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Editor's Note

I long for the days when I was young and time seemed to drag. When it took forever for holidays and special events to come around. As we get older time just seems to rush past us.

We are already in the middle of the year and the days are starting to lengthen again. This edition includes some topical information from Dr Salome Nagel regarding the addition of cholicalciferol to many of our traditional anticoagulant rodenticides.

This requires us to modify our management of suspected cases as well as treatment of proven cases. Dr Christiaan Triegaardt has also written an informative piece on practical considerations to improve the outcome for cutaneous and subcutaneous malignant masses.

Our ou staatmaker, Dr Izak Venter has also contributed an article on immune mediated eyelid conditions. I hope you enjoy the read.

Liesel

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VET360 aims to be a leader in the field of continuing veterinary development in Southern Africa by providing veterinary professionals from diverse disciplines with tools to help them meet the challenges of private practice. The magazine aims to make information accessible, both paper and electronic, and provide clinical, business and other veterinary information in a concise form to enable the practitioner to rapidly acquire nuggets of essential knowledge

We welcome any comments, contributions, topic suggestions and letters for publication. Send them to:
The Editor, PO Box 232, GROENKLOOF, 0027
Tel: (012) 346 1590, 082 575 6479. Fax: 086 671 9907
Email: publications@vetlink.co.za

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Publisher: Madaleen Schultheiss, madaleen@vetlink.co.za

Advertising Enquiries: AgriConnect
Ilse Liveris
Senior commercial consultant
072-708-4401 / ilsel@agriconnect.co.za

Illa Hugo
Senior Commercial
082 898 3868 / sales@agriconnect.co.za

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Editor : **Liesel van der Merwe** BVSc MMedVet (Med) Small Animals Small Animal Medicine Specialist, Department of Companion Animal Clinical Studies, University of Pretoria, South Africa

Striving towards Wellness: Emotions – the thing we never talk about

"When we deny our emotion, it owns us"
Brene Brown

Retha Watson
(MA Industrial Psychology)

Within the scientific and medical community, there is usually not much space for emotions and feelings. Medical science is dictated by empirical facts and does not allow emotions to be part of decisions and problem solving.

In the last two wellness articles, we were confronted with the fact that we remain human beings and therefore we do have emotions, and we have empathy for our patients and patient caregivers.

We experience emotions and subjective feelings on a daily basis in abundance and if this is not managed, it is possible for us to suffer from compassion fatigue, anxiety and depression, all emotion related.

In the 80's in South Africa, a well known theologist postulated that we get unemotional and emotional people, introverted and extroverted, these personalities were divided into four main personality groups. Many that followed this believed that they are unemotional and simply do not have the ability to express emotions.

Research today has refuted this, in simple terms, each person has emotions, they just experience them differently and communicate them differently, or supress their emotions completely.

Emotion is a cognitive process, completely reliant on appraisal and evaluation from our memories, judgements and perceptions. This evaluation process contains specific elements when we are confronted with a situation.

All emotions have an appraisal, however not all appraisals contain emotions. For example; we can evaluate the colour of a wall with no emotion involved or we, can evaluate the colour of a wall with emotion involved, all depending on:

- Our perceptions and what it means to us;
- Evaluation of our memory and what information that memory gives us;
- The emotion reaction;
- Our attention;
- The judgement;
- Attitude creation towards the situation;
- The decision on immediate and future actions;
- Problem solving.

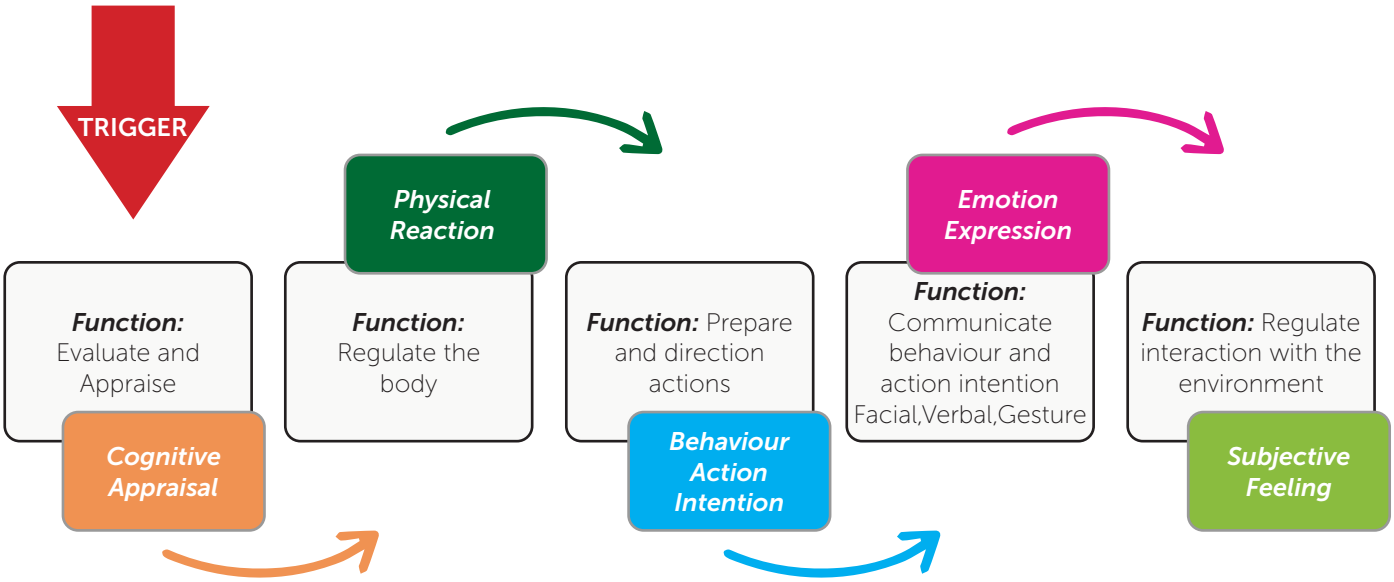
The intensity and the experience of the emotion relies on previous experiences (memories) and our perceptions.

