to monitor. Monitoring is accomplished through subjective methods (e.g. clinical appearance) and objective methods (e.g. electronic systems). During anaesthesia, the patient should have minimal jaw tone and no palpebral reflex. The femoral pulse should be palpable, and the perfusion time should be two sec-

Figure 2B. A dental technician monitoring, relaying and recording anaesthesia monitor findings.

onds or less. Breathing during balanced anaesthesia should be even and regular.

Electronic monitors may detect anaesthetic complications before being recognised by a trained clinician. In these situations, seconds count. Often, the advanced warning systems can head off problems before they become critical or do long-term damage. Electronic monitoring systems help to provide positive patient outcomes and reduce stress during the procedure.

Although many veterinary hospitals have accumulated a variety of monitoring devices, often single-parameter monitors, it would be wise to migrate to a 5-in-1 multiparameter monitor for all anaesthetic procedures (*Fig* 2C). An ideal monitor includes noninvasive blood pressure measurement, capnography, pulse oximetry, electrocardiography and temperature. The advantages of a multiparameter monitor over individual devices include:

Figure 2C. A cat monitored during anaesthesia with 5-in-1 parameter electric and apnea monitors.

1. More efficient for patient set-up and alarm management
2. Consistent operating menu
3. Ability to store, print and download data for all parameters
4. Economy of scale — less expensive than acquiring individual devices
5. Single point of contact for service, maintenance and troubleshooting

Be aware that choosing a monitor that has been designed specifically for use on animals can make a significant difference in performance and accuracy.

Local anaesthesia. Every oral procedure performed that may be painful should be accompanied by local anaesthesia. The two most commonly performed local anaesthetic blocks are the maxillary (*Fig* 2D) and mandibular (*Fig* 2E), which can be performed either intraorally or extraorally.

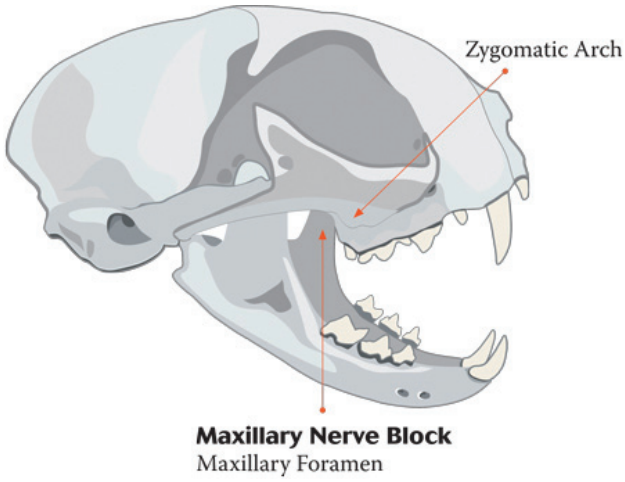

Figure 2D. Site for a maxillary nerve block. (Illustrations by Sathyanarayana)

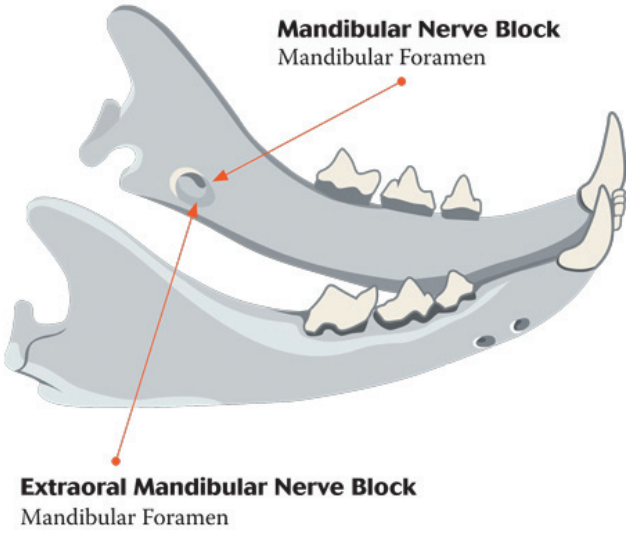

Figure 2E. Site for a mandibular nerve block.

Honourable mentions for A-analgesia, apical attached gingiva.

B and C are for the Basic Concepts of operative dentistry

Creating a dental treatment plan can be confusing and frustrating. As with other veterinary disciplines, dental diagnosis and care entails approximately one-third understanding of anatomy, physiology and dental principles; one-third recognition of disease; and one-third access to proper equipment and expertise to perform needed care.

Most dental problems can be treated by one of the eight options below:

1. Do nothing with the observed pathology other than future follow up. No immediate treatment is needed where there is a functional abnormality (i.e. even though the dentition is not normal, the animal is not experiencing adverse effects). One example of a functional abnormality is an enamel fracture that does not penetrate the dentin sufficiently to affect the pulp and where radiographs do not show pathology. Other cases where no treatment is the best course include functional malocclusion (Figure 3) and when the root of a tooth shows external resorption that does not extend into the oral cavity (*Fig* 4).

Figure 3. This left mandibular canine is malpositioned caudal to the maxillary canine but is not expected to cause a problem.

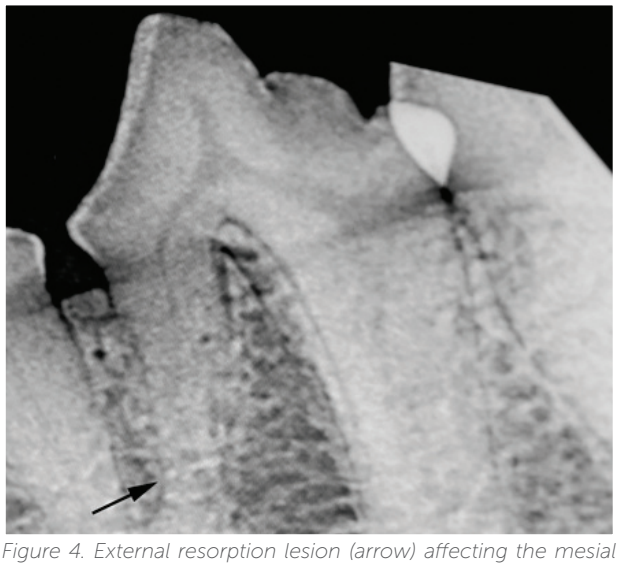

Figure 4. External resorption lesion (arrow) affecting the mesial root left mandibular fourth premolar. No intervention is necessary at this time.

2. Teeth cleaning, irrigation, polishing and application of professional plaque barrier gel or dental sealant. These measures are indicated in cases of stage 1 gingivitis (inflamed gingiva without evidence of support loss) and stage 2 nonpocket periodontal disease (less than 25 percent support loss) as evidenced by gingival recession.
3. Periodontal treatment
   - Local antimicrobial administration of clindamycin hydrochloride or doxycycline hyclate can be used to treat stage 1 bleeding on probing points (*Figs* 5A and 5B) and stage 2 (less than 25 percent of support loss) and stage 3 (25 to 50 percent support loss) periodontal disease when cleaned periodontal pockets (in contrast to gingival recession) are present. The pet owners

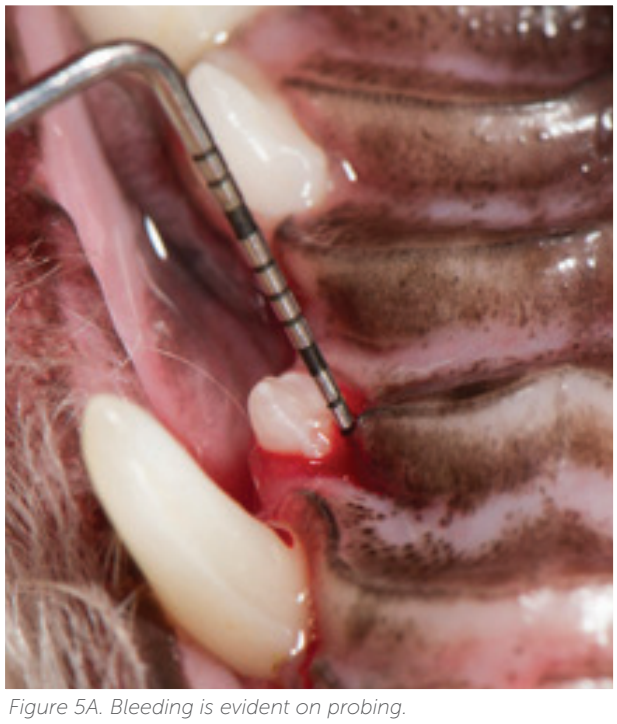

Figure 5A. Bleeding is evident on probing.

Figure 5B. Application of clindamycin hydrochloride.

can provide additional home care to control periodontal disease progression. *The periodontal gels Clindoral (TriLogic Pharma) and Doxirobe (Zoetis) are not available in South Africa, human equivalents will need to be used. Ed.*

- Surgery can save teeth if the tooth and patient are appropriate. Operculectomy (removal of the gingiva over a partially erupted tooth crown) is indicated in a young dog or cat (less than 8 months old) whose tooth is expected to fully erupt once the obstructing gingiva is excised (*Figs* 6A-6C). Open flap exposure for cleaning and débridement is used to expose a tooth root in selective cases where the periodontal pocket extends greater than 5 mm and the client is committed to save the pet's teeth despite a guarded prognosis.

4. Endodontic care
   - Vital pulp therapy can be performed when a complicated tooth fracture is acute (no longer than two days). The treatment usually results in a vital tooth with a good prognosis. If the fractured tooth with pulp exposure has been present for more than two days, extraction or

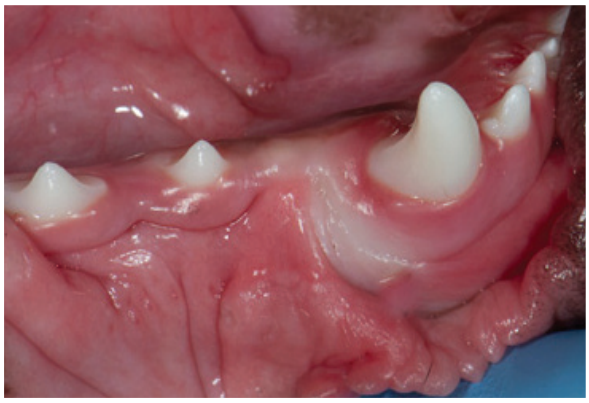

Figure 6A. A missing mandibular first premolar evident on oral examination.

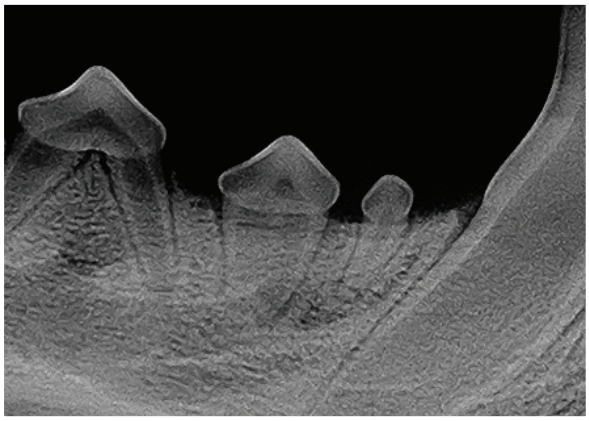

Figure 6B. The premolar is present on an intraoral radiograph.

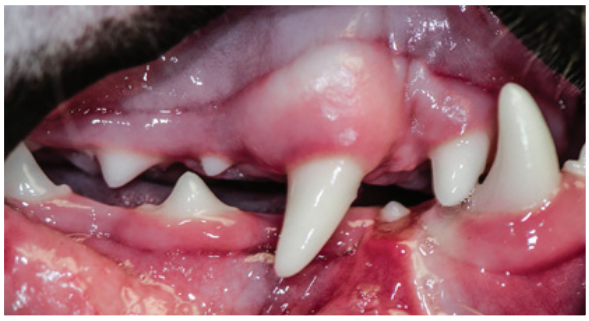

Figure 6C. After an operculectomy, the premolar erupts normally.

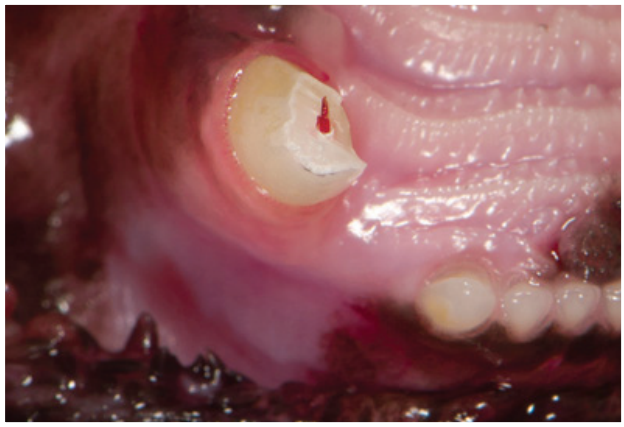

Figure 7A. A complicated left maxillary canine fracture in a cat.

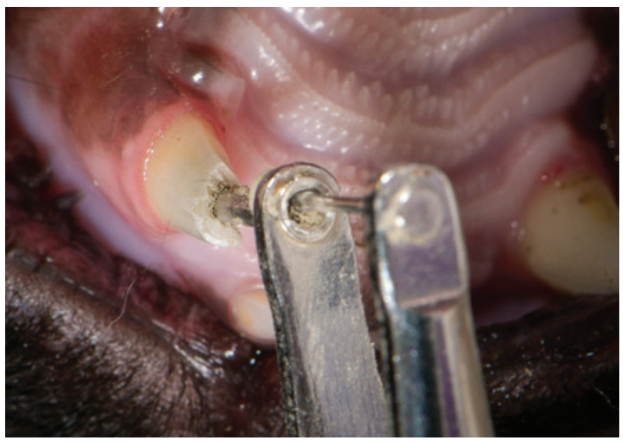

Figure 7B. Application of mineral trioxide aggregate (MTA) on vital pulp.

Figure 7C. The restored vital canine.

conventional root canal therapy should be performed with a more predictable outcome (*Figs* 7A-7C).

- Root canal therapy is the treatment of choice for end-stage pulp disease secondary to fracture, chronic pulpitis or caries. Ideal therapy depends on the animal's age, the age of pulp exposure, the tooth's condition and periapical health (Figures 8A and 8B).

5. Crown reduction with gingival closure. This intervention can be used to treat Type 2 tooth resorption with evidence of root replacement (*Figs* 9A-9D). Crown reduction and restoration are indicated for cases of malocclusion teeth interfering with the opposing gingiva.
6. Orthodontic intervention. Orthodontic buttons and elastics can be used to reposition teeth into functional occlusion and for maxillary or mandibular fracture stabilization. Inclined planes made from acrylic or metal can move mesioverted mandibular canines into functional positions (*Figs* 10A-10C).

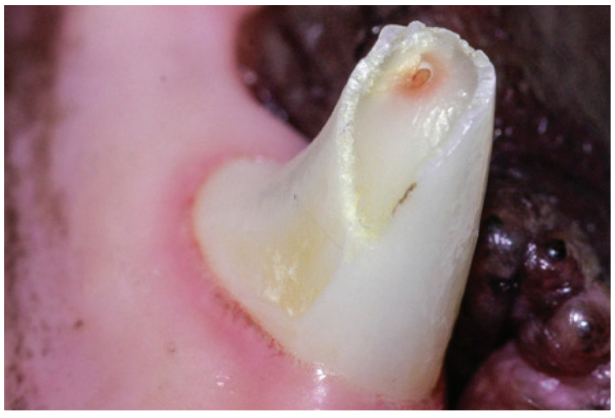

Figure 8A. A complicated crown fracture more than two days old.