The trigger incident is basically an incident which means a lot to you due to those memories and perceptions.

For example, a person had a previous negative experience/ trauma of bullying in school, a trigger event of a similar situation at work will intensify the emotion experience of that person, the same situation may occur to a person that has never been bullied and a different emotion experience will be observed.

Our emotions occur in split seconds, the process similar to that of dominoes stacked upright to fall in a pattern, when the first domino is touched.

The Emotion Process



We cannot ever say that we do not have emotions – "I feel nothing or numb" – well that is a feeling too. When we are aware of the emotion process, have knowledge of emotions and are able to communicate and manage our emotions effectively, we have emotional competence – the building blocks to emotional intelligence actualisation.

Emotion Labour and Emotion Management

Research shows us that depression and anxiety as psychological illnesses relate directly back to emotions and stands central to various health matters. The experience of negative emotions and the suppression of such emotions makes us physically and psychologically ill, This is because of our working or social environments and the expectation that there is not a space to communicate negative emotions – this is called emotional labour.

The question will then be, how do I express my negative emotions and still remain professional?

- **Ventilate emotions in a safe space** – trusted colleague, friend or professional.
- **Name (verbalise and acknowledge) the emotion** – frustration, anger, guilt, jealousy, helplessness, despair. Try and match the intensity of that emotion with a metaphor.
- **Normalise the emotion** – "it is okay to feel this emotion, most people will feel this emotion in the same situation". If you normalise to another person, "This emotion is okay, I feel this emotion too, many times..."
- **How can we manage this emotion?** Go for a walk/run, exercise, creative activities, writing in a journal, process the emotion – why did I feel this way? What triggered this intense negative emotion? Where in my past have, I experienced a similar situation? What perception or judgment can I change to adjust the impact of the emotion?

Emotion Communication and Expression

One of the most important results from social dysfunction is that we are unable to express our emotions effectively.

This will have an impact on our social relationships and interactions with other people. When we do not show emotion, it does not mean that we do not experience emotions. Learn a vocabulary for your emotions and subjective feelings and acknowledge and name the emotion when you experience it. Try and read other people's emotions and how they communicate those emotions, sometimes effectively, sometimes quite destructively.

Experiencing emotions is not a shameful thing, it is part of our physical and psychological make-up. Once we accept that, we can regulate and communicate the emotion effectively without it impacting negatively on our relationships.

Empathy

Once we are able to understand that every person has unique emotions, that they experience and express it in different ways and that their emotion at that point in time is important to them, then we to grasp the concept of empathy.

We have empathy when we try and understand how that person is feeling at that stage and what and what the reason for that particular emotion is. We do not have to experience the same emotion, but you can start a dialogue about it.

When you enquire about other's emotions, they have a clear message that you care and that you are interested in them. We are more comfortable with questions relating to facts and what happened? We rarely ask others about their emotions, usually because we are afraid that it may intensify the emotion – it will not; or our own reaction to the emotion – it is okay to be sad or angry with someone sometimes.

If you can put yourself in the proverbial shoes of another person in your social interactions, you will portray empathy. This skill will immediately improve your relationships on all levels as well as your interaction with your environment.

We have seen many quotes on being kind as we do not know what another person is going through at a given point in time. Our main objective with empathy is not is not to have value.

You also do not have to try and rationalise or justify the emotion the person is experiencing. As long as the person knows that their emotion is important to you and that it is okay to have that emotion.

Emotion Intelligence

Emotional intelligence refers to the capability of a person to manage and control his or her emotions and possess the ability to control the emotions of others as well. In other words, they can influence the emotions of other people too.

Emotional intelligence is a very important skill in various aspects of organisations and our social spheres.

It is said to have five main elements such as:

- self-awareness,
- self-regulation,
- motivation,
- empathy, and
- social skills.

Let's understand each one of them in detail. What is **self-awareness?** If you are self-aware of what you are going through, you would be in a better position to understand others and how you affect people around you. It also means you are aware of your strengths as well as weaknesses.

What is **self-regulation?** Self-regulation is the next step wherein you think before speaking. It is an important aspect where you can regulate yourself. This will impact others in a positive way rather than in negatively. Hold yourself accountable in case you make a mistake and try to remain calm in every situation.

What is **motivation?** When you are motivated to do a series of tasks you will be in a better position to influence others. Work towards your goals consistently.

Show your employees or colleagues how the work is done and lead by example. Even if you are faced with a challenge try and find something good about the situation.

What is **empathy?** When you are able to put yourself in other's shoes and think about a situation, it is known as empathy. Every successful person should know how to empathise with others, if you want to earn their respect.
What are social skills?

The last aspect is social skills, and it is one of the important aspects. Social skills are all about communicating your point of view. You are able to build a rapport with others which makes the relationship more comfortable.

"However, emotions can lie, because they are not products of reality but of our interpretation of reality"
Les Parrot; Love's, Unseen Enemy

All the information has been factually and scientifically sourced and available on request. Retha Watson is the developer of an Emotion Competence to Intelligence training course and have sourced the information from research.

The training is an experiential learning experience and focus in detail on all the aspects mentioned in the article. counselling after hours in order to provide support with long working hours in mind.

WCC has established a service of online counselling after hours in order to provide support with long working hours in mind.

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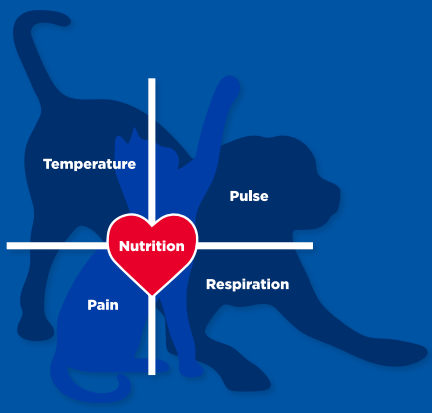

SCIENCE DID THAT.

Autoimmune Eyelid Disease in Dogs

Dr Izak Venter
BVSc, MMedVet (Ophthal)
Digital Veterinary Ophthalmology Services
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Introduction

The eyelids of canines are immunologically active structures. A dense network of blood vessels, lymphatic vessels and immune cells characterizes this. Consequently, these structures are susceptible to various immune-mediated processes. These processes can manifest either in isolation or concurrently with systemic clinical signs. Fortunately, such occurrences are uncommon.

Two primary categories encompass immune-mediated eyelid diseases:

- Primary Autoimmune Disease: This category refers to conditions where the immune system attacks the body's own tissues (self-antigens) within the eyelids.
- Secondary Immune-Mediated Disease: In this category an exogenous material triggers an autoimmune response, leading to the disorder. Examples of such exogenous material include infectious agents and medications.

We will cover several conditions in this article.

1. Medial canthal ulcerative blepharitis

Medial canthal ulcerative blepharitis is a juxtapalpebral disorder, primarily affecting the medial canthus. This condition exhibits are breed predisposition, with German Shepherds, Long-Haired Dachshunds, and Toy and Miniature Poodles are commonly diagnosed.

In German Shepherds, medial canthal blepharitis may co-occur with pannus (chronic superficial keratitis) and immune-mediated plasma cell infiltration of the third eyelid (Fig 1). Similarly, Long-Haired Dachshunds with this condition might also present with superficial punctate keratitis. The disease typically occurs bilaterally.

Histopathological examination of affected tissue reveals infiltration by lymphocytes and plasma cells. Additionally, sebaceous glandular hyperplasia (enlargement) may be observed. In some cases, the presence of epithelial cell antibodies has been identified, suggesting a potential link to pemphigus, an autoimmune blistering disease.

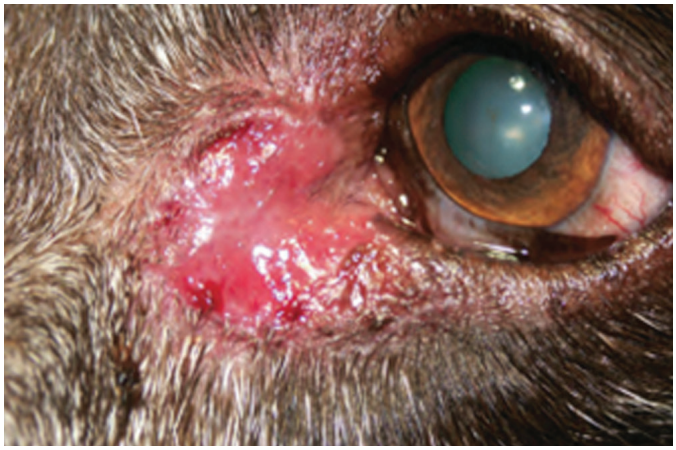

Figure 1. Immune-mediated medial canthal blepharitis in an adult mongrel German hunting dog.
Source: [Stades FC, Gelatt KN. Diseases and surgery of the canine eyelid. In: Gelatt KN, editor. Veterinary ophthalmology. 4th edition.]

Medial canthal ulcerative blepharitis generally responds well to topical ophthalmic treatment with corticosteroids. In non-responsive cases oral doxycycline and corticosteroids as well as topical tacrolimus may be added to the treatment protocol.

2. Uveodermatologic Syndrome/Vogt-Koyanagi-Harada-Like Syndrome[VKH]

Uveodermatologic syndrome is an idiopathic condition theorised to have resulted from a Th-1 lymphocytic cell, immune-mediated attack on melanocytes in the uvea and skin. This is an extremely frustrating condition to treat, and the long-term prognosis is poor. The loss of pigmentation of the nose and eyelids is the primary clinical sign observed.

Other key ocular examination findings include:

- Presence of aqueous flare.
- Other signs of uveitis including episcleral congestion, corneal oedema and bullous retinal detachments.
- Secondary cataracts and glaucoma.

- Progressive depigmentation of iris and retinal pigment epithelium.
- Development of hyper-reflective tapetal fundus, with vascular attenuation and optic nerve atrophy

The condition is commonly bilateral, but unilateral disease has been reported. Breed predisposition is important; affected breeds include Akita, Siberian Husky, Golden Retriever, Samoyed, Rottweiler, Chow Chow, Shetland Sheepdog, and others.

No specific diagnostic test is available but the clinical signs are very indicative (Fig 2). Histopathologic examination of skin biopsy reveals lichenoid dermatitis, histiocytes, and giant cell infiltration, as well as decreased levels of melanin in the epidermis and hair follicles.

Initial therapy involves immuno-suppressive doses of oral prednisone, oral doxycycline and niacinamide has been suggested as an adjunctive treatment. The use of doxycycline and niacinamide for autoimmune skin disease is discussed later in this article.

The uveitis and possible secondary glaucoma should also be addressed, and treatment options include topical prednisolone acetate/dexamethasone as well as topical dorzolamide/brinzolamide if the intraocular pressure is increased. In nonresponsive cases, azathioprine should be added to the treatment. Most patients will develop severe glaucoma despite aggressive treatment often requiring bilateral enucleations.

3. Immune-mediated blepharitis pemphigus complex

The pemphigus complex encompasses a group of uncommon autoimmune disorders characterized by the presence of autoantibodies targeting the intercellular matrix of the epidermis. This immunological phenomenon leads to a type II hypersensitivity reaction, ultimately manifesting as cutaneous lesions.