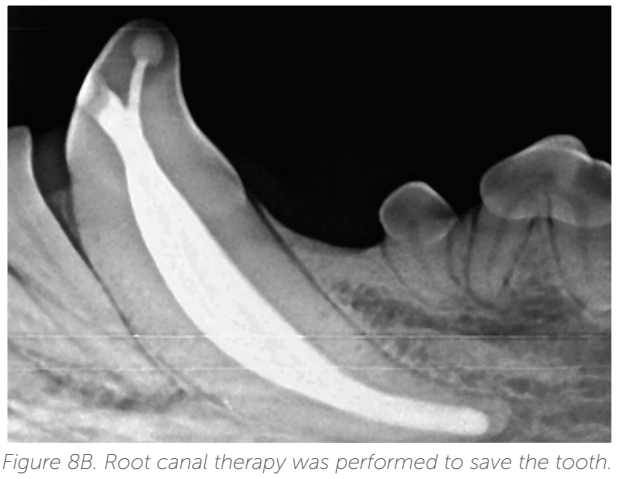

Figure 8B. Root canal therapy was performed to save the tooth.

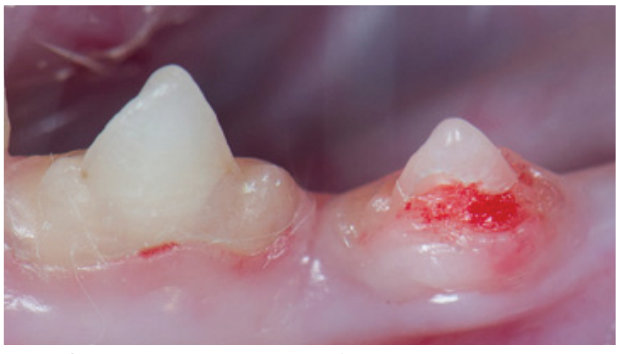

Figure 9A. Tooth resorption in a cat's mandibular third premolar.

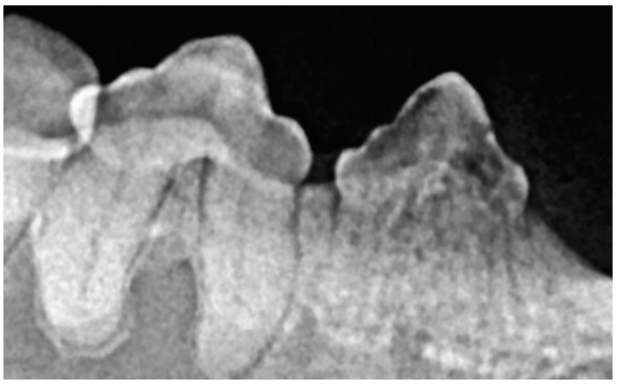

Figure 9B. A radiograph of the same tooth consistent with type 2 root replacement resorption.

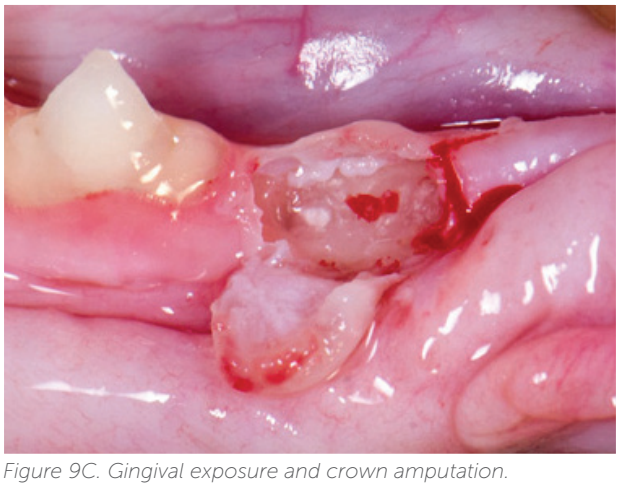

Figure 9C. Gingival exposure and crown amputation.

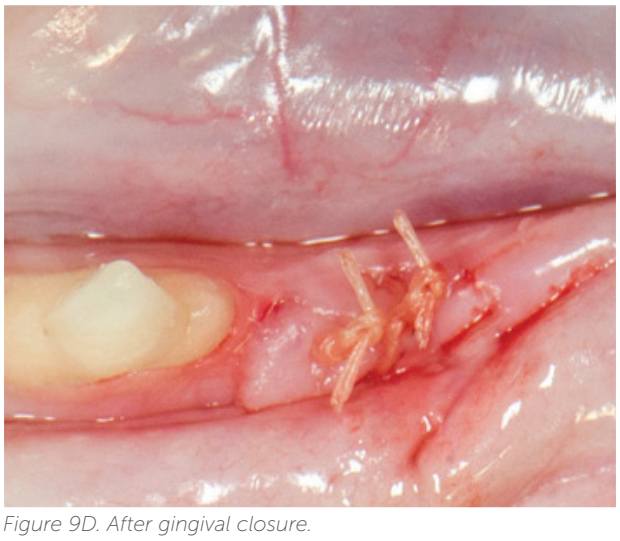

Figure 9D. After gingival closure.

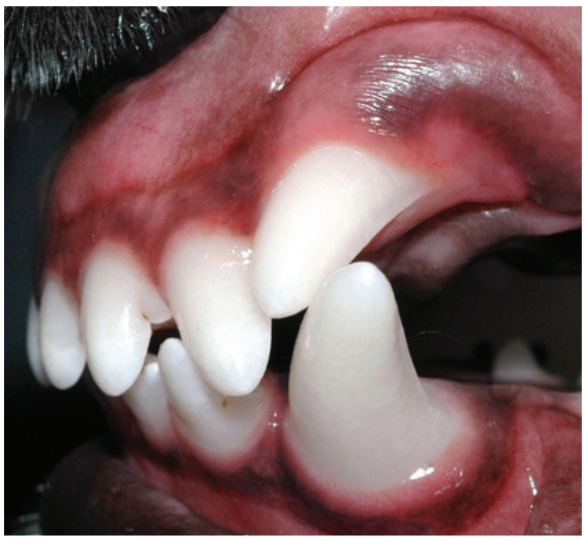

Figure 10A. A mesioverted maxillary canine in a Shetland sheepdog.

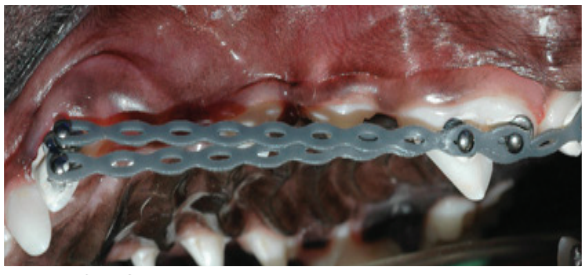

Figure 10B. Orthodontic buttons and elastics were used to pull the canine caudally.

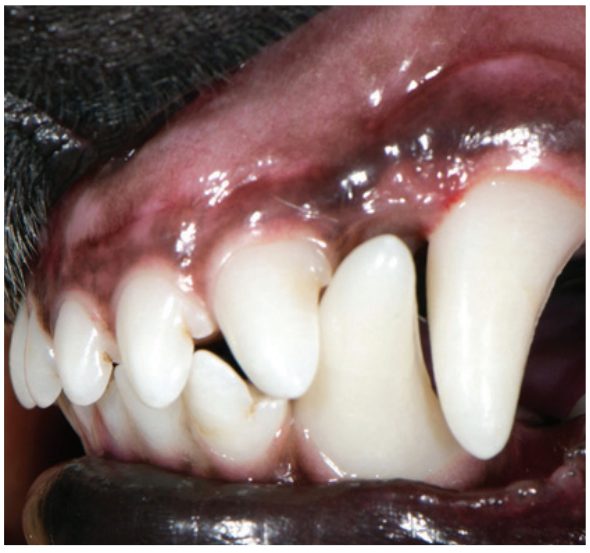

Figure 10C. A functional occlusion five months later.

7. Oral surgery. Surgery is the treatment of choice to care for many oral masses, both benign and malignant. When planning oral surgery, the goal is to achieve a 1-cm tumor-free margin for benign masses and a 2-cm or greater margin for malignant oral tumors.

8. Extraction
   - Tooth extraction is indicated when stage 4 periodontal disease is present (the tooth has more than 50 percent support loss based on probing depths, greater than stage 2 mobility or gingival recession that has progressed past the mucogingival line).
   - Extraction is the best therapy when the tooth has between 25 and 50 percent support loss and the owner or the patient will not allow appropriate allow home care.
   - Some fractured teeth are best extracted, especially those that have pulp exposure and stage 3 or 4 periodontal disease or marked internal resorption.
   - Extraction is indicated when root canal therapy is not a viable option due to the owner's wishes or the practice's capability (and lack of a referral option).
   - Feline teeth should be extracted via flap exposure when, in addition to the resorption, there is visible periodontal ligament and normal opacity on intraoral radiographs (Types 1 and 3).
   - Many cats affected by oropharyngeal inflammation that do not respond to home plaque control care benefit from the extraction of the teeth distal to the canines; those that still do not respond should have all the teeth extracted.
   - Extra (supernumerary) teeth that cause crowding, predisposing the normal teeth to periodontal disease, should be extracted.
   - Persistent primary (deciduous) teeth should be removed at the time of diagnosis to prevent the potentially harmful location of the adult teeth
   - Extraction is the treatment of choice for advanced caries.

Honourable mentions for B & C— bacteria, bupivacaine hydrochloride, bur, calculus, caries.

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References: 1. Pollmeier M, Toulemonde C, Fleishman C, Hanson PD. Clinical evaluation of firocoxib and carprofen for the treatment of dogs with osteoarthritis. Vet Rec. 2006;159(17):547-551. 2. Data on file at Merial.

Omeprazole is a Superior Gastric Acid Suppressant in Cats

Summarised by Dr Liesel van der Merwe BSVC MMed(Vet)Med Small Animals

Parkinson K, Tolbert K, Messenger A *et al.* Evaluation of the Effect of Orally Administered Acid Suppressants on Intragastric pH in Cats. Journal of Veterinary Internal Medicine. 2015(29) pp: 104 - 112

Why they did it?

Gastric acid suppressants including H2 receptor antagonists (H2RA) and proton pump inhibitors (PPIs) are the most widely prescribed medications for the adjunctive treatment of diseases which disrupt the gastric mucosal barrier in cats. Because of their small size and the fact that we are using human formulations - tablets need to be divided to achieve the correct dose. This study was designed to do 3 things: compare the effect of *per os* administered fractionated omeprazole tablets, reformulated omeprazole paste and famotidine on intra-gastric pH in cats, determine in which category of pH (0-1, 1-2, 2-3, 3-4, 4-5, 5-6, 6-7, 7-8) the pH was for the most time from days 4-7 of treatment and finally to evaluate the serum levels of omeprazole in the cats on bloods drawn on day 7 of treatment .

What they did:

Six healthy research colony cats with no evidence of GI disease were used. In a randomised open-label 4-way crossover design each cat was administered: placebo (250mg Lactose capsule *per os bid*), famotidine (0.88 – 1.26mg/kg *per os bid*), fractionated omeprazole tablets (0.88 – 1.26mg/kg *per os bid* or omeprazole reformulated paste (0.88 – 1.26mg/kg *per os bid*) for 7 days followed by a minimum 10 day washout period.

An omeprazole paste was reformulated using Gastroguard® to a suspension of 10mg/ml by mixing the paste with cod liver oil (1:39) and storing it at 7°C, away from light, to be used within 90 days.

On day 4 a Bravo™ pH capsule was fixed into the stomach endoscopically. Intra-gastric pH recordings were obtained telemetrically every 6 seconds for days 4 – 7 of treatment.

Blood samples were obtained from cats receiving omeprazole at 0.25, 0.5, 1, 2, 4, 6 and 8 hours after morning administration on day 7.

What they found:

The mean percentage time gastric pH is ≥3 and ≥4 has been found, using meta-analysis in humans , to be the ideal baseline for mucousal healing, with a goal in humans of being at this level for at least 75% and 67% of the day respectively. The table below summarises results for this parameter.

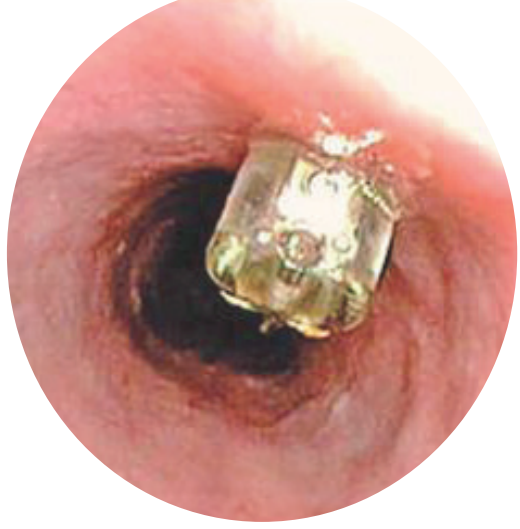
Both omeprazole formulations, dosed bid, significantly increased intra-gastric pH compared to either the famotidine or the placebo ($P<0.0001$). No significant differences were found between the fractionated or reformulated omeprazole comparing the above parameter.

The area under the concentration –time curve (AUC), median and range has been shown to best reflect the inhibitory effects of omeprazole on gastric acid secretion and was similar between both formulations. The Tmax (time to maximum serum levels) was earlier in the paste formulation

Take home message:

These results suggest that either of the omeprazole formulations, dosed twice daily, provide significantly superior acid suppression in cats compared to famotidine (H2RA) or placebo. The fractionated enteric coated omeprazole tablet remains efficacious despite disruption of its enteric coating, and shows no significant difference to the reformulated omeprazole paste.

Treatment	Placebo *bid*	Ranitidine *bid*	Omeprazole *oid*	Omeprazole *bid*
% time intra-gastric pH ≥ 3	16. ± 14.2%	42.7 ± 18.6%	68.4 ±35%	73.89 ± 23.2%
% time intra-gastric pH ≥ 4	9.6 ± 10.1%	22.4 ± 14.7%	57.8 ± 37.1%	55.7 ± 25.3%



The Efficacy of Ranitidine and Once-Daily or Twice-Daily **Orally** Administered Omeprazole on Intragastric pH in Cats

Summarised by Dr Liesel van der Merwe BSVC MMed(Vet)Med Small Animals

Sutalo, S, Ruetten M *et al*, 2015. The Effect of Orally Administered Ranitidine and Once-Daily or Twice- Daily Orally Administered Omeprazole on Intragastric pH in Cats. Journal of Veterinary Internal Medicine (29): 840 – 846

Why they did it?

The clinical efficacy of gastric acid anti-secretory drugs is largely unknown. Acid suppressant medications are however commonly used in cats clinical practice at dosages extrapolated from studies in dogs. The goals of the study were to determine normal gastric acid profiles in healthy cats, to investigate the effect of omeprazole and ranitidine on intragastric pH in a placebo controlled study and finally to compare once daily and twice daily dosage regimens of omeprazole in cats.

What they did?

The study was a randomised crossover design using a placebo on 8 healthy European shorthair olony cats . All cats underwent a gastroduodenal endoscopy and biopsy procedure prior to inclusion. Treatments were administered per os for 7 consecutive days. Washout time was a median of 12 days (7 – 24 days). All drugs were administered in gelatin capsules (which were evaluated and shown to dissolve within 5 minutes in a range of pHs') which were fed with a small amount of food (shown not to affect absorption). The treatments were-: placebo (empty gelatin capsule) bid, ranitidine at 1.5 -2.3 (median 1.9)mg/kg bid, omeprazole at 1.1 -1.3mg/kg (median 1.2)mg/kg oid and omeprazole at 1.1 -1.3mg/kg (median 1.2)mg/kg bid.

(NOTE: one enteric coated granule from the capsules contains 1.1mg of omeprazole – so cats were given 1 granule / kg body weight to approximate 1 mg/kg dose. Used in paediatric patients like this as well. The journal scan of the article by Parkinson *et al* in this issue shows that subdividing the enteric coated tablets is also effective)

Gastric pH was measured using a capsule (Bravo™ system) fixed to the gastric mucosa. The capsule was placed endoscopically on day 4 of treatment. The pH was telemetrically monitored at 6 sec sampling intervals for 4 days.