The complex encompasses five recognized variants: vulgaris, foliaceous, erythematosus, vegetans, and bullous. Pemphigus foliaceous is the most frequently diagnosed variant in small animals.

A hallmark feature of the pemphigus complex is its potential to involve mucocutaneous junctions, particularly the eyelids. This involvement often presents as inflammation and ulceration of the eyelid margins. Facial lesions specifically affecting the eyelids can manifest as pustules or vesicles that progress to rupture, leaving behind erosions, ulcers, crusting, scaling, and hypopigmentation (Fig 3).

Of the recognised variants, pemphigus vulgaris is considered the most severe. It has a broader target tissue range, encompassing the oral cavity, nail beds, skin, eyelids, lips, and nares. Pemphigus foliaceous and vulgaris can be life-threatening, while pemphigus erythematosus is generally milder, rarely causing systemic signs and demonstrating a good response to treatment.

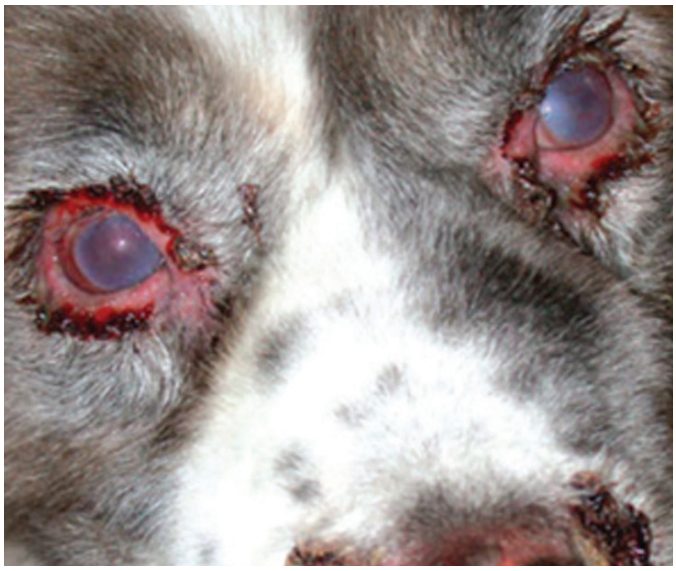

Figure 2. Two-year-old castrated male Siberian husky with uveodermatologic syndrome. Note the ulceration, crusting, and depigmentation of the eyelids and nasal planum. Corneal oedema and miotic pupils indicative of uveitis are visible.
Source: [White BL, Belknap EB. 2015. Metropolitan Veterinary Hospital, Akron, OhioClinical Approach to Canine Eyelid Disease: Blepharitis. Today's veterinary practice]

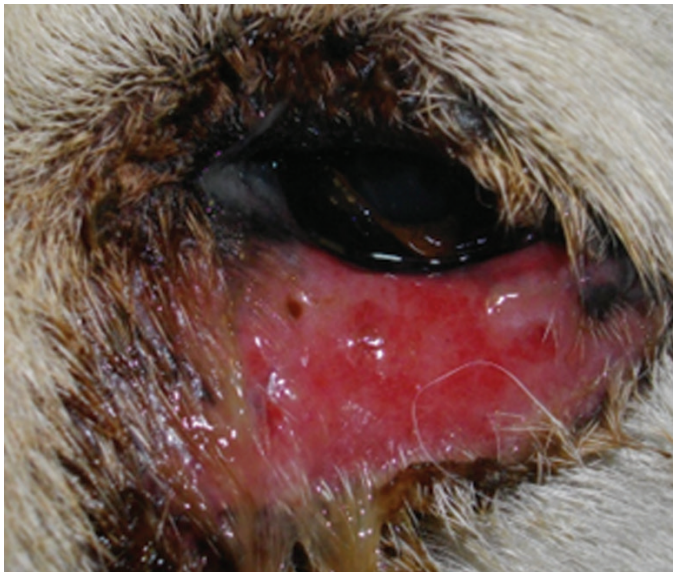

Figure 3. Immune-mediated blepharitis pemphigus complex. Note the severe mucopurulent discharge and extensive eyelid ulceration.

Diagnosis. Biopsy with histopathologic examination is important for differentiation between the variants:

- Pemphigus foliaceous: Neutrophils or eosinophils present within vesicle or pustule, intragranular and sub-corneal acantholysis with cleft and vesicle formation, and acantholytic epidermal cells found at surface of erosions
- Pemphigus erythematosus: Condition is identical to

pemphigus foliaceus except for the fact that it often has a lichenoid cellular infiltrate of mononuclear cells, plasma cells, and neutrophils or eosinophils or both.
- Pemphigus vulgaris: Suprabasilar acantholysis is present with resultant cleft and vesicle formation. Basal epidermal cells remain attached to the basement membrane zone like a row of tombstones. The inflammatory reaction may be scant and perivascular or prominent and interstitial to lichenoid.

Treatment. General long-term treatment includes topical and systemic corticosteroids, combined with additional immune suppression using cyclophosphamide, azathioprine, or cyclosporine for refractory cases. A combination of doxycycline and niacinamide has also been reported. Blepharoplasty may be indicated for correction of secondary cicatricial entropion

4. Canine Discoid Lupus Erythematosus

Canine discoid lupus erythematosus (DLE) represents a distinct entity within the broader spectrum of lupus erythematosus. This group of diseases encompasses various clinical presentations with a common underlying theme of autoimmunity.

However, DLE manifests as a relatively benign, localised skin condition without systemic involvement. Notably, no documented cases demonstrate progression from DLE to the more severe canine systemic lupus erythematosus.

While a breed predisposition exists in German Shepherd dogs, sun exposure appears to be a significant contributing factor in roughly half of all cases. This photosensitivity suggests a role for ultraviolet radiation in the disease process. Clinically, DLE presents with facial dermatitis characterised by crusting, loss of pigmentation (depigmentation), erosions, and ulcers (Fig 4). These lesions primarily affect the nasal planum and muzzle, although eyelid and oral involvement have also been reported.

Diagnosis of DLE relies on a combination of the patient's history, physical examination findings, and confirmation through a skin biopsy. Fortunately, the prognosis for canine DLE is favourable.

Management strategies focus on minimizing sun exposure and utilizing topical immunosuppressive medications such as glucocorticoids, cyclosporin, or tacrolimus. Refractory cases may necessitate the use of systemic glucocorticoids. It is important to note that long-term therapy, possibly lifelong, is often necessary to control the disease.

5. Canine Juvenile Cellulitis

Canine juvenile cellulitis (CJC) is a well known lymphocutaneous disease predominantly affecting puppies under eight months of age. Certain breeds, including Dachshunds, Golden Retrievers, Labrador Retrievers, Gordon Setters, and Lhasa Apsos, exhibit a higher predisposition.

This relatively uncommon condition manifests as a granulomatous and pustular disorder, primarily targeting the face, pinnae, and submandibular lymph nodes. The typical clinical presentation involves an acutely swollen face, with particular emphasis on the eyelids, lips, and muzzle (Fig 5). This is often accompanied by a noticeable enlargement of the submandibular lymph nodes (lymphadenopathy).

Within a short period (24-48 hours), papules and pustules erupt around the lips, muzzle, chin, bridge of the nose, and the periocular area. In some cases, lesions may extend to the feet, abdomen, thorax, vulva, prepuce, or anus.

Figure 4. Canine discoid lupus erythematosus. Note the nasal depigmentation. Early signs of periocular depigmentation are also present.

Figure 5. Canine Juvenile Cellulitis, note the typical swollen muzzle and eyelids.
[Source: Dr Liesel van der Merwe]

These lesions frequently develop fistulae, and form crusts. Affected eyelids are typically painful but not pruritic. The puppies are systemically affected and are pyrexic and lethargic with a decreased appetite.

Bloodwork may reveal an elevated white blood cell count (leukocytosis) with a predominance of neutrophils (neutrophilia) and potentially a normocytic, normochromic anaemia. While a clinical diagnosis of CJC is possible, definitive confirmation relies on cytologic and histopathologic evaluations.

The exact cause of CJC remains elusive. However, a theory suggests a bacterial hypersensitivity reaction, potentially explaining the positive response to corticosteroids and the rapid progression of the disease. Early and intensive systemic therapy is crucial to prevent severe eyelid scarring.

The recommended approach involves immunosuppressive doses of systemic corticosteroids, gradually reduced (tapered) after 3-4 weeks of resolved clinical signs. If cytological or clinical signs indicative of a secondary bacterial infection are present, systemic bactericidal antibiotics such as cephalexin, amikacin or amoxicillin clavulanate should be incorporated into the treatment plan.

Treatment of autoimmune blepharitis

Autoimmune blepharitis often necessitates immunosuppressive therapy to manage the underlying inflammatory process. While effective, these medications can have significant side effects, requiring careful monitoring and consideration of potential risks and benefits.

Glucocorticoids: These medications are the mainstay of treatment for many cases of autoimmune blepharitis. However, their use is not without drawbacks. Glucocorticoids can induce numerous adverse effects that mimic the signs of Cushing's syndrome (idiopathic hyperadrenocorticism).

These potential complications include increased urination and thirst (polyuria/polydipsia), increased appetite (polyphagia), lethargy, muscle wasting, exercise intolerance, vulnerability to secondary infections (particularly of the lungs, skin, and urinary tract), and even severe skin changes like calcinosis cutis (deposition of calcium salts in the skin).

Cytotoxic Agents: Azathioprine represents another option for immunosuppressive therapy. Azathioprine can potentially be teratogenic, and suppress bone marrow function, necessitating regular monitoring of complete blood counts (CBC) to prevent potentially life-threatening complications like thrombocytopenia, leukopenia, or anaemia. In some dogs, azathioprine can also cause severe and sudden liver damage (hepatotoxicity).

Proposed alternative treatments. These include essential fatty acid supplementation, vitamin E, and a combination of tetracycline/doxycycline and niacinamide. Rothstein E et al. reported in a study evaluating treatment with tetracycline and niacinamide for DLE in 20 dogs, 14 (70%) responded well and did not need to undergo classic immunosuppression.

Tetracycline, a bacteriostatic antibiotic, has many biologic properties at certain dosages, including inhibition of leukocyte chemotaxis, inhibition of prostaglandin synthesis, inhibition of the complement system, inhibition of lipases and collagenases, and suppression of lymphocyte blastogenic transformation and antibody production. Niacinamide has been shown to prevent degranulation of mast cells, block antigen IgE-induced histamine release, and decrease protease release.

Conclusion

Autoimmune eyelid disease can vary from a mild to severe painful sight threatening disorder. Biopsy and clinical description can help differentiate the different autoimmune conditions as well as excluding a possible infectious or neoplastic aetiology.

References avaialbe on request

An Update on Rodenticide Toxicity in Dogs

Dr Salome Nagel BVSc MMedVet(Med) Small Animals
Pioneer Veterinary Medical Consultancy
salomepioneervet@gmail.com

Due to increasing awareness of death of non-target species (e.g. owls eating poisoned rats), and residual environmental (soil and aquatic) concentrations of first and second generation anti -rodenticides, cholcalciferol is becoming a more frequent component of rodenticides. Cholecalciferol containing rat poisons require less feeding, are more toxic to the rodent and are less toxic to prey species such as owls.

Cholecaciferol however makes the diagnosis and management of suspected rodenticide exposure less straightforward. It is essential to try to obtain a label or package insert of the rodenticide to check if it is a plain anticoagulant or also had a cholecalciferol component.