Three factors were compared: the percentage of time intra-gastric pH was ≥ 3 and also ≥ 4 from days 4 -7 of treatment, in which category of pH (0-1, 1-2, 2-3, 3-4, 4-5, 5-6, 6-7, 7-8) the pH was for the most time from days 4-7 of treatment and finally the adverse effects to the cat with regards to defaecation frequency and faecal scores as soft faeces occur in humans and dogs with per os administered omeprazole.

What they found?

Gross gastroduodenal evaluation was normal in all eight cats, but on histopathology all the cats had severe colonisation of the mucousal surface with Helicobacter spp. Small clusters of neutrophils were observed in the lamina propria of all the cats. The final histologic diagnosis was gastric colonisation by Helicobacter spp. The prevalence of colonisation by Helicobacter spp without concurrent gastritis is high in cats, so the findings were considered typical of what is seen in cats

A total of 745580 pH points were measured (94.7%). The table reflects the findings at intra-gastric pH ≥ 3 and ≥4. Intra-gastric pH varied widely across all pH categories but twice daily omeprazole treatment groups spent the most time in intra-gastric pH categories 3-4 to 6-7. There was no difference between the groups on faecal scores.

There was a large degree of individual variation in response to omeprazole , and much less to ranitidine. One cat had almost 100% intra-gastric acid suppression on bid omeprazole whereas another on had only about 3%. This occurs in humans as well and non-responders are thought to have a defect in the p-450 cytochome metabolism. (author - so test case numbers could ideally have been higher)

Take home message

A significant increase in the gastric pH on days 4-7 was only seen in the cats treated with bid omeprazole. The effects of ranitidine and the placebo on intra-gastric pH did not differ in this study. Twice a day administration of omeprazole at 1mg/kg appears to be the treatment of choice for cats with acid related gastrointestinal disease.

Treatment	Placebo bid	Ranitidine bid	Omeprazole oid	Omeprazole bid
% time intra-gastric pH ≥ 3	9.4 ± 8%	16.5 ± 9%	24.4 ±22.8%	67.0 ± 24.0% *
% time intra-gastric pH ≥ 4	7.0 ± 6.6%	9.6 ± 5.9%	16.8 ± 19.3%	54.6 ± 26.4%**

**p=0.011 ** p =0.044*

Feline Hyperthyroidism

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Feline hyperthyroidism is a multisystemic disorder arising from excess production of the active thyroid hormones (triiodothyronine [T3] and/or thyroxine [T4]) from an abnormally functioning thyroid gland. Approximately 97- 99% of cases result from benign nodular hyperplasia, adenomatous hyperplasia or adenoma of the thyroid gland.

The autonomous secretion of T4 and T3 produces a negative feedback effect on the pituitary gland, suppressing the release of thyroid-stimulating hormone (TSH) such that any normal thyroid tissue atrophies. In 70% to 75% of cats with hyperthyroidism, both thyroid glands are affected. Only 1% - 3% of cases are caused by malignant thyroid carcinoma. Hyperthyroidism is seen mainly in middle-aged to older cats, with the age at presentation ranging from 4 to 23 years (mean of 13 years). Only 5% of hyperthyroid cats are younger than 10 years at time of diagnosis. There is no sex or breed predisposition, although Siamese and Himalayan (colour-point Persian) cats seem to be under-represented in some studies.

Incidence and Risk Factors

The worldwide incidence of feline hyperthyroidism has steadily increased since the early 1980s and it is now one of the more frequently diagnosed disorders in small animal practice. Despite an increase in incidence however, the exact cause of the disease is still unknown. A number of theories have been proposed involving factors related to diet (possibly including the presence of goitrogens, eating canned cat food, iodine content, or frequent food changes), environmental causes (possibly associated with cat litter, environmental toxins, pollution, or exposure to allergens), a genetic mutation, abnormal immune responses, or altered hormonal responses.

Several substantial epidemiological studies from the USA, UK, Hong Kong and New Zealand have revealed some contradictory results, but also some common risk factors.

These include an increased risk in indoor cats, female cats, cats in multi-cat households, cats with dental disease (independent of age), use of topical flea preparations and pesticides, use of cat litter (not linked to increased risk in indoor cats), consumption of certain flavours of canned foods (fish or liver and giblet flavour) and increased risk in non-pure bed cats.

Clinical signs

The clinical signs of hyperthyroidism vary in severity and cats diagnosed earlier these days and so often show less of the classic clinical signs. The disease is insidiously progressive and owners may consider signs, especially when mild, part of the "normal" aging process. Thus months may pass before veterinary care is sought.

General features of the disease include:

- Weight loss (one of the most common clinical signs and seen in more than 80% of cats with the disease)
- Hyperactivity
- Polyuria and polydipsia
- Unkempt hair coat
- Cardiovascular signs (very commonly seen) including tachycardia, heart murmurs, gallop rhythm, increased apical pulse, evidence of systemic hypertension (e.g. retinal detachment), left ventricular hypertrophy seen on echocardiography and evidence of congestive heart failure (e.g. dyspnoea, pulmonary crackles in cats with pulmonary oedema and/or pleural effusion)
- Palpable goitre
- Gastrointestinal signs such as polyphagia or anorexia (seen in apathetic hyperthyroidism), vomiting and less commonly diarrhoea
- Muscle weakness and atrophy (rarely seen)

Cats showing depression, lethargy and reduced appetite are referred to as apathetic hyperthyroid cases and have traditionally accounted for less than 10% of all hyperthyroid cases. Some of these cats are overweight rather than thin and most of them are suffering from either severe cardiac complications associated with their hyperthyroidism or have underlying neoplastic disease.

The overwhelming majority of hyperthyroid cats have a palpable goitre. Increasingly however, cats with 'cold' thyroid nodules have been identified (i.e. the presence of palpable thyroid nodules without detectable hyperthyroidism). Many of these cats are in fact suffering from subclinical hyperthyroidism. It is now thought that most hyperthyroid cats go through a 1-3 year period of subclinical hyperthyroidism before developing overt hyperthyroidism. During this period, the cat has a total plasma T4 concentration within the normal reference range in combination with persistently low levels of TSH (< 0.03 ng/ml). Many of the cats later go on to develop hyperthyroidism, so close monitoring of these patients is justified.

Diagnosis

As hyperthyroid cats are being diagnosed earlier, when showing fewer clinical signs than in the past, there has been a change in clinical emphasis. Instead of simply confirming a diagnosis in a cat presenting with the classical clinical signs the goal is now to diagnose hyperthyroidism in cats with few, if any, clinical signs or ruling out the disease in cats presenting with varied problems that may or may not be related to hyperthyroidism.

It is now thought that most cats with hyperthyroidism go through a 1-3 year period of subclinical hyperthyroidism before developing overt disease

General screening laboratory tests, diagnostic imaging (echocardiography, radiography) and electrocardiography may provide supportive evidence of hyperthyroidism or detail the extent of cardiac involvement. Screening laboratory tests are also useful in eliminating other diseases with similar clinical signs or in depicting concurrent disorders potentially masked by hyperthyroidism that may be important in treatment decisions and ultimate prognosis, such as the presence of chronic renal disease, diabetes mellitus or neoplasia.

Changes found on routine screening tests include:

- Elevated liver enzyme activities (alanine aminotransferase (ALT) and alkaline phosphatase (ALP). At least one of these liver enzymes is elevated in 90% of hyperthyroid cats.
- Leukocytosis, eosinopenia and erythrocytosis
- Mild hypokalaemia and hyperphosphataemia, in the absence of azotaemia, are also seen in a small number of affected cats
- Hyperthyroid cats have also been shown to be more vulnerable to bacterial lower urinary tract infections, with a prevalence of 12% in one study, so cystocentesis and urine culture is indicated. Urinalysis is also indicated as part of detailed screening for concurrent illnesses such as chronic kidney disease and diabetes mellitus.

In most cats, the diagnosis can be confirmed by measuring resting serum total thyroxine levels (tT4). Serum tT4 concentration includes both the protein-bound and free levels of T4 circulating in the blood and in most cats, hyperthyroidism can be diagnosed on the basis of high resting serum tT4 concentration (tT4 > 50nmol/L). Measurement of serum total T3 (tT3) alone is not usually recommended because it is less sensitive than measuring tT4 (about 30% of hyperthyroid cats have normal tT3 values). Occasionally normal resting serum tT4 concentrations are recorded for cats with hyperthyroidism. This could be due to within or between day variations in mildly affected animals or the effects of concurrent non-thyroidal illness.

If hyperthyroidism is suspected despite a normal, albeit high, tT4 concentration (tT4 30-50 nmol/L), there are a number of possible investigative options:

Retest the tT4

In cats in which overt, manageable underlying disease is identified, such concurrent disease should

be first managed before proceeding with further thyroid testing. Once concurrent disease is resolved, the "euthyroid sick effect is removed" and most hyperthyroid cats will develop a clearly high tT4, confirming the diagnosis. Conversely, in cats without any overt underlying disease, simply repeating the serum tT4 concentration after 2 weeks may be diagnostic if the tT4 is fluctuating in and out of the reference interval. In some cats with preclinical disease, it may take a number of weeks or even months for the serum tT4 concentrations to increase into the range diagnostic for hyperthyroidism.

Test serum free T4

In cats with mild hyperthyroidism and tT4 values within the upper third of the reference interval, serum free T4 (fT4) concentrations can also aid in diagnosis. Serum fT4 concentrations are more consistently elevated (less fluctuation) in hyperthyroid cats than are tT4 concentrations.

Although fT4 is more sensitive than tT4 for diagnosing hyperthyroidism, the test specificity for fT4 is poor, with up to 20% of sick (and some clinically normal) euthyroid cats having false-positive fT4 results. Caution is therefore advised in using serum measurements of fT4 as the sole diagnostic test for hyperthyroidism. As a thyroid function test, fT4 should always be interpreted with a corresponding tT4 measurement. A tT4 value within the upper third of the reference range (30-50 nmol/L), combined with a high fT4 concentration, is consistent with mild hyperthyroidism, whereas a low or low–normal tT4 with a high fT4 is usually associated with non-thyroidal illness.

Serum fT4 is currently measured by one of two methods: Radio-immuno assay (RIA) using kits designed for use in humans and a modified equilibrium dialysis (MED) technique. The MED technique is the most accurate method for determining serum fT4 concentrations but unfortunately is not available in South Africa.

Test serum cTSH

The use of canine TSH (cTSH) assays in cats has recently received attention in the diagnosis of feline hyperthyroidism. A reference range for cTSH of 0.03-0.15 ng/ml has been defined for older cats.

In cats with subclinical and occult hyperthyroidism, cTSH levels are low or undetectable. Therefore if tT4 levels are in the upper half of the reference range (>30 nmol/l) and cTSH levels are low or undetectable (< 0, 03 ng/ml), hyperthyroidism can be diagnosed.

A recent study evaluating the usefulness of cTSH as a diagnostic test for feline hyperthyroidism using thyroid scintigraphy as the gold standard, concluded that measurement of serum cTSH concentration is a very sensitive, but nonspecific, diagnostic test. Approximately 98% of hyperthyroid cats had serum cTSH concentrations that are suppressed below the limit of quantification (<0.03 ng/ml), but approximately 30% of the older euthyroid cats in that study also had undetectable serum cTSH concentrations.

The current commercial cTSH assay cannot accurately measure the very low concentrations needed to clearly distinguish between the low-normal serum cTSH concentrations found in some euthyroid cats from the truly low or totally suppressed concentrations found in most hyperthyroid cats. It was shown that combining serum cTSH with tT4 or fT4 concentrations lowered the test sensitivity of cTSH from 98 to 97%, but markedly increased overall test specificity (from 69.9 to 98.8%).

The conclusion reached was that testing in parallel by combining serum cTSH concentration with either tT4 or fT4 concentrations improved the ability to correctly differentiate hyperthyroid cats with occult or mild disease from euthyroid cats suspected of having thyroid disease, especially when serum concentrations of tT4, T3, or fT4 were within the upper limits of their reference interval or only marginally increased.

Dynamic thyriod tests

Other options for diagnosis of suspect cases include dynamic thyroid tests (T3 suppression, TSH/TRH stimulations tests) and thyroid imaging (scintigraphy, ultrasound and CT). Dynamic thyroid tests are not always straightforward to interpret and are much less frequently performed since they require multiple samples to be collected and may result in side-effects.

Thyriod Imaging

Scintigraphy is performed to determine whether there is increased activity in the thyroid glands relative to the activity in the salivary glands 20-60 minutes after intravenous or subcutaneous injection of pertechnetate. It is a very accurate and reproducible test used to differentiate between cats with unilateral or bilateral disease and to check for the presence of ectopic thyroid tissue not palpable in the neck. This is extremely helpful when planning surgery. However as it requires access to specialist facilities, it is not routinely available.

Ultrasonography has been used to document the dimensions and volume of the thyroid glands in euthyroid and hyperthyroid cats and has been shown to have 85.7% agreement with scintigraphy in defining normal and abnormal thyroid lobes. However, it is technically demanding and very operator dependent. CT has been used to determine the dimensions and volume of thyroid tissue in clinically normal cats;

however the value of such imaging in the diagnosis of hyperthyroidism remains undocumented. Although both CT and ultrasonography can provide information regarding thyroid volume and morphological information, including invasiveness and can guide sampling, they are unable to provide functional information and may not identify ectopic thyroid tissue or metastatic disease.

When investigating a cat for possible hyperthyroidism, it is also important to consider all possible differential diagnoses and to look for evidence of multiple interacting diseases. This is because hyperthyroidism is seen most commonly in older cats and this group of patients is often affected by more than one disorder. Diabetes mellitus, renal disease, malassimilation syndromes (including inflammatory bowel disease, early intestinal lymphosarcoma, pancreatitis, and exocrine pancreatic insufficiency), acromegaly, and hyperadrenocorticism are perhaps the most important differential diagnoses.

Treatment

Spontaneous remission of hyperthyroidism has not been reported and prevention is not possible as the aetiology still remains elusive. Because of the benign nature of the thyroid lesions, if treated appropriately, hyperthyroidism carries a favourable prognosis. Failure to institute therapy will result in insidious progression of clinical signs to emaciation, severe metabolic and cardiac dysfunction and ultimately death.

Treatment options include medical management with anti-thyroid drugs, surgical thyroidectomy, thyroid ablation using radioactive iodine (the gold standard in human medicine) or most recently, feeding an iodine-restricted diet. Each treatment option has its own advantages and disadvantages. Selecting one therapy over the other depends on the age of the cat, severity of thyrotoxicosis, presence of concurrent illnesses, facilities available, potential complications, costs and the owner's lifestyle and willingness to accept the form of treatment advised. There are a few studies directly comparing the outcome of each of the treatment methods available.

One study of 167 hyperthyroid cats suggested that the median survival time (MST) in cats treated with radioactive iodine was significantly longer (4 years) compared with that of cats treated with anti-thyroid drug therapy alone (2 years). Owner/cat compliance and adverse drug reactions play a significant role in this shorter survival time. Pre-existing renal failure also adversely affects survival time irrespective of treatment type and results in a MST of about 6 months.

Before deciding which treatment to use, the cat should be assessed for concurrent disease, especially renal disease, systemic hypertension, and heart disease, all of which occur commonly in association with hyperthyroidism. It is particularly important to assess the cat's renal function (check blood urea nitrogen [BUN], creatinine concentrations, serum SDMA levels and urine specific gravity). This is because resolution of the hyperthyroid state often is associated with an increase in BUN and creatinine concentrations and a decrease in glomerular filtration rate (GFR) and effective renal blood flow. Because of this, some cats without prior evidence of renal insufficiency or with only mild renal impairment develop signs of uraemia after treatment for hyperthyroidism. To determine what effect resolving the hyperthyroid state may have on renal function, a short course of medical therapy is recommended before considering radiotherapy or surgery. Cats which show significant azotaemia should then be maintained on medical therapy and should not be considered suitable candidates for radiotherapy or surgery. Similarly, cats which develop significant azotaemia after radiotherapy or surgery should be given L-thyroxine to maintain a euthyroid or mild hyperthyroid state.