Types of rodenticides:

Rodenticides are classified as follows:

- The anticoagulant rodenticides:
  - First-generation anticoagulant rodenticides (FGARs), e.g., Chlorophacinone, diphacinone, coumatetralyl, warfarin.
  - Second-generation (superwarfarin) anticoagulant rodenticides (SGARs) e.g., Brodifacoum, bromadiolone, difenacoum, difethialone, flocoumafone.

- Non-anticoagulant rodenticides
  - Bromethalin
  - Cholecalciferol
  - Strychnine
  - Zinc Phosphide

This article will discuss anticoagulant and cholecalciferol rodenticides.

General Initial Decontamination

Animals will often present after ingestion of rodenticide was either witnessed or evidence of ingestion was found

(chewed packaging, remnants of bait blocks, suspicious substance found in vomitus). Correct identification of the active ingredient in the ingested product and post-exposure timing is crucial to successful management Owners should provide evidence of intoxication and estimated quantity if possible

With recent ingestion (within 1-2 hours), the patient should be decontaminated with induction of emesis (see table 3) or gastric lavage. Gastric lavage (multiple flushes using a large bore tube with 10ml/kg water at body temperature) is performed if there is a large volume of toxin ingested, the toxin has a narrow margin of safety, or emesis is contraindicated (e.g., a high risk of aspiration such as a recumbent animal).

Table 3 – Decontamination via emesis induction:

Emetic agent:	Dose:	Species:
3% Hydrogen peroxide	2.2 ml/kg (max. 45ml) (use with caution, can cause gastric & oesophageal irritation/ ulceration)	Dog
Apomorphine	0.02 mg/kg SC 0.03 mg/kg IV formulation 0.04 mg/kg IM 6.25 mg tablet formulation conjunctival (rinse eyes with saline thoroughly afterwards)	Dog
Xylazine	0.44 mg/kg IM 1.1 mg/kg SC or IM (reversal agent yohimbine 0.1mg/kg IV for dogs)	Cat Dog
Dexmedetomidine	40 µg/kg IM (reversal agent atipamezole)	Cat

Both anticoagulant and cholecaliciferol compounds are highly lipophilic and accumulate in the liver and fat and undergo enterohepatic circulation. Activated charcoal is therefore administered at 1 to 5g/kg with a cathartic (sorbitol 70% solution at 1 to 2 ml/kg). Sorbitol increases gastrointestinal motility, decreases absorption, and enhances elimination of charcoal through the gastrointestinal tract. Dosing is 1 to 2 g/kg orally every four to six hours for three to four doses, depending on patient progress.

Warm water enemas with lubrication can also be performed to promote gastrointestinal tract emptying and expel any toxin remnants.

The concurrent administration of a parenteral antiemetic (e.g. maropitant 1 mg/kg SC q 24h or ondansetron 0.1-0.3 mg/kg IV q 6-12 h) should be considered because of the high prevalence of vomiting from the activated charcoal or cathartic administration or if another agent was used to induce emesis.

Anticoagulant rodenticides (AR):

Warfarin was the first AR developed in 1948 following an outbreak of a previously unrecognised haemorrhagic cattle disease in the Northern United States and Canada in 1920, where a haemorrhagic agent coumadin was found in mouldy sweet clover.

Anticoagulant rodenticides (ARs) include the first- and second-generation (or superwarfarin) anticoagulant rodenticides (SGARs) and are very commonly used in rodent management control programs in many different settings.

The first-generation anticoagulants are multiple-feed rodenticides and are slower in action, requiring multiple feeds over several days to a week or more to induce death. Because of the slow onset of action of these baits the rodent doesn't develop bait shyness.

Heritable warfarin resistance to first-generation ARs has however been identified in many species of rats and mice, and this has led to the development of the second-generation ARs or SAGRs which are single-feed anticoagulants, making them much more potent for inducing death after once-off feeding.

Anticoagulant rodenticide (AR) toxicity:

Ingestion of ARs antagonizes the enzyme Vitamin K_1 epoxide-reductase which is responsible for the maintaining levels of active Vit K in the liver. Vitamin K is essential for the carboxylation of, and thus activation of, clotting factors II, VII, IX and X and the anticoagulant proteins C and S. Circulating plasma levels of activated clotting factors are sufficient to maintain normal coagulation for several days but as plasma levels become depleted, the extrinsic (VII), intrinsic (IX, X), and common (II) coagulation pathways are affected.

Clinical bleeding typically ensues 2 to 5 days post exposure, with progression to a life-threatening coagulopathy, unless Vitamin. K_1 is supplemented. ARs are highly lipid soluble compounds and accumulate in the liver.

Due to enterohepatic circulation ARs have a prolonged half-life resulting in a prolonged anticoagulant state and a long duration of treatment with Vitamin. K_1 for up to 4 weeks is sometimes necessary.

Diagnosis

Dogs with AR often present clinically with non-specific with non-specific signs due to hypovolaemia secondary to internal haemorrhage. It is important to note that patients typically present with signs of compensated or late shock , generally without any clear evidence of bleeding. They show lethargy, pallor, a dyspnoea, tachycardia and

inappetence – the clinical presentation being linked to the rate and amount of internal haemorrhage.

According to a multi-centre 2022 study evaluating cases from 2010 to 2020 the most common sites of haemorrhage were pleural space (haemothorax 37%), pulmonary parenchyma (24%), abdominal cavity (haemabdomen) (24%), skin/subcutaneous haematomas (21%), gastrointestinal tract (18%), pericardium (13%), oral cavity (13%), nasal cavity (11%), ocular (8%), and urinary tract (7%). Overall, 73% of dogs had some evidence of cutaneous or mucosal bleeding and 53% of dogs had cavitatory bleeding. Haemorrhage was observed in 45% of dogs at one site, while 55% had haemorrhage at multiple sites. The location and total number of sites with haemorrhage was not associated with survival or transfusion requirement.

Baseline prothrombin time (PT) is the first test to show a change, due to the short half-life of factor VII (4-6 hours) and becomes prolonged from 36-48hrs post-ingestion. Testing of PT prior to this time is unnecessary, unless the patient has been chronically ingesting ARs. As the plasma concentration of other functional Vitamin K_1-dependent clotting factors also decrease, activated partial thromboplastin time (aPTT) and activated clotting time (ACT) become prolonged as well (see table 1).

Therefore, with AR toxicity a disproportionally prolonged PT is found compared to aPTT. Platelet count may be decreased due to active bleeding through consumption or loss but is not directly affected by ARs and is usually still near the lower normal range.

Table 1 – Vit. K dependent coagulation factors and their half-lives:

Coagulation factor:	Half-life:	Coagulation factor test:
Factor II	40hrs	Increased PT
Factor VII	6hrs	Increased PT
Factor IX	14hrs	Increased PTT or aPTT
Factor X	16hrs	Increased PT

Note. Modified from "Coagulopathies in veterinary patients. When is it rat poison?" by D. Liu, L.G. King, https://todaysveterinarypractice.com/wp-content/uploads/sites/4/2016/06/T1409F02.pdf.

Previously, the thrombotest or PIVKA test (proteins induced in Vitamin K antagonism or absence) was thought to be a more sensitive and diagnostic indicator of AR toxicity than prothrombin time (PT). A 2007 study has however shown that the PIVKA test detects abnormalities of the extrinsic coagulation pathway just like the PT test does and is not more specific for vitamin K antagonism or deficiency.

Treatment:

ARs have different margins of safety and the ingested dose should be calculated for the toxic compound or at least roughly estimated and compared to the toxic dose (LD50) for that compound (see table 2). It is interesting to note that cats are much more resistant to the effects of ARs and are rarely affected. Non-toxic dose ingestion of a compound (LD10) does not require treatment unless the patient is geriatric, neonatal, has an underlying hepatic disorder or has previously ingested an AR.

Table 2 – Acute oral toxicity (LD50, mg/kg) of various ARs:

Anticoagu-lant:	Canine LD50	Feline LD50
Brodifacoum	0.25-1 mg/kg	25 mg/kg
Bromadiolone	10 mg/kg	> 25 mg/kg
Difenacoum	50 mg/kg	100 mg/kg
Diphacinone	3-7.5 mg/kg	14.7 mg/kg
Difethialone	5 mg/kg	> 16 mg/kg
Flocoumafen	0.0 7 5 - 0.2 5 mg/kg	> 10 mg/kg
Warfarin	20-50 mg/kg	6-40 mg/kg
Factor X	16hrs	Increased PT

Note. From "https://www.inchem.org/documents/ehc/ehc/ehc175.htm

Baseline PT should be measured 36-48 hours post-ingestion, and if prolonged, treatment with oral Vit. K_1 at 2.5-5 mg/kg PO q 24h with a meal for 7 days (for FGARs) to 30 days (for SGARs) should be given, with PT re-assessed 2-3 days after discontinuation of Vit. K_1. If PT increases following withdrawal of Vitamin K_1, oral dosing should be resumed for 2-3 weeks, and the PT again re-tested 2-3 days post-Vit. K_1 treatment. Where the AR has not been identified, a minimum treatment period of 21 days with Vit. K_1 is recommended. Readers are cautioned that Vit. K^3 is a synthetic form of Vit. K and is ineffective in anticoagulant poisoning and should not be used.

Patient Management

If a patient presents with coagulopathy, treatment should be aimed at stabilising the animal with transfusion of blood products or plasma, oxygen therapy, supportive fluid therapy, and injectable Vitamin K_1. Vit K injectible (Konakion®) must be given slowly IV as it may trigger an anaphylactic reaction. Subcutaneous and intramuscular administration should be split up into multiple sites as there is a risk of haematoma formation and poor resorption rates. Oral treatment with Vit. K_1 should be continued for the AR compound as stated above and PT checked accordingly.

Cholecalciferol-containing rodenticides:

The extensive worldwide use of SGARs has led to

the development of resistance against ARs in some rodent species. Cholecalciferol-containing products were initially combined with coumatetralyl in especially European countries, but since 2020 many baits contain only cholecalciferol. The cholecalciferol or Vit. D_3 containing rodenticides have become a popular alternative to traditional SGARs due to their reported increased effectiveness in controlling rodent infestations. Rodents have a specific sensitivity to cholecalciferol and this low tolerance means that very little feeding is required for toxicity (2 days), making this an ideal rodenticide for mice that are notoriously difficult to control because of their sporadic feeding habits. The rodents will die within 4 – 7 days.

EFEKTO Eco-Rat™ is a green colourband pesticide and falls within the least hazardous class of pesticides. The product label states that there is a reduced risk of secondary poisoning for pets such as dogs and cats and very little risk of secondary poisoning to raptors like owls. The Griffon poison information centre endorses Eco-Rat™ as an effective rodenticide that reduces the risk of secondary poisoning to owls and raptors due to very low avian toxicity.

A 2023 study in Malaysia examined the efficacy of cholecalciferol to control wood rats (when compared to FGARs) and the impact of secondary poisoning on captive barn owls fed the cholecalciferol-poisoned rats. Cholecalciferol baits had the highest rodent mortality rates (71%) and all barn owls survived the 7-day alternate feeding test with cholecalciferol-poisoned rats throughout the study up to 6 months after exposure.

Despite cholecalciferol-containing rodenticides posing less of a risk to raptors and owls, in the veterinary sphere it is still considered one of the deadliest rodenticides to pets leading to severe and prolonged hypercalcaemia that is difficult to treat and results in multi-organ damage.