Interestingly in a recent study that compared the survival of cats that developed mild azotaemia (> 177 nmol/L) with cats that did not develop azotaemia following medical therapy, no difference was found between the two groups. These results suggest that the importance of the mild azotaemia that commonly develops after treatment for hyperthyroidism may have been overemphasized previously and does not change long-term outcomes in these cats. The same study however did find that in cats that were inadvertently over-treated and made hypothyroid, the development of azotaemia was of significance as it negatively affected prognosis.

Medical Management

Medical management entails the use of anti-thyroid drugs. It is used in cats for long term control and is advised prior to surgery to decrease the metabolic and cardiac complications associated with anaesthetising hyperthyroid cats and to control clinical signs in cats awaiting radioactive iodine therapy.

Drugs of the thiurylene class, mainly methimazole and carbimazole, are most frequently used for both the pre-operative and long term medical management of hyperthyroidism because of their consistent and potent effect in lowering thyroid hormone concentrations. Methimazole and carbimazole are antithyroid drugs that block T3 and T4 synthesis. They share the same mechanism of action; carbimazole is almost entirely broken down to methimazole in *vivo* (carbimazole, 5 mg, is broken down to methimazole, 3 mg). It usually takes 1 week of treatment to achieve a significant decrease in tT4 concentration.

The dose for both drugs is 2.5 to 5.0 mg per cat administered orally every 12 to 24 hours initially, increasing to every 8 to 12 hours as necessary, adjusted to maintain a euthyroid state, and given for the rest of the cat's life. The drugs are most effective given twice

or three times daily. The dose should initially be low (especially when renal insufficiency is suspected), and the renal values should be monitored as the dose is gradually increased. When cat and owner compliance is good, the successful response rate is approximately 85%.

Difficulty in administration causes many treatment failures. In an attempt to make medicating simpler, many clinicians are now using topical transdermal applications of methimazole, and initial studies seem to show promise. Advantages of medical therapy are numerous, including easy implementation, no requirements for special facilities, good availability and a reasonable cost to the client. Almost all cats are potential candidates and very few contra-indications, other than presence of thyroid carcinoma, exist.

The main disadvantages of medical therapy in this species, is the high degree of owner and patient compliance required and the rapid recurrence of signs if compliance is lost. In addition adverse and serious side effects occur in about 8-15% of cats treated with carbimazole or methimazole, which may require complete drug withdrawal. The most common is vomiting, anorexia and depression. Blood dyscrasias, facial excoriation, hepatotoxicity, renal decompensation, coagulation abnormalities, generalised lymphadenopathy and even acquired myasthenia gravis occur less commonly. These side effects normally occur within the first 3 months of treatment.

Other medical treatments available include the use of iopanoic acid and beta blockers. The results of studies evaluating the efficacy of cholecystographic agents such as iopanoic acid, in the treatment of feline hyperthyroidism have been disappointing and the drug is not recommended for long term management of the disease but may be suitable for short -term management prior to surgery or radioiodine therapy. Beta blockers such as propanolol and atenolol as well as potassium iodate have also been used successfully in pre-surgical stabilisation but longer term studies are required to evaluate their use for long-term medical management.

Control of hyperthyroidism by percutaneous injection of ethanol into solitary thyroid nodules as well as ultrasound-guided thyroid radio-heat ablation has been described in a small number of cats, but these techniques have been associated with serious side-effects and are unlikely to be a viable option for the majority of hyperthyroid cats.

Surgical Thyroidectomy

Surgical thyroidectomy is highly effective, almost always curative and widely available for cats, but the procedure can be associated with significant morbidity and mortality. Hyperthyroid cats also represent an anaesthetic risk due to the systemic effects of the disease. The most common serious post-surgical complications in cats include hypocalcaemia, due to hypoparathyroidism when the parathyroid glands are inadvertently removed, laryngeal paralysis, voice changes and Horner's syndrome. Treatment failure can occur due to inappropriate unilateral thyroidectomy where there is bilateral disease, incomplete removal of tissue or less commonly the presence of hyper-functional ectopic thyroid tissue. This can be avoided by performing thyriod scintingraphy pre-operatively.

Radioactive Iodine Treatment

Radioactive iodine treatment, the therapy of choice in human toxic nodular goiter, is also successfully used in the cat. It is simple, safe and effective and is possibly the best curative treatment for most hyperthyroid cats. Permanent hypothyroidism is rare as is recurrence after successful treatment and other side effects are minimal. The main disadvantages of such treatment are that it is potentially extremely hazardous to personnel, requires prolonged hospitalisation, is costly and is only available in a limited number of centres.

Iodine Restricted Diet

A novel option for treatment of feline hyperthyroidism now exists in the form of a commercially available iodine restricted diet (Hills Y/D®). An iodine restricted food was developed based on the hypothesis that feline hyperthyroidism can be managed nutritionally by limiting the amount of dietary iodine available for production of thyroid hormones. Feeding an iodine restricted food has been shown in multiple feeding trials over the past 10 years, in over 100 cats with naturally occurring disease, to decrease thyroid hormone concentration and alleviate clinical signs of hyperthyroidism.

Three additional studies documenting the safety and efficacy of iodine restricted food as the sole therapy of hyperthyroid cats showed that a food with iodine levels at or below 0.32 ppm per dry matter basis was able to convert 90% of the hyperthyroid cats in the study to a euthyroid state within 8-12 weeks. In all these studies biochemical features of renal function remained stable and no other biochemical abnormalities were observed. The only drawback in using an iodine restricted diet is that feeding compliance has to be 100% for the diet to have an effect. Any exposure to products or foods containing iodine, such as compounding agents for medications, treats, supplements, cleaning agents, other pet food, prey food or home prepared meals or treats and even in some cases municipal water, should be discontinued.

The diet has been shown not to have any adverse effects if fed to healthy cats and as such, the maintenance of normal thyroid concentrations and lack of clinical signs of hypothyroidism in these cats indicates that adequate amounts of iodine is provided to healthy animals fed the diet.

Prognosis

Without treatment, cats with hyperthyroidism usually die of concurrent renal disease, heart disease, liver disease, or systemic hypertension. With treatment, the prognosis varies from extremely good to guarded, dependent on the presence of heart disease, renal disease, and systemic hypertension; whether or not any systemic damage has become permanent before treatment of the hyperthyroidism; and which treatment options are available. On average, the mean life expectancy of treated cats is approximately 2 years.

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

Practitioners should be know which assay techniques are being used by their commercial or in-house laboratories.

Serum tT4 can now be measured in five different ways .

- Radioimmunoassay (RIA) has long been considered the gold standard but regulations regarding radioactivity, and the lack of automation have resulted in very few major commercial veterinary laboratories using it apart from diagnostic laboratories within veterinary university hospitals and research facilities.
- Chemiluminescent enzyme immunoassays (CEIAs) (eg, Immulite Total T4 assay and Canine Total T4 assay; Siemens Healthcare) utilise the same type of antibody testing as RIA; however, instead of measuring a radioactive isotope bound to the hormone, this method counts light emissions. The CEIA methodology, which has been validated for use in the cat. South African animal laboratories use this method. However, it is important to be aware that Immulite consists of more than one type of tTechnology: Immulite 1000 and 2000/XPi, and that there are both 'human' and canine versions of the Total T4 assay that can be used.
- A point-of-care enzyme-linked immunosorbent assay (ELISA) test kit is also commercially available for in-house use on feline seru and offers a cost-effective way to determine serum tT4 immediately in house.
- A homogeneous enzyme immunoassay (EIA) method for serum tT4 determination (eg, DRI hyroxine Assay; Microgenics) is now being used by many commercial veterinary laboratories. Like the CEIA and ELISA techniques, this EIA method has been validated for use in the cat and dog.
- Other chemiluminescent or enzyme immunoassay equipment (eg, Cobas; Roche Diagnostics, Architect System; Abbott Laboratories, ADVIA Centaur; Siemens Healthcare) can be used together with the appropriate human T4 kits. Most of these human kits have not been validated for use in cats or dogs. In addition, a major limitation of these human assays is the lack of sensitivity and poor performance, especially when attempting to measure the lower tT4 concentrations found in cats.

The correlation of serum tT4 concentrations provided by all of these assay methods is generally good, with the exception of the human T4 kits, which are never recommended, no assay has 100% test sensitivity and specificity. Compared with RIA (the gold standard), CEIA has been shown to provide very similar test results whereas the in-house ELISA methods appear less reliable and have been shown to overestimate the tT4 concentration When results of the two methods, used in 50 cats, were categorised (low, borderline low, normal, borderline high or high), they were discordant in up to 28 (56%) of the samples.

In samples from 100 cats, with a wide range of expected tT4 values, 60 with untreated hyperthyroidism and 40 which were being monitored after treatment with radioiodine, tT4 was measured both by EIA and CEIA. The results were compared and tT4 values obtained by each method were significantly correlated ($R = 0.97$; $P < 0.001$). But 11 cats with untreated hyperthyroidism had normal tT4 values when measured by EIA but had clearly high tT4 concentrations when measured by CEIA. Overall, when results of the two methods were categorised (ie, low, normal or high range), serum T4 values were discordant in 16 (24%) of the 66 cats. The bottom line is that no matter what assay method is used for tT4 measurement, there is the potential for false-negative and false-positive results. Therefore, serum tT4 results must always be interpreted in the light of the cat's history, clinical signs and other laboratory findings.

From: Mark E Peterson More than just T4: Diagnostic testing for hyperthyroidism in cats
Journal of Feline Medicine and Surgery (2013) 15, 765–777

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3. Diagnosis of Demodicosis in Dogs & Cats. Karen A. Moriello SMS code = a22801
2. A review of sterilisation practices and impact on the individual animal, in dogs and cats. Drs K de Cramer and K May SMS code = a26581
1. Rehabilitation of neurological patients. Dr Megan Kelly, SMS code = a97907

CPD Questions

AC/1534/16

1. Which one of the conditions listed below is a common cause of feline hyperthyroidism is INCORRECT?
   a. Benign nodular hyperplasia.
   b. Adenomatous hyperplasia.
   c. Thyroid adenocarcinoma.
   d. Unilateral thyroid adenoma.
   e. Bilateral thyroid adenoma.

2. Which one of the factors listed below is NOT considered one of the risk factors for feline hyperthyroidism?
   a. Indoor lifestyle.
   b. Pure bred cats
   c. Presence of dental disease.
   d. Cats where topical flea preparations were used.
   e. Cats exposed to cat litter.

3. Which one of the clinical signs mentioned below is NOT consistent with a diagnosis of apathetic hyperthyroidism in a cat?
   a. Depression
   b. Lethargy
   c. Reduced appetite
   d. Possible weight gain
   e. Bradycardia

4. Which one of the changes listed below is NOT one of the routine screening changes seen in hypothyroid cats.
   a. Elevated ALT and or ALP.
   b. Erythrocytosis.
   c. Mild hypokalaemia.
   d. Mild hyperphosphataemia.
   e. Mild azotaemia.

5. Which one of the following statements regarding feline hyperthyroidism is INCORRECT?
   a. Weight loss is one of the most common clinical signs.
   b. The majority of cats have a palpable goitre.
   c. Cardiovascular changes are less common than initially thought.
   d. Cats with nodules which are euthyroid are being increasingly identified.
   e. Cats go through a 1-3 year period of subclinical hyperthyroidism.

6. Which one of the following statements regarding the confirmatory diagnosis of feline hyperthyroidism is INCORRECT?
   a. A tT4 confirms the diagnosis in most cats - resting tT4 >50nmol/L
   b. A tT4 in the top normal reference range excludes a diagnosis of feline hyperthyroidism
   c. A tT3 is less sensitive than tT4 – 30% of affected cats will show false negative results
   d. A fT4 is more sensitive than tT4 for diagnosing hyperthyroidism in cats but has a poor test specificity – false positives in sick cats.
   e. A tT4 in the top normal reference range combined with a high fT4 is consistent with mild hyperthyroidism.

7. Which one of the following statements relating to cTSH in cats is INCORRECT?
   a. The cTSH assay is used in the measurement of feline TSH.
   b. In cats with subclinical hypothyroidism the cTSH are low or undetectable.
   c. Measurement of cTSH is very a sensitive but non-specific diagnostic test.
   d. In a study evaluating cTSH almost no euthyroid cats had suppressed cTSH levels
   e. The majority of hyperthyroid cats had very suppressed cTSH levels

8. Which one of the following statements concerning scintigraphy in cats with hyperthyroidism is INCORRECT?
   a. Scintigraphy compares the activity of the thyroid glands to that of the salivary glands.
   b. Scintigraphy is a very accurate and reproducible test.
   c. Scintigraphy can detect unilateral, bilateral and ectopic thyroid tissue involvement.
   d. Scintigraphy assists with surgical planning.
   e. Scintigraphy can easily cause the patient to develop hypothyroidism.

9. Which one of the following statements regarding the medical treatment of hyperthyroidism in cats is INCORRECT?
   a. Initial medical treatment is advised to stabilise the patient for surgery and general anaesthesia.
   b. Initial medical therapy is important to evaluate the effect of treatment on renal function.
   c. Cats which were azotaemic before initiation of medical treatment had no difference in outcome from those which were not.
   d. Cats which develop significant azotaemia after surgical treatment should be given L-thyroxine to maintain a euthyroid or mildly hyperthyroid state.
   e. Cats which develop mild azotaemia after reaching a euthyroid state had the same survival times as those who did not.

10. Which one of the following statements regarding the medical treatment of hyperthyroidism in cats is INCORRECT?
    a. Methimazole and carbimazole block T3 and T4 synthesis.
    b. It will take at least 4 weeks to achieve a significant decrease in tT4 levels.
    c. The drugs are most effective given 2 – 3 x / day.
    d. Side effects, if they occur, will only do so after prolonged use of the medication.
    e. The successful response rate is 85% if patient and owner compliance is good.

Diagnosing Inflammatory Bowel Disease In The Dog And Cat

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Canine and feline inflammatory bowel disease (IBD) is a chronic (> 3 weeks), idiopathic, inflammatory enteropathy. These idiopathic inflammatory changes may respond to immunosuppression but it is vital to rule out all known causes before calling the disease idiopathic.

Introduction and description

IBD is currently under intensive investigation. With evolving theories of pathogenesis, aetiologies and treatment, IBD will probably continue to be redefined. As an example, histopathological findings together with new technologies such as 16sRNA pyrosequencing and fluorescent *in situ* hybridisation (FISH) and candidate gene sequencing have helped clarify certain previously idiopathic diseases that were incorporated under the IBD umbrella. Granulomatous colitis (GC) and Histiocytic ulcerative colitis (HUC) seen in Boxers and French Bulldogs is now known to be highly associated with attached invasive *Escherichia coli* (AIEC); these cases have a long term response to fluoroquinolone antimicrobials and do not respond well to immunosuppressive therapy.

Classification

Traditionally IBD has been classified histologically according to the type of inflammatory cell infiltration and the site of inflammation, for example lymphoplasmacytic enteritis or granulomatous colitis. Secondary changes in the intestinal mucosa including, villous atrophy, fusion of villi, fibrosis, lacteal dilation and crypt changes are considered important in characterising the significance of these infiltrates. IBD can be clinically classified into food responsive enteropathies (FRE), antibiotic responsive enteropathies (ARE) and idiopathic IBD.

A GAME CHANGER

Granulomatous colitis (GC) and Histiocytic ulcerative colitis (HUC) seen in Boxers and French Bulldogs is now known to be highly associated with attached invasive *Escherichia coli* (AIEC); these cases have a long term response to fluoroquinolone antimicrobials and do not respond well to immunosuppressive therapy.

Clinical Signs

The clinical signs of (IBD) are similar to other chronic enteropathies (CE) and are determined by the site, extent, chronicity and severity of disease. Vomiting, for example, may reflect upper gastro-intestinal involvement; large volume diarrhoea, depression, melena, anorexia and ascites and other severe systemic signs may indicate small intestinal involvement; and small volume frequent stools with tenesmus and mucus or haematochezia may indicate colonic disease. Patients may also have a diffuse disease distribution.

The clinical signs may fluctuate in severity over time and overlap with many other organ diseases such as Addison's disease, hepatic and pancreatic diseases. Clinical signs are therefore broad spectrum and non-pathognomonic but usually strongly indicative of intestinal involvement.

Pathogenesis

The pathogenesis of IBD is unknown. Current theories include hypersensitivities as a result of immune system dysregulation and loss of tolerance to luminal antigens; pathological events increasing mucosal barrier permeability and exposing previously privileged innate immune system receptors to luminal commensals and dietary components; genetic mucousal innate immune system derangements predisposing the host to invasion by its own microbiota; loss of inflammatory cell apoptosis; food intolerance or other nutrition induced host microbiota dysbiosis and host microbiota mutations.

Diagnosis

Diagnosis of IBD requires that systematic elimination process is followed.

Clinical examination

A clinical examination always includes signalment, history, hands off examination and physical examination. It is used to localise the patient's disease to at least include the intestine, determines the chronicity (> 3 weeks), and determines the distribution and severity of the disease. The severity of the disease and the detection of extra-intestinal clinical signs determine the urgency and extent of diagnostic investigations and therapeutic intervention. For example severe IBD may cause protein losing enteropathy (PLE) with consequential loss of muscle condition score, body condition score, development of hypoproteinemia and cavity effusions; these patients need to be aggressively worked-up and treated. Other patients may have relatively mild clinical signs without weight loss or debilitation allowing a progressive methodical work up which may include empirical treatment for parasites, a therapeutic dietary intervention and an empirical antibiotic trial.

A minimum database for potential IBD patients

The first diagnostic challenge faced is ruling out primary intestinal pathogens, extra-intestinal disease, and concomitant diseases. A complete blood count (CBC), serum chemistry profile, urine analysis, and multiple faecal wet preparations and faecal flotations, are recommended in all cases to help rule out extra-intestinal disease and to help establish the severity of the intestinal disease.

An extended database selected for individual IBD patients

Faecal α1-protease inhibitor is useful to document PLE in untreated canine IBD cases. Faecal culture may be useful for the detection of salmonellosis or campylobacteriosis in some severe cases; cultured *E.coli* is not significant unless they are determined to be attached invasive type, which can only be determined with highly specialised techniques.

Evaluation of serum cobalamin levels may be useful in cats and dogs with IBD to help guide replacement therapy; it is also low in cats with lymphoma and pancreatic disease. Clinically significant hypocalcaemia and hypomagnesemia has been reported in cases of PLE in Yorkshire Terriers. Canine and feline pancreatic lipase immunoassay may be useful to help rule out pancreatitis in selected cases. A canine trypsin-like immunoassay (TLI) is used to rule out exocrine pancreatic insufficiency (EPI), especially in German Shepherd dogs (GSDs). Dogs with concurrent EPI and IBD have severe weight loss and may exhibit coprophagia and an increased appetite in addition to typical IBD signs. Thyroid hormone levels and FIV and FeLV status should be known in cats.

Radiography and ultrasonography are useful to detect extra-intestinal disease, intestinal obstruction as well as to help define intestinal disease. As the work up evolves, endoscopy or laparoscopy/laparotomy procedures are conducted. In severe cases they are ordered immediately.

Stepwise work up of non-critical patients with symptoms of IBD

What follows is a step wise approach that attempts to encompass the many clinical variations seen in practice.

1. Rule out intestinal helminths and protozoal infections

It is considered standard practice to thoroughly screen all dogs and cats with a CE for helminths and protozoa multiple times (at least 3X) very early in the diagnostic work up. Faecal tests for giardia can be supplemented by a faecal ELISA that is highly sensitive and specific. It is also considered standard practice to empirically treat for helminths and protozoa even if the screening tests are negative.

Giardia has variable therapeutic sensitivities. Possible treatments are metronidazole, febantyl and fenbendazole. Success of therapy should be reassessed with appropriate tests. In cases resistant to treatment environment, therapeutic sensitivity and a genetic immunopathology which may predispose the patient to infection are considered. In these cases drug combinations are used together with a high fibre diet and environmental decontamination.

In cats especially, *Pentatrichomonas* spp and *Tritrichomonas* spp must also be considered. These parasites can be seen on faecal wet prep mounts, diagnosed on faecal polymer PCR or in the case of *Trichomonas* spp cultured from faeces using the bovine TTF culture medium. Tritrichomonas is treated with ronidazole. Spontaneous remissions do occur and an asymptomatic carrier state is recognised. Ronidazole resistance has also been encountered.

Other more obscure intestinal pathogens, for example *Cryptosporidium* spp, *Histoplasma* spp., *Toxoplasma* spp., *Mycobacteria* spp., Protothecosis and

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Pythiosis may only be diagnosed cytologically or on histopathology if the work up is pursued.

2. Rule out Food Responsive Enteropathy (FRE)

Before conducting a diet trial the severity of the disease should be established. A canine inflammatory bowel disease activity index (CIBDAI) or a canine chronic enteropathy clinical activity index (CCECAI) index in the dog and a feline inflammatory bowel disease activity index (FIBDAI) in the cat is established. Some authors advocate the addition of an albumin value and histological score to the CIBDAI activity index. Whilst this may be necessary in individual cases, and in research, the practicality of repeat endoscopic procedures in clinical practice is limited.

Interpretation of index score

The FIBDAI is not graded as it is in the dog and does not differentiate food responsive enteropathy (FRE) from idiopathic IBD cases. The numeric number is simply used as a reference for evaluating post treatment success which is set 75% of the original pre-treatment score. A good response to a therapeutic trial is seen as a CCECAI score reduction from mild

Table 1: The CIBDAI & CCECAI Indices

Parameter	Clinical Inflammatory Bowel Disease Activity Index (CIBDAI)	Canine Chronic Enteropathy Clinical Activity Index (CCECAI)
Attitude/Activity	0 – normal 1 – slightly decreased 2 – moderately decreased 3 – severely decreased	0 – normal 1 – slightly decreased 2 – moderately decreased 3 – severely decreased
Appetite	0 – normal 1 – slightly decreased 2 – moderately decreased 3 – severely decreased	0 – normal 1 – slightly decreased 2 – moderately decreased 3 – severely decreased
Vomiting	0 – normal 1 – mild (1x/wk) 2 – moderate (2-3x/wk) 3 – severe (>3x/wk)	0 – normal 1 – mild (1x/wk) 2 – moderate (2-3x/wk) 3 – severe (>3x/wk)
Stool consistency	0 – normal 1 – slightly soft 2 – very soft 3 – watery diarrhoea	0 – normal 1 – slightly soft 2 – very soft 3 – watery diarrhoea
Stool (or mucus/ blood) Frequency	0 – normal 1 – slightly increased (2-3x/d) 2 –moderately increased (4-5x/d) 3 – severely increased (>5x/d)	0 – normal 1 – slightly increased (2-3x/d) 2 –moderately increased (4-5x/d) 3 – severely increased (>5x/d)
Weight loss	0 – none 1 – mild (<5%) 2 – moderate (5 – 10%) 3 – severe (>10%)	0 – none 1 – mild (<5%) 2 – moderate (5 – 10%) 3 – severe (>10%)
Albumin levels		0 –albumin >20g/L 1 – albumin 15 – 19g/L 2 – albumin 12 – 14g/L 3 – albumin <12g/L
Ascites and peripheral oedema		0 – none 1 – mild 2 – moderate 3 – severe (possible pleural effusion)
Pruritis		0 - no pruritis 1 - occasional itching 2 – regular itching, stops if sleeping 3 – wakes up to scratch

Interpretation of index score

CIBDAI			
0 - 3	4 - 5	6 - 8	> 9 (18)
Clinically insignificant	Mild disease	Moderate disease	Severe disease

CCECAI				
0 - 3	4 – 5	6 - 8	9 - 11	> 12 (24)
Clinically insignificant	Mild disease	Moderate disease	Severe disease	Very severe disease

to moderate disease down to insignificant disease in dogs; and a FIBDAI score reduction from 5 to 9 down to 0 in cats. By definition a 75% reduction in FIBDAI score is considered positive.

A well conducted food diet trial will solve both dietary hypersensitivities and food intolerances; both of which can cause IBD symptoms. Most clinical trials see a high percentage of patients responding to diet. Canine patients with lower CIBDAI or CCECAI scores (<5) and clinical signs associated with diffuse lower intestine disease are more likely to respond to dietary therapy than those with very high (>5) CIBDAI or CCECAI scores. No such predictions can be made using the FIBDAI score in cats.

Dietary hypersensitivities are immunological reactions to dietary antigens and dietary intolerances are non-immunological reactions to perturbations within the diet itself. For this reason some animals will respond to highly digestible, fat restricted and variable fibre diets that do not have any antigenic modifications. Usually single protein diets, containing a highly digestible novel protein source or hydrolysed diets are used. Size and structure of dietary proteins appears to affect their potential for immunoreactivity.

The role of the immune system and host microbiota in dietary hypersensitivities are being investigated. Perinuclear anti-neutrophilic cytoplasmic antibodies (pANCA) were significantly higher in diet responders than idiopathic IBD patients in one study but the clinical usefulness has not been established. Skin reactions are usually only seen with dietary hypersensitivity.

In the case of hydrolysed diets, potential problems include palatability issues and molecule sizes that still evoke an immune response. A complete dietary history is necessary to select the protein and carbohydrate sources in an elimination diet to suit that patient. Dietary history forms are available on the WSAVA website. Immediate, Type I and II, as well as delayed, Type IV hypersensitivities are possible with dietary antigens.

Establishing cause and effect in these cases requires strict owner compliance. Response to diet may occur within 10 to 15 days or as long as 8 to 12 weeks. Canine diet responders have an excellent prognosis but complete responders should undergo a re-challenge after 3 months because most of these patients remain asymptomatic on a fresh supply of their original type of food. The reason for this is unknown but may be related to dietary intolerances or dietary induced changes in the resident microbiome of the patient rather than dietary hypersensitivities. Unfortunately some references document a poor response to diet in cats and others warn that many initial responders may relapse and go on to require immunosuppressive therapy. For a diet trial to work the patients must be eating; appetite stimulants may be helpful in some anorexic cases.

Dietary hypersensitivities are immunological reactions to dietary antigens and dietary intolerances are non-immunological reactions to perturbations within the diet itself.

3. Conduct an empirical antibiotic trial in non-responders to exclude Antibiotic Responsive Enteritis (ARE)

Gastrointestinal dysbiosis is the new term describing unhealthy disturbances in the intestinal microbiota replacing the old term of small intestinal bacterial overgrowth (SIBO). There is currently no way of adequately and convincingly diagnosing bacterial dysbiosis in a clinical situation short of a therapeutic trial; for this reason the condition is recognised as an antibiotic responsive enteropathy (ARE).

Antibiotics empirically used include amoxicillin, metronidazole, oxytetracyclines, sulfasalazine and tylosin. The choice of antibiotics depends on the clinician's bias. Metronidazole is often advocated but potential carcinogenic side effects of long-term administration are a concern. Tylosin is well tolerated long term and may also treat occult cryptosporidium infection in some cases, it is also one of the few antibiotics besides fluoroquinolones, with some literature based evidence rationalising its use. Tylosin has activity against gram-positive and gram-negative cocci, gram-positive rods, and mycoplasma; it is not effective against *Escherichia coli* and *Salmonella* spp.

It is therefore not used as targeted therapy against specific bacterial enteritis but rather to transiently change the intestinal microbiota possibly by promoting the growth of beneficial commensal bacteria while suppressing deleterious bacteria. Starting dose is 15 mg/kg PO BID mixed with the food or given via gelatine capsule.

Patients that do respond to antibiotics are categorised as having ARE. The use of fluoroquinolones is retained specifically for where AIEC are suspected. He duration of the antibiotic course is usually 1-2 weeks before patients are monitored for a relapse of clinical signs. Frequency and severity of relapses determine the need for continuous, intermittent regular or reactive only treatment.

The role of Helicobacter infection in IBD is not well defined. In one small study fluorescence in situ hybridisation techniques were used to diagnose the organism intraglandular in the epithelium, in the presence of gastritis. The same technique was used to show resolution after "triple therapy" and dietary therapy was instituted. Triple therapy is only recommended in those patients with clinical signs and histopathological evidence of gastritis in the presence of Helicobacter organisms.

4. Conduct a probiotic trial

In one study adding a probiotic cocktail to known FRE had no added benefit over diet therapy (Sauter *et al* 2006). In other studies a clear, safe benefit is suggested provided their use is continued (German *et al* 2010, Herstad *et al* 2010, Bybee *et al* 2011). It also may seem counterintuitive to give GI antibiotics with probiotics however clinical improvement is often seen when given in combination with each other.

In another study in non FRE relapsing chronic IBD cases probiotics (VSL#3 strains) significantly improved clinical and histological scores, decreased CD3+ T-cell infiltration, increased regulatory T-cell markers (FoxP3+ and TGF-β+) and normalised intestinal dysbiosis in dogs with IBD compared to a combination of prednisolone and metronidazole (Rossi *et al* 2014).

The dogs in this study treated with VSL#3 also showed significantly increased plasma citrulline indicating restoration of the mucosal barrier although this clinical parameter was not compared to the immunosuppressive treatment group (Dossin *et al* 2011). This finding is significant and warrants futher investigation. Probiotics are thought to have immunomodulatory effects and antimicrobial activities directed toward intestinal pathogens (Rath 2003). Recommendations are to use a product produced by a reputable veterinary company backed by research for at least 2 months.

5. Repeat an ultrasonographic examination

Ultrasonographic studies are used to document abdominal changes consistent with IBD, aid in endoscopic biopsy site selection and help to detect extra-intestinal disease and intestinal neoplasia. Hyperechoic mucosal striations are indicative of lacteal dilation in dogs.

Other common ultrasonographic findings associated with IBD include small intestinal wall thickening, hyperechoic mucosal speckles, abdominal effusion and mesenteric lymphadenopathy. Older cats with diffuse thickening of the muscularis layer of the intestine are more likely to have T-cell lymphoma than IBD. Lymphadenopathy is equally associated with both diseases. Fine needle aspiration can also be performed.

6. Conduct endoscopic and histopathological examinations

Endoscopy is increasingly the preferred method of obtaining samples in dogs as it is minimally invasive, widely available and allows direct visualisation of the gastrointestinal mucosa. Endoscopic examination, biopsy and histopathology, using the WSAVA gastroenterology standardisation group guidelines, are recommended for animals presenting with severe disease or those not responding to initial therapeutic trials. Inter-operator variation in endoscopic interpretation is reduced by the use of standardised endoscopy evaluation forms and standardised histopathological templates and criteria.

Plasma citrulline is considered a sensitive indicator of GIT permeability. (Dossin et al 2011)

Histopathology allows grading and typing of mucosal inflammation and secondary changes. Other causes of intestinal disease, including helicobacter gastritis, cryptosporidium enteritis and neoplasia, may also be diagnosed. Where possible the more difficult to reach ileum is also biopsied. The ileum consistently shows mucousal changes most commonly in most patients.

This is especially important in cats to avoid missing focal ileal disease and small cell lymphoma (SC-LSA). Some cases may require sampling of focal disease and submucosal diseases surgically - via laparoscopy or laparotomy as the diagnosis may be inconclusive in endoscopically derived samples.

Laparoscopy (laparotomy) is suggested as the preferred method of obtaining samples in the cat where early small cell intestinal lymphoma often requires differentiation from IBD, and commonly affected areas tend to be ileum and jejunum which are more difficult to sample endoscopically.

Despite this most cats with IBD are still biopsied endoscopically because it is minimally invasive, more available and cheaper and immunophenotypic staining and other tests can be used to help differentiate small cell lymphoma from IBD on endoscopic samples.

IBD has been traditionally classified according to the inflammatory cells predominantly infiltrating the intestinal mucosa and which part of the intestine is affected. Lymphocytic-plasmacytic enteritis is the most common type of IBD seen in small animals.

Other forms are varying populations of eosinophilic, neutrophilic, histiocytic (mainly PAS-positive macrophages) and granulomatous (mainly PAS-negative macrophages) mucousal infiltrates causing gastroenterocolitis in individual patients. Linking type of histological inflammation to various studies on the innate immune system and intestinal microbiota has led to some improvements been made in the treatment of IBD.

The reader is referred to the open publications available from the WSAVA gastroenterology standardisation group for a thorough discussion on the standardisation of endoscopic examinations, biopsy sampling techniques and histopathological interpretation of IBD cases; as well as standardised endoscopic and histopathology examination forms.

Other breed specific enteric disease are also reported in Boxers, French Bull Dogs, German Shepherd Dogs (GSDs), Shapei's, Yorkshire Terriers, Soft Coated

Wheaten Terrier's, Irish Setters, with their individual nuances.

Monitoring Response to Therapy

Follow up endoscopy and histopathology:
Although repeated IBD activity index scores are the most common way patients are monitored repeat endoscopic and histopathology studies have been regularly performed in academic/research studies. Improving IBD index scores is usually associated with an improved intestinal mucosal appearance endoscopically and improved intestinal mucosal integrity and, if specialised organism detecting techniques are used, with a decrease in mucosal adhesion and invasion of potential pathogens; but the actual lamina propria inflammatory cell numbers may not be decreased in these repeat biopsies.

One explanation for this may be decreased post event apoptosis of the resident lymphocyte populations another may be continued unresolved underlying perturbations.

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Remainder of references available on www.vet360.vetlink.co.za

Exophthalmos

An overview of common causes and treatment

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The purpose of this article is to describe the more common causes of exophthalmos and their treatment. The authors did not aim to supply a complete differential diagnosis list and also do not discuss the less common causes.

There is very specific terminology used in ophthalmology which describes the position of the globe or size of the globe.

These include:

- Buphthalmos: Enlargement of the eye due to glaucoma.
- Enophthalmos: Abnormal recession of the eye into the orbit.
- Exophthalmos: Abnormal protrusion of the eyeball.
- Phthisis bulbi: Shrunken or fibrotic globe as a result of ciliary body damage following trauma or internal eye infection or inflammation.
- Proptosis: Abnormal protrusion of the eyeball beyond the orbital rim. Usually associated with trauma.

When examining an animal with a prominent eye, there are a number of techniques/features which can be used to aid your diagnosis.

1. Measurement: Measure the corneal diameter and compare it to the opposite eye. This allows you to easily differentiate buphthalmos from exopothalmos.
2. Retropulsion: In cases of exophthalmos there will be more resistance to retropulsion of the globe.
3. Protrusion of the third eyelid: This occurs due to pressure on the fat pad at the base of the third eyelid. This clinical signs is uncommon in cases of buphthalmos but occurs frequently in exophthalmos.
4. Observing the patient: View the head by looking directly down the snout as well as from a dorsal position so as to appreciate any asymmetry if it is present (Fig 1).
5. Examine the oral and nasal cavities: The mouth should be examined as extension of oral pathology can affect the orbital region. Both nares should be examined for discharges as nasal neoplasia or fungal infections may erode the medial orbit wall and cause secondary ocular pathology.

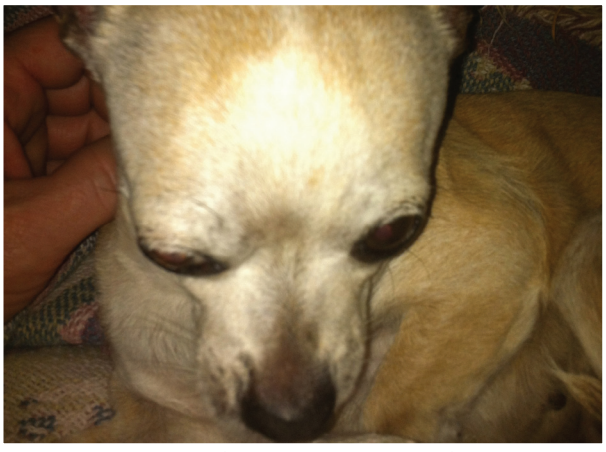

Figure 1: Asymmetry of the eyes as observed from above. The left eye is protruding

A complete and thorough history is imperative in cases of exophthalmos. Acute onset is usually related to retrobulbar abscess, trauma or foreign body whilst a slow progressive exophthalmos may be caused by cystic disease, proliferative disease or neoplasia. Orbital neoplasia is rare in animals less than 2 years of age.

Etiology of Exophthalmos

1. Physiological / anatomical exophthalmos

Brachycephalic breeds have a very shallow orbit which causes the globe to be more prominent. This condition is known as physiological exophthalmos. Due to this exophthalmos pigmentary keratitis and or corneal ulceration is commonly present in these patients. These breeds are also very susceptible to traumatic proptosis (Fig 2).

2. Traumatic exophthalmos

Traumatic exophthalmos can result from displacement of fractured bones and/or soft tissue swelling with haemorrahge. Most frequently this is a sequelae to blunt or penetrating trauma caused by motor vehicle accidents or collisions with stationary objects.

Figure 2: Traumatic proptosis in a Pekingese dog.

During the examination of the patient special emphasis should be placed on examination of the orbital margins. Fractures of the nasal and frontal sinuses can cause leakage of air causing orbital emphysema and exophthalmos. Radiology and ocular ultrasonography are valuable diagnostic tools for this form of exophthalmosis.

Treatment:
One should approach patients with orbital trauma with the following principles:
- Prevent additional swelling and haemorrhage.
- Prevent exposure of the cornea and conjunctiva.
- Prevent orbital infection.

These principles can be implemented by:
- keeping the animal confined and quiet.
- using analgesics or tranquilizers if indicated.
- applying cold packs to the orbital region.
- performing a temporary tarsorrhaphy.
- parenteral corticosteroids.
- broad spectrum antibiotics.

Systemic hyperosmotic agents and nonsteroidal anti-inflammatory drugs **should not** be used. The latter can be used once haemorrhage or severe bruising has stabilised.

3. Inflammatory exophthalmos

Inflammatory orbital disease can be caused by bacteria, fungi, parasites and non infectious causes such as eosinophilic myositis. The process usually begins as orbital cellulitis, which localises and, at a later stage, organises to form an abscess.

Agents causing inflammation may gain entrance to the orbit via;
- the haematogenous route.
- wounds due to external cranial trauma.
- penetrating wounds from the oral cavity into the retrobulbar space.
- infection in the adjacent paranasal sinuses and nasal cavity.
- sialoadenitis or abcesses of the zygomatic salivary gland.
- secondary rupture of the proximal nasolacrimal duct in cases of dacrocystorhinitis.
- foreign bodies from the oral cavity or those penetrating the orbital adnexa or conjunctiva.

Clinical signs.
- Acute, usually unilateral, exophthalmos.
- Protrusion and congestion of the third eyelid.
- Decreased globe mobility.
- Serous to mucopurulent ocular discharge.
- Periocular pain.
- Severe pain on opening the mouth.
- Lethargy and fever are common and assist in indicating an inflammatory cause.
- An inflammatory leukogram may also be present.
- A normotensive globe.

The classic history is an acute onset of signs and reluctance to eat.

Ocular ultrasonography is a cost effective method to differentiate cellulitis from a drainable retrobulbar abcess and to determine the presence of a foreign body.

Treatment:
Broadspectrum systemic antibiotics [amoxicillin and clavulanic acid] as well as systemic non steroidal inflammatory drugs should be used. Topical ophthalmic lubricants should be used to protect the cornea from exposure keratitis.

Orbital abcesses may require drainage of the orbital space into the mouth.This is done by incising only the buccal mucosa directly behind the last molar on the affected side with a scalpel blade and then placing a curved haemostat into the hole and gently pushing it up into the retrobulbar region and opening the forceps points. Exudate should drain from the oral wound. Remove the haemostat whilst keeping the points open. By opening and closing the jaw more exudate can be "pumped" out of the wound.

4. Eosinophilic myositis

This is an inflammatory disease of unknown cause. An autoimmune aetiology is suspected as autoantibodies to the 2M muscle myofibres of the muscles of mastication have been identified in affected dogs. The condition occurrs predominently in German Shepherd Dogs, Weimeraner, Samoyed and Dobermans but is also seen in other breeds.

Clinically one sees recurrent signs of swelling of the masticatory muscles, protrusion of the third eyelid, dysphagia, enlarged mandibular lymphnodes and occassionally blindness (Fig 3). Concurrent fever and anorexia is common. This bilateral condition must be differentiated from orbital cellulitis or abscessation which is usually unilateral.

Diagnosis and treatment:
Diagnosis is based on biopsy and histologic examina-

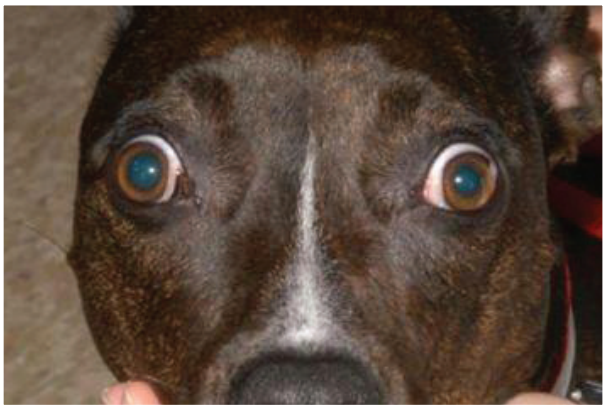

Figure 3: A dog showing bilateral exopthalmus due to myositis of the ocular muscles

tion of the temporal muscles. Peripheral eosinophilia is an inconsistent finding. Elevated creatine kinase may be seen during the acute phase. Uncontrolled myositis will eventually lead to atrophy of the affected muscles.

Treatment.
This involves the administration of corticosteroids [Prednisilone, 1-2mg/kg oid] for 7 days then gradually tapering the dose for a total of 4-6 weeks. Without treatment, inflammatory episodes run for 1-3 weeks.

5. Zygomatic mucocoeles / sialocoele:

The zygomatic salivary gland is situated in the rostral portion of the pterygopalatine fossa with the duct entering the mouth lateral to the last upper molar tooth behind the papilla of the parotid duct. Mucocoeles of this gland are usually associated with trauma causing leakage of saliva and secondary inflammation. Clinically these manifest themselves with exophthalmos, exposure keratitis and secondary corneal ulceration and swelling on temporal region of the orbit.

Diagnosis and treatment
Diagnosis is based on:

- Retrograde sialography - The oral ostium of the zygomatic salivary duct is located above the carnasial tooth. This is cannulated with a 24G Jelco cannula and contrast media is injected up the duct. Dorsoventral and lateral radiographs are taken. The extent of the mucocoele will be demonstrated by the contrast study.
- Fine needle aspirate of the buccal swelling and collection of a clear tenacious, golden fluid that forms strands.
- Digital pressure on the globe may cause saliva to ooze from the oral ostium of the duct.

Should the swelling persist the zygomatic salivary gland can be surgically removed via the lateral or dorsal approaches which require an orbitotomy.

6. Orbital neoplasia

Orbital and retrobulbar neoplasia can arise from epithelial, vascular, neural, bone, and connective tissues of normal orbital contents as primary tumours or extend secondarily into the orbit from the adjacent sinuses, nasal or cranial cavities as well as metastatically.

The most common primary malignant neoplasms in dogs are osteosarcomas and optic nerve meningioma and the secondary neoplasms are adenocarcinomas and malignant melanoma. Secondary orbital neoplasia in cats is more common than primary, with lymphosarcoma, osteosarcoma and malignant melanoma being the most prevalent.

Diagnosis and treatment:
The clinical picture is of a slow, unilateral, progressive exophthalmos with globe deviation. There is usually no pain when opening the mouth. Many of the other clinical signs of exophthalmos, mentioned before, will assist to confirm the diagnosis. Specialised diagnostic procedures can be used to make a definitive diagnosis. These include:

- Survey radiography
- Ocular ultrasonography.
- Ultrasound guided fine needle biopsy or aspiration for cytology.
- Magnetic Resonance Imaging [MRI] (Fig4).
- Computerized tomography [CT]
- Exploratory orbitotomy.

In some cases remission can be achieved with combined application of surgery, radiation or chemotherapy. Generally the prognosis is poor as the average survival time from diagnosis is less than 3 years. Exenteration or enucleation is the most widely performed treatment.

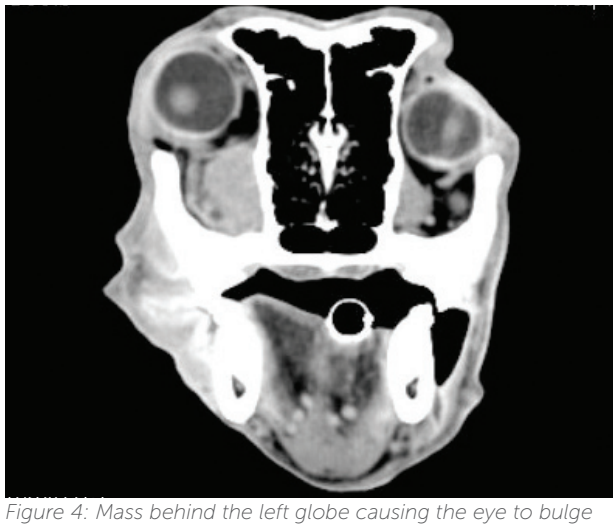

Figure 4: Mass behind the left globe causing the eye to bulge

Summary

Exopthalmos MUST be differentiated from buphthalmos. There are a number of possible causes and a proper quick diagnostic work up is essential as this is a sight threatening condition and in most cases vision can be preserved if the correct diagnosis is made and the appropriate treatment started.

Principles of Gastric Surgery in the Dog

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The stomach is generally very forgiving, however if taken lightly gastric surgery can have fatal consequences. The position of the stomach in the cranial abdomen makes exteriorisation difficult.

Anatomy

The stomach is divided into four areas, which are important to identify when performing gastro-intestinal surgery. The cardia is the region where the oesophagus blends into the stomach. The fundus is the large out-pouching of the stomach along the greater curvature. The body of the stomach lies between the fundus and the pylorus and forms the reservoir of the stomach. The pylorus is the distal third of the stomach or the outflow tract. The pylorus is divided into two parts the thin walled antrum of the pylorus and the thick muscular pyloric sphincter.

The greater omentum is attached at the greater curvature and the lesser omentum at the lesser curvature. The greater omentum forms the large leaf of omentum in the abdomen. The lesser omentum forms a part of the hepatogastric ligament.

Healing of the Stomach

Full thickness incisional healing of the stomach occurs rapidly. The stomach has an extensive blood supply making the regular phases of healing rapid. Collagen for repair is not only produced by fibroblasts but the smooth muscle cells of the stomach also contribute to collagen formation. The return of normal gastric strength after incisional trauma is about 14 days.

Surgical Preparation

Generally surgery of the stomach is an emergency and surgical preparation involves patient stabilisation rather than a nil per os period etc. These patients should have fluid and acid base and electrolytes abnormalities corrected prior to surgery.

In elective procedures food should ideally be withheld for 8 hours to allow for gastric emptying. Fasting for longer than 12 hours can actually have negative effects. Increased fasting duration leads to increased gastric acid decreased pH, which can lead to increased gastro-oesophageal reflux.

General Surgical Principles

Remember Halsted's principles of surgical technique minimise tissue trauma, practise precise haemostasis, preserve the blood supply, use aseptic technique, minimise tension on the tissue, use accurate tissue apposition and obliterate dead space. The stomach is generally very forgiving, however if taken lightly gastric surgery can have fatal consequences. The greatest risk is leakage of gastric content, which can lead to significant morbidity and mortality.

The position of the stomach in the cranial abdomen makes exteriorisation difficult. The supporting ligaments such as the hepato-gastric and hepato-duodenal ligaments can be carefully transected, however it is essential to identify the important structures such as the common bile duct that run in these structures to prevent damage.

The stomach is approached by a ventral midline celiotomy for most procedures. A midline incision of sufficient length is made to allow proper visualisation (no keyhole surgery!). Trying to save time by making a small incision will increase the risk of contamination and postoperative morbidity.

When handling the stomach it is ideal to minimise the use of instruments. Stay sutures are ideal in handling, stabilising and exteriorising the stomach and preventing abdominal contamination. Once the stay sutures have been placed, the proposed area for surgery should be packed off from the rest of the abdomen with moistened abdominal swabs. Put the moistened abdominal swabs down first to contact the serosa and then dry ones on top and adjacent to the bowel to be opened as needed to absorb contamination. Keep tissues moist to protect them and make handling less traumatic.

An avascular area midway between the greater and lesser curvature is the most common site for surgical procedures of the stomach. A stab incision is made in the stomach and a large incision is extended from the stab incision. Note that mucousal eversion is normal and occurs readily. Once surgery has been completed the stomach should be closed in a double layer closure. An inverting suture pattern such as a Cushing, Connell or Lembert should be used. These patterns should not however be used in surgery of the pylorus as they can decrease the outflow diameter of the pylorus. A simple continuous pattern is generally recommended for surgery in this area. If you're using stay sutures, make sure the incision does not have tension on it when you are closing the stomach because when you let go of the stay sutures your closure will be loose.

Only a monofilament absorbable suture material should be used in the stomach. Multi-filament materials have increased tissue drag and are not ideal for use in the delicate gastric tissues. The most commonly used monofilament materials are Polydiaxanone, Polyglyconate and Poliglecaprone 25. Polydiaxone is not ideal as it shows an increased degradation rate approximately 10 times normal in an acidic environment. Polyglyconate and Poliglecaprone show an initial increase in degradation rate but this stabilises and they are the materials of choice. Chromic catgut shows rapid degradation in gastric juices and should not be used.

Once the incision into the stomach has been closed all instruments and gloves should be changed. A new surgical set is opened and the abdomen closed. **Nothing that was used to open the stomach should be used to close the abdomen.** The abdomen should be lavaged with warmed ringers lactate. The ringer's lactate should be warmed by placing it in a hot water bath or incubator. Heating bags up in the microwave can lead to uneven heating of the fluid and burns to the patient

Specific Surgical Procedures

Gastric Biopsy

Most gastric disease can be diagnosed by a partial thickness biopsy taken via gastroscopy. However there are some diseases that require a full thickness biopsy, which has to be taken by a celiotomy. To perform this, a small gastrotomy is performed and a full thickness section of stomach is removed and sent for histopathological examination. The gastrotomy site is closed according to general principles.

Gastrotomy

A gastrotomy is performed to remove foreign bodies from the stomach and linear foreign bodies. It is performed via a midline celiotomy, and through the ventral surface of the stomach. An avascular area is selected between the greater and lesser curvature. Stay sutures should be used used and handled by an assistant to prevent contamination of the abdomen. Closely inspect the deep area of the fundus for foreign bodies as well as the pylorus and cardia. The site is closed as previously described. Always obtain postoperative radiographs to ensure that nothing has been left behind.

Partial Gastrectomy or Gastric Wall Invagination

These procedures are commonly performed to remove non-viable gastric tissue after gastric dilatation/volvulus. It incorporates many of the basic surgical principles such as stay sutures and packing off the abdomen to isolate the stomach and prevent contamination with gastric content. The big difficulty is deciding on which tissue is viable and non-viable. This is a very subjective assessment and multiple factors should be taken into account. The colour of the stomach, palpation of wall thickness, capillary refill time and peristalsis should all be taken into account when deciding on gastric wall viability.

There is no difference in outcome between performing a gastric invagination or a gastrectomy. A gastrectomy tends to take a bit longer and thus increase surgical time, however the benefit is that all the necrotic tissue is removed and will not be a cause of problems in the future if the animal survives. To perform a gastrectomy a zone between viable and non-viable stomach wall is selected, usually starting in the area just near the cardia. Stay sutures are placed and the non-viable section removed placing more stay sutures as you go. These are held by an assistant to prevent contamination of the abdomen with gastric content. The ventral and dorsal surface of the viable stomach is now sutured in a two-layer closure with an inverting pattern. Basic principles apply for the lavage and closure of the stomach and abdomen.

The gastric invagination procedure is quicker and thus making the surgical time shorter, however in patients that survive there is the risk of a large non-healing gastric ulcer in the portion of the stomach wall invaginated that requires long term treatment or repeat surgery. This procedure is performed by selecting the same zone between viable and non-viable tissue and then placing an inverting suture to invert the non-viable stomach wall into the lumen of the stomach. It is best to start at the cardia for ease of suturing. Basic principles apply for lavage and closure.

Gastropexy

Gastropexy is the gold standard in preventing gastric dilatation volvulus form occurring or recurring. A gastropexy by definition, is the formation of a permanent adhesion from the stomach to the adjacent body wall. The most common indication for a gastropexy is to prevent gastric dilatation volvulus (GDV), however it is used in the treatment of hiatal hernias.

Gastropexy for GDV is generally performed between the pylorus and the right body wall. There are a fair number of different techniques that can be performed. These are the circumcostal, belt loop, incisional, laparoscopic and tube gastropexy to name a few. The strength of the first two is nearly double that of an incisional. However these two take longer to perform and have increased associated discomfort and pain.

Another important factor is to make the incision in the stomach through the seromuscular layers leaving only the mucosa intact. It is essential not to penetrate the mucosa. This can lead to motility disorders abscessation and draining tracts. The incision in the body wall should penetrate the muscle layer to ensure adequate adhesion formation. The two incisions should be around 4-5 cm in length. The same principles apply to the other techniques for performing gastropexy. Release abdominal wall incision traction or close the Balfour retractor; as this will allow anatomical apposition of the two sites

For treatment of hiatal hernias a gastropexy of the fundus to the left body wall, an oesophagopexy and closure of the hiatus in the diaphragm are performed. Basic principles as for a pyloric gastropexy are applied here just a change in the position it is performed.

Pyloroplasty and Pyloromyotomy

Pyloroplasty is performed for alleviation of gastric outflow obstruction. The most commonly performed pyloroplasty performed is a Y-U pylorolasty. A full thickness Y shaped incision into the pylorus is made using basic principles. This is then closed in a U shaped closure, thus increasing the diameter of the outflow tract. A Heineke-Mikulicz Pyloroplasty is simple to perform but will not increase the outflow tract as much as a Y-U. This is performed by making a full thickness longitudinal incision in the pylorus. This is then closed in a horizontal closure. A simple interrupted or continuous suture should be performed and not an inverting suture as this can decrease outflow tract diameter.

A pyloromyotomy is simpler to perform but does not allow for inspection and taking of samples of the pylorus. It is performed by a partial thickness incision through the seromuscular layers leaving the mucosa intact. This is then left un-sutured to allow the pylorus to dilate. Its use is restricted to a handful of applications.

Post-Operative Care

It is advised to continue fluid therapy until the animal is eating and drinking. If there is a protracted period of anorexia expected post surgery then a feeding tube should be placed. An oesophagostomy tube is idea and simple to place after surgery. Oral intake should be initiated no longer than 7 hours post surgery. This is essential for healing of the intestinal system as eating stimulates stimulates peristalsis and aids in recovery.

There is no need for postoperative antibiotics in gastrointestinal surgery, unless there was an established peritonitis prior to surgical treatment. No antibiotic will stop a leak in the intestinal system or treat the effects - they will only delay its discovery. The recommended protocol is pre-operative antibiotics 30 minutes prior to surgery then every hour during surgery.

There is no evidence to justify continuing antibiotics after surgery. Any vomiting should be investigated as soon as possible as it can be an early sign of intestinal breakdown. In cases of vomiting it is indicated to l perform serial abdominal scans to check for free abdominal fluid and monitor the patients' temperature for pyrexia. Prokinetic agents are useful post operatively and will often stop the vomiting caused by ileus. In cases of gastric invagination use of long-term proton pump inhibitors and coating agents are indicated.

Shaping the future of animal health

Conditions of the Canine Foot Pad

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Foot pad conditions are usually part of generalised skin conditions or systemic conditions that also cause skin lesions and clinical signs elsewhere on or in a patient. The most important differential diagnoses are discussed in this article.

There are not many foot pad conditions and therefore when the footpads are affected, they can aid in the diagnosis of a particular condition. There are also a few conditions that affect the foot pads only.

When a patient is presented with a foot pad condition it is important to take a good history and do a full clinical examination. Consider the age (e.g. genodermatoses, parasites and infectious diseases in young dogs versus auto-immune and metabolic conditions in older dogs).

It is also important to determine whether there are concurrent systemic signs, e.g. necrolytic migratory erythema (NME), whether one or more pads are affected and whether there are signs of skin disease elsewhere on the body. Skin or foot pad biopsies for histopathology are required in many cases to make a final diagnosis.

HOOKWORM DERMATITIS: Hookworm dermatitis is caused by Ancylostoma caninum where infected larvae, present in soil or mud, in poorly kept kennels or highly contaminated environments, penetrate the skin and burrow into the hair follicles of thin skinned areas (axillae/inguinal regions/ventral abdomen), the foot pads and interdigital areas where they continue their life cycle. Young animals living in groups in unhygienic conditions and hunting dogs are highly predisposed.

The thin skinned areas commonly have papules, crusts and excoriations and the foot pads are usually oedematous, thickened and ulcerated. Paronychia and deformed nails may be present in chronic cases. Systemic signs such as diarrhoea, anaemia and weight loss are often present. Diagnosis is made with faecal flotation. Histopathology usually reveals eosinophilic perivascular dermatitis and sometimes larvae can be found. Prognosis is good if the affected dog is dewormed and removed from the contaminated environment.

DEMODICOSIS: Demodicosis is a differential diagnosis for every case of pododermatitis. Usually this is a generalised skin disease that can involve the paws but rarely if may affect the paws only and in very rare cases the foot pads as well. The feet become inflamed and may show hair loss, redness, swelling, crusting and scaling. The foot pads become crusty and thickened.

The diagnosis can be made by microscopic examination of hair plucks or skin scrapings from the paws, but biopsy and histopathology are required in most cases. Treatment with systemic and topical parasiticides for many weeks to months is usually required for successful management. Secondary bacterial infections are common and should be treated.

HARD PAD DISEASE:

There are 2 different types of hard pad disease: The first type is a crusting, thickened foot pad and is seen with e.g. pemphigus foliaceous, zinc responsive dermatosis, distemper infections and necrolytic migratory erythema. These foot pads may be painful and cause lameness.

Most commonly these cases will have other accompanying skin lesions and systemic signs, although rare cases of pemphigus foliaceus may present with foot pad lesions only. Biopsies are the most helpful diagnostic procedure. Treatment will depend on the underlying cause.

The second type is a foot pad with excessive fronds of normal appearing keratin. This is most obvious at the margins of the pads and represents overgrowth of the normal keratin mounds of the pads. Inflammation and exudate are not present. Lameness and pain are usually not detected. This condition appears more as if the pads grow abnormally fast and do not desquamate.

PEMPHIGUS FOLIACEUS: Pemphigus foliaceus is the most common of the pemphigus complex. It is characterized by a scaling, crusting and pustular dermatitis that usually affects the face, ear pinnae and foot pads. Some cases may become generalized. The foot pads are usually swollen and hyperkeratotic with scaling, crusting, hardening and fissuring. In severe cases there may be pustule formation, greenish/yellowish discoloration and significant epithelial loss of the foot pads. Cytological examination of the purulent material will reveal large numbers of neutrophils and/or eosinophils and acantholytic keratinocytes which are highly suggestive of pemphigus foliaceus. Bacteria are usually not present on cytology of intact pustules, but may contaminate ruptured lesions. There are usually not systemic signs, but lesions may be painful. The diagnosis is supported by cytology and should in all cases be confirmed by histopathology. This condition is treated with immunosuppressive dosages of prednisolone with or without azathioprine. Topical corticosteroid therapy may be useful for stubborn localised lesions.

Pemphigus foliaceus affecting the footpad (Photo: Dr Heidi Schroeder)

CUTANEOUS DRUG REACTIONS: Cutaneous drug reactions are adverse reactions following systemic and topical drug administration. The pathogenesis is complicated involving both immunological and non-immunological reactions. All types of hypersensitivity reactions have been reported. Clinical signs are highly variable. In most cases the clinical signs are mild (urticaria, erythroderma), but can also be very serious and sometimes fatal (erythema multiforme, toxic epidermal necrolysis and drug-induced pemphigus foliaceus).

Cases present with various lesions which may include papules, plaques, erosions and ulcers on various parts of the body, including the foot pads and pad margin. Drugs that have been reported to cause such reactions include antibacterial (sulphonamides, cephalosporins), antifungal (itraconazole, griseofulvin) and antiparasitic drugs (ivermectin, moxidectin), vaccines, topical drugs, hormones and tranquilisers.

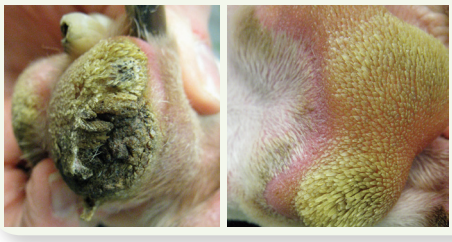
There is often a lag period of 5 to 21 days before clinical signs manifest. In most cases discontinuation of the medication and treatment of secondary infections, if present, results in resolution of the skin lesions. Some cases require more aggressive treatment e.g. glucocorticoids and other immunosuppressive drugs. Adverse effects can persist for 1 to 3 weeks after discontinuation of the offending medication.

FOOT PAD HYPERKERATOSIS:

Familial foot pad hyperkeratosis: Familial foot pad hyperkeratosis has been described in Irish setters, Kerry blue terriers and Labrador and Golden retrievers. Other breeds may be affected as well. Symptoms are usually present by 6 months of age. All foot pads are usually affected with crusting, fissures and pronounced compact keratin that in some cases can produce horns. These lesions can result in severe lameness and pain. Histopathology shows moderate to severe hyperplasia with marked papillated and diffuse hyperkeratosis. Treatment is discussed below. Affected dogs should not be bred.

Idiopathic nasodigital hyperkeratosis: This is an idiopathic hyperkeratosis characterised by hyperkeratosis of the foot pads with or without involvement of the nasal planum. Idiopathic nasodigital hyperkeratosis is seen in older dogs and characterised by increased horny tissue on the nose and/or foot pads. The nose/foot pads appear vegetative with projections of firm, feathered and cracked horny tissue on the nose and/or foot pads. Sometimes only the margins are affected but in severe cases the entire nose and/or foot pad is involved. Certain breeds, such as the Cocker spaniel, may be predisposed. Histologically, idiopathic nasodigital hyperkeratosis is characterized by epidermal hyperplasia and marked orthokeratotic to parakeratotic hyperkeratosis.

The diagnosis is generally based on history and clinical findings, and, in some cases, histopathology. There is no specific treatment for these conditions. Symptomatic treatment may be considered when the hyperkeratosis is a problem. Excess keratin may be removed with regular filing, scissors or a blade and fast growing nails should be trimmed regularly. Daily foot soaks in 50% propylene glycol may be of benefit. A topical corticosteroid-antibiotic cream may be used for fissured lesions. Owners should be warned of the potential "mess factor" associated with these topical therapies. Secondary bacterial infections should be treated where present. Oral Vitamin A therapy may be of benefit.



Idiopathic nasodigital hyperkeratosis of the footpads (http://foothills-animalhospital.blogspot.

CUTANEOUS VASCULITIS:

Cutaneous vasculitis involves inflammation of the blood vessel walls and perivascular connective tissue, followed by ischaemia and necrosis. This condition may be primary but is usually secondary to infections (bacteria, rickettsia, fungi), auto-immune disorders (e.g. SLE), chronic diseases (neoplasia, diabetes mellitus) and hypersensitivity disorders. Over 50% of vasculitis cases are idiopathic. With acute vasculitis the face (nose, lips), ear pinnae, digits, foot pads, scrotum and oral mucosa are mainly affected. Skin lesions initially include ecchymotic plaques and haemorrhagic pustules. With time fissures and "punched out" ulcers appear.

These necrotic lesions are painful. Systemic signs such as fever, anorexia, muscle and joint pain and lymphadenopathy are common. Chronic vasculitis typically affects the ear pinnae, tail tip, digits and foot pads where progressive ischaemia results in well circumscribed lesions. Systemic signs are uncommon in chronic vasculitis cases. The diagnosis is supported by histopathology of skin biopsies of the margins of lesions. Although many cases of vasculitis are idiopathic, attempts should be made to identify an underlying cause.

Treatment for idiopathic vasculitis often includes pentoxifylline as a first choice. Other therapies include prednisolone alone or in combination with azathioprine, chlorambucil or cyclosporine. Tetracycline/niacinamide may be used for milder forms. Topical corticosteroids and 0.1% tacrolimus may be useful for localized lesions.

CANINE DISTEMPER:

Canine distemper is a multisystemic, often fatal disease, caused by a paramyxovirus virus. It commonly causes gastrointestinal, respiratory and neurological disease, but can also cause vesiculopustular dermatitis and foot pad hyperkeratosis ("hard pad" disease). Classic skin signs such as hyperkeratosis of the foot pads often develop after clinical cure or when the earlier clinical manifestations have cleared.

The foot pad lesions may sometimes be seen in older dogs with an incomplete vaccination history. Since vaccination can prevent distemper, distemper related skin disorders are rare. Diagnosis can be confirmed by histopathology when intra-cytoplasmic and intranuclear inclusion bodies can be seen. There is no specific treatment.

SYSTEMIC LUPUS ERYTHEMATOSUS (SLE):

SLE is an auto-immune condition where multiple circulating auto-antibodies participate in immune-mediated tissue injury directed against the dog's own body. The most common clinical findings include cyclical fever, polyarthritis, kidney disease and skin disease. Skin lesions are diverse and include erythema, ulcers and crusts of the face (nose, lips), ear pinnae, limbs, axillae and inguinal area. Ulceration and thickening of the foot pads may also occur.

Diagnosis of SLE can be difficult due to the cyclic nature of the disease. Specific diagnostic tests include the antinuclear antibody test (ANA test) and biopsies for histopathology and immunopathology. SLE is treated with immunosuppressive dosages of prednisolone with or without azathioprine or cyclophosamide.

NECROLYTIC MIGRATORY ERYTHEMA (NME) aka Superficial Necrolytic Dermatitis, Hepatocutaneous Syndrome, Metabolic Dermatosis, Metabolic Epidermal Necrolysis: NME is a rare, ulcerative, crusting condition that affects the mucocutaneous junctions (muzzle, lips, nose, eyelids, anus, genitalia) and pressure points (elbows, feet). Hyperkeratosis and fissuring of the foot pads commonly occurs. All foot pads are usually involved and the interdigital spaces and nail folds are also typically inflamed and crusted. The paws often become very painful resulting in lameness and reluctance to walk. Secondary infections with bacteria, fungi or yeasts are common. The disease is generally seen in middle aged to older dogs and may wax and wane. Foot pad and skin lesions are common early manifestations of this condition and precede or accompany chronic liver disease (e.g. cirrhosis, drug-induced hepatitis (phenobarbital) and chronic active hepatitis) or very rarely glucagon-secreting pancreatic tumours (glucagonomas). The lesions are thought to be manifestations of nutritional abnormalities related to amino acids, essential fatty acids or zinc. Systemic signs usually occur later and may include weight loss, lethargy, anorexia, icterus, polyuria and polydipsia. Concurrent diabetes mellitus is relatively common (especially later in the course of the disease). Histopathologic findings of skin biopsies are classic and highly characteristic.

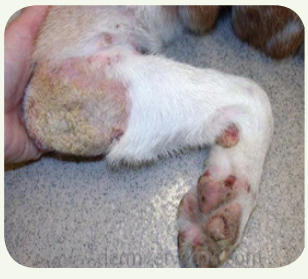
These include a superficial perivascular to lichenoid dermatitis, parakeratotic hyperkeratosis, and a marked intra- and intercellular oedema limited to the upper half of the dermis. These histologic findings appear as a red, white and blue colouring band within epidermis. Once a diagnosis of NME has been made, hepatic ultrasonography should be performed. Hepatic ultrasound usually reveals a hyperechoic network surrounding hypoechoic areas of parenchyma, likened to a Swiss-cheese or honeycomb appearance. This pattern is considered pathognomonic in the dog for NME. There is no effective treatment and the prognosis is guarded. Corticosteroids should not be used as many of these patients are diabetic or prediabetic. Temporary improvement may be achieved with nutritional support. Amino acid supplementation either with intravenous amino acid preparations or with oral supplementation e.g. egg yolks (1 egg yolk per 5 kg body mass), essential fatty acids and zinc can be given. A low fibre, highly digestible diet is recommended. Treatment of the underlying liver disease would be the best option. Drugs such as colchicine and milk thistle are often used. Where a glucagonoma can be surgically removed, the skin lesions start to resolve a week after surgery and completely resolve within 45 days.

Necrolytic migratory erythema of the footpads (http://www.dermatologyforanimals.com/faq-38/)

ZINC RESPONSIVE DERMATOSIS: Zinc is very important in ensuring epithelial tissue integrity. Any qualitative or quantitative deficiency in zinc, may therefore cause skin problems. Two types of zinc responsive dermatosis have been described.

Type 1 is a syndrome that has been described in young dogs. It is considered to be a genodermatosis involving a defect in intestinal zinc absorption and possibly zinc metabolism at cellular level. Nordic breeds are predisposed with 75% of cases reported in Siberian Huskies. The age of onset is usually between 6 months and 3 years. This condition mainly affects the face (lips, ear pinnae, bridge of the nose, periorbital area), foot pads and perianal region. Typical skin lesions include erythema, scaling and crusting of all affected areas. Systemic signs, such as pyrexia, are common. Diagnosis is based on history, lesion distribution and histopathology of skin biopsies. Prognosis is good, but recurrences are common. Treatment typically consists of zinc therapy (zinc methionine), low dose prednisolone to help intestinal zinc absorption, antibiotics in cases of secondary bacterial infections and keratomodulating shampoos for the skin lesions.

Type 2 is a syndrome seen in large breed puppies on poorly balanced diets (excessive cereal, phytate or calcium) or in adult dogs with intestinal malabsorption. Dermatological signs are identical to those seen with type 1 and systemic signs, in particular pyrexia, are common. Diagnosis is based on history, clinical signs and histopathology of skin biopsies. The prognosis is excellent in general. Zinc should be given in conjunction with a balanced diet for 3 to 4 weeks and can then be stopped. In cases of malabsorption the supplementation may be lifelong unless the cause of the malabsorption can be diagnosed and treated successfully.

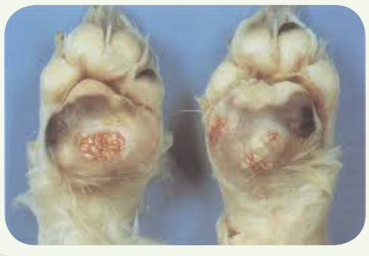


A: Zn-responsive dermatitis affecting the feet and elbow (http://dermvettacoma.com/zinc-responsive-dermatosis/)
B: Zn-responsive dermatitis affecting footpads (http://www.siberianhuskyhealthfoundation.com/images/Untitled-1.jpg)

FOOT PAD FOREIGN BODIES: Foot pad foreign bodies are very common and easily missed. The entry wound may be very small and the foreign body not visible to the naked eye. Glass and other sharp hard objects are usually the cause. Well localised discomfort is usually present and the patient often presents with lameness. The treatment is general anaesthetic and surgical excision or removal.

CALCINOSIS CUTIS: Calcinosis cutis is a collective description for a group of conditions that result in pathological calcification in the skin. These include dystrophic calcification (seen with hyperadrenocorticism and diabetes mellitus), metastatic calcification (seen with renal insufficiency) and it may be idiopathic (calcinosis circumscripta). Skin lesions are usually firm, cutaneous or subcutaneous nodules.

There is no typical distribution pattern but foot pads are commonly involved. Several cases of foot pad calcification have been reported in small dogs with renal dysplasia. The lesions usually develop after or at the same time as other signs of renal insufficiency. Diagnosis is based on skin and renal signs, histopathology of skin biopsies and further tests to evaluate the kidneys (serum chemistry, urine examination, ultrasound). The prognosis is guarded in most cases depending on the cause of the renal disease.



Calcinosis cutis (Gross TL. 1997. Calcinosis circumscripta and renal dysplasia in a dog. From: Veterinary Dermatology 8(1): 27 – 32)

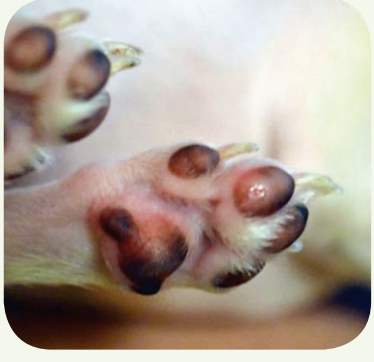
FOOT PAD BURNS: Chemical and thermal superficial foot pad burn wounds may present with varying loss of epidermis and dermis. These wounds are treated with topical silver sulfadiazine cream and bandaged with a non-adherent, semi-occlusive bandage. Initially, daily wound dressing and bandage change is recommended. For superficial burns, re-epithelialization may be complete by seven to nine days. With deeper injuries, healing may take up to 21 days, depending on the size of the wound.

Foot showing burns of the footpads (http://livewagbark.com/wp-content/uploads/2015/07/5751084148_5085e13502_b.jpg)

FOOT PAD FROST BITE: When the environmental temperature drops below 0°C, blood vessels close to the skin start to constrict to preserve core body temperature by diverting blood toward the core and away from the cooler parts of the body. This can reduce blood flow in some areas of the body, especially the extremities, to critically low levels. The combination of cold temperature and reduced blood flow can allow the tissues to freeze, causing severe tissue injury. The paws, ears and tail are the most common tissues to be affected. If a dog is wet or damp, these areas are more vulnerable to frostbite.

The initial clinical signs associated with frostbite include discoloration (pale, grey, blue), coldness and swelling of the affected area, pain, blisters or skin ulcers and areas of blackened or necrotic skin. As frostbitten tissues thaw, they may become red and very painful due to inflammation. The clinical signs of frostbite may take several days to appear. Severely frostbitten areas will become necrotic, die and slough off. During this time secondary bacterial infection commonly occurs. Diagnosis is usually based on the history and clinical findings. Treatment is symptomatic and may include treatment for shock and pain. Antibiotics are used to prevent secondary bacterial skin infections.

The prognosis for frostbite depends on the extent of the injuries. Mild cases of frostbite usually resolve with little permanent damage while more severe frostbite may result in permanent disfiguration or alteration of the affected tissues. Amputation of a severely affected body part may even be required.

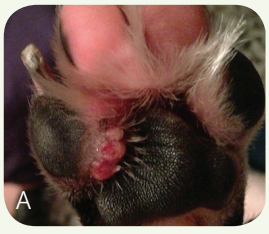


Foot showing frostbite (http://www.famouschihuahua.com/chihuahua-health-concerns/winter-paw-care/)

FOOT PAD TRAUMA: Foot pad trauma is one of the most common causes of foot pad lesions. Examples are lacerations as a result of sharp trauma or blisters, abrasions and erosions which are frequently found in dogs who accompany their owners jogging on hard surfaces, on hot tar or who obsessively play tug-of-war. Superficial and full thickness injuries to foot pads generally heal in a similar fashion when compared to other skin surfaces, however they heal more slowly and with more complications.

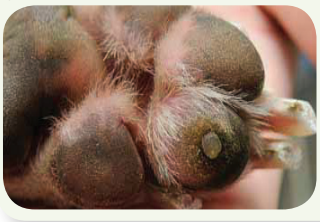
Injured foot pads are exposed to contaminated surfaces, self-trauma and excessive motion and tension and are difficult to prepare aseptically. As a result, contamination of foot pad wounds is more common. Most superficial pad lacerations heal without complications, regardless whether they are repaired or not, if bandaged appropriately. Full thickness pad lacerations should be repaired with a two-layer technique and bandaged to relieve motion and pressure on the repair.

CANINE PAPILLOMA VIRUS: Papilloma viruses (Papovavirus family) exhibit a marked tropism for squamous epithelium, are species specific and potentially contagious. This virus can cause a variety of clinical entities in dogs from warts in the oral cavity and on the skin, cutaneous horns on the foot pads to cutaneous inverted papillomas. Diagnosis is made clinically due to the characteristic lesions but may be confirmed by histopathology. There is no specific treatment. Many virus induced lesions may regress spontaneously after a few months. Treatment that may be attempted for papilloma virus infections include interferon and a vaccine created from surgically excised tissue.



A: Canine papilloma virus (http://www.askavetquestion.com/answer_np.php?id=4717-wart-on-dogs-foot)
B: Canine papilloma virus (http://www.allwidewallpapers.com/extra-pad-on-dogs-paw/ZXh0cmEtcGFkLW9uLWRvZ3MtcGF3/

Papilloma viruses have also been implicated as one of the causes of a condition called Canine Foot Pad Papilloma. This condition has primarily been reported in active and retired racing Greyhounds but may affect any breed. The foot pad lesions are firm, well demarcated, circumscribed hyperkeratotic lesions that have a central core of keratin which is often conical. The gross appearance is that of scar tissue. These lesions cause pain and local inflammation and have been called corns. Corns can appear on multiple foot pads and cause lameness. Other proposed causes are foreign bodies and repetitive mechanical trauma from pressure and abrasions.



Corn in foot pad (From: Fawcett A, Phillips A. 2012. Clinical snapshot: A Corn in a Whippet. Compendium for continuing education; September 2012: 34: 9)

VITILIGO: Vitiligo is a condition associated with an acquired loss of pigment in the skin due to selective melanocyte destruction. The pathogenesis is not clear but an auto-immune mechanism is most likely. Vitiligo is sometimes associated with other auto-immune conditions such as juvenile diabetes mellitus. Dermatological signs include achromic macules at mucocutaneous junctions (nose, eyelids, lips and anus) and depigmentation (without inflammation) of the foot pads, nails and coat.

Diagnosis is usually made on clinical findings with or without histopathology. Histopathology shows relatively normal skin which is devoid of melanocytes. There is no specific treatment as the condition is merely cosmetic. Topical 0.1% tacrolimus has occasional success. Spontaneous remission sometimes occurs.

ICHTHYOSIS: Ichthyosis is a congenital disorder resulting from a defect in one or more steps of stratum corneum differentiation. Epidermolytic and non-epidermolytic forms have been described.

Breed specific forms have been identified. Breeds affected include Golden and Labrador retrievers, Cairn and Norfolk terriers, Jack Russell terriers, Rottweilers and American bulldogs.

Dermatological signs vary with the form and breed affected and vary from a mild exfoliative dermatosis to generalised thick, large scales adherent to the skin. Foot pad involvement does not occur in all cases, but where it does, it is characterised by extreme hyperkeratosis and exaggerated thickening of the digital, carpal and tarsal foot pads. Severe cases usually present at the age of 2 months, but milder cases may only present in young adulthood.

Diagnosis is made by histopathology of skin biopsies. Treatment is symptomatic and includes moisturising agents, keratomodulating shampoos to correct excessive scale production and improve skin hydration and essential fatty acid supplementation to limit water loss.

PRACTICAL TIPS

- In a generalised skin condition, where the foot pads are also affected, take samples and biopsies from areas other than the footpads, where possible.
- Foot pad wounds bleed profusely and can be slow to heal.
- It is easy to miss representative lesions when taking biopsies of foot pads.
- Histopathological interpretation of foot pad skin is challenging.
- Foreign bodies should be ruled out as a cause at an early stage, especially in patients who present with lameness and where only one foot pad is affected.
- Consider pain management in all cases.
- Aggressive medical treatment is frequently required.
- Topical treatments will always be a beneficial adjunctive therapy, also useful palliatively.
- Foot coverings will help to protect the foot and increase contact time of topical treatments.
- Review diagnosis in cases of poor response to treatment.

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

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