Physiology:

Cholecalciferol (or Vit. D_3) is crucial for calcium homeostasis in conjunction with parathyroid hormone, with the thyroid-secreted hormone calcitonin having a lesser impact. Conversion of 7-dehydrocholesterol to pre-Vit. D_3 , the final step in the synthesis of cholesterol by ultraviolet B-induced photolysis in the skin occurs in humans but is inefficient in dogs and cats.

Cholecalciferol in dogs and cats is thus mainly obtained through the diet (plant and animal sources). Ingested cholecalciferol is absorbed in the small intestine and transported by Vit. D-binding protein to the liver where hydroxylation by 25-hydroxylase to calcidiol (Vit. D_2) occurs. Calcidiol is the main circulating form of cholecalciferol and is transported to the proximal tubules in the kidney where it is further converted by 1-α hydroxylase to calcitriol which is the active form of Vit. D_3. The main functions of calcitriol in the body are to

- promote intestinal absorption of calcium and phosphorous by producing calcium binding proteins or calbindins in the small intestine.
- calcitriol facilitates the reabsorption of renal excreted calcium and phosphorous.
- at high doses, calcitriol increases mobilisation of calcium from bone.

Pathophysiology:

Cholecalciferol-containing rodenticides have a very narrow margin of safety, and a very little amount needs to be ingested for toxicity to occur. The LD50 in dogs is 85 mg/kg (based on the rodenticide concentration of 0.075%) but dosages as low as 0.1-0.5 mg/kg can result in clinical signs and hypercalcemia.

Ingestion of toxic levels of cholecalciferol results in severe hypercalcemia and hyperphosphatemia (phosphate often increases before calcium, typically >1.7 mmol/l The serum calcium to phosphorus product (Ca-Phos product) is calculated by multiplying total serum calcium concentration (mg/dL) with serum phosphorus concentration (mg/dL). If calcium and phosphorous are measured in mmol/L, the following calculation can be used to convert mmol/L to mg/dL:

ca-phos product = (4 x serum Calcium in mmol/L) x (3.1 x serum phosphate in mmol/L)

If the Ca-Phos product exceeds 6 mmol/L calcium phosphate is predisposed to precipitate in blood vessels, joints, and soft tissues as described in humans. This process is known as metastatic calcification and is especially important in organs that secrete protons, such as the stomach and kidneys, although other organs and tissues such as the myocardium, lung, and liver are also commonly mineralized as seen in patients with chronic kidney disease. Secondary acute kidney injury develops, and these patients become azotaemic within 12 - 36 hours following toxic ingestion if untreated.

Most animals with total serum calcium concentration greater than 3.5 mmol/L will show some systemic signs, and those with serum calcium concentrations greater than 4 mmol/L are usually severely ill.

Clinical signs:

Typically, clinical signs are only seen 1-3 days post-ingestion when azotaemia due to acute kidney injury is already evident.

Clinical signs are attributed to the hypercalcaemia and include polydipsia, polyuria, anorexia, depression, weakness, vomiting, dehydration and constipation. Signs of uraemia may be present (halitosis, haemorrhagic diarrhoea). Uncommonly, cardiac arrhythmias, seizures, and muscle twitching can occur. Severe hypercalcemia that has developed rapidly can result in death.

Diagnosis:

The antemortem diagnosis of cholecalciferol toxicity is based on a history of recent exposure and compatible clinical signs. A serum chemistry profile will demonstrate hypercalcemia (total and ionized), hyperphosphatemia,

and azotaemia (elevated blood urea nitrogen and serum creatinine). Differentials for hypercalcemia should be investigated when a patient has an uncertain history of cholecalciferol exposure. (see table 4). If serum 25-monohydroxy vitamin D_3 (25(OH)D_3) concentration can be measured it will be elevated and serum intact parathyroid hormone concentration will be reduced.

Treatment:

Cholecalciferol toxicity requires immediate and prolonged treatment by decontamination, reducing vit D absorption, treating the hypercalcaemia and reducing osteoclastic activity.

Baseline measurements of serum calcium, phosphorus, blood urea nitrogen, and creatinine concentrations should be obtained. These parameters need to be monitored daily for four days. If the values remain normal and the patient remains asymptomatic, no further monitoring or treatment is necessary.

1. **Decontamination:**
   Decontamination by induction of emesis (see table 3) or gastric lavage should be performed for acute ingestion. Cholecalciferol is a fat-soluble vitamin and undergoes enterohepatic recirculation. Therefore, the administration of multiple oral doses of activated charcoal over several days is warranted.

2. **Reducing Vit. D absorption:**
   Cholestyramine, like activated charcoal, inhibits Vit. D absorption from the intestine and is dosed at 0.3-1 g/kg q 8h for 4 days or more until the patient is normocalcaemic.

3. **Hypercalcaemia should be aggressively treated by promoting calciuresis:**
   If the patient is hypercalcaemic, the aim should be to keep the total calcium concentration < 3 mmol/L (ionised calcium < 1.3 mmol/L) and the phosphorous < 2.3 mmol/L, to prevent soft tissue mineralisation.
   - Intravenous 0.9% saline diuresis limits renal calcium absorption (any replacement fluid will be of benefit).
   - Prednisolone (1-2mg/kg q 12h) decreases resorption of calcium from bone, decreases gastrointestinal absorption of calcium and promotes renal excretion of calcium.
   - Furosemide (2 mg/kg IV q 8h) decreases sodium and chloride reabsorption in the kidney leading to enhanced Ca excretion. Thiazide diuretics should not be used; they are calcium-sparing.
   - Phosphate binders (aluminium hydroxide) reduce intestinal phosphate absorption and should be administered at 30 to 90 mg/kg/day with meals and a low-calcium, low-phosphorus diet (Hills k/d) should be fed (generally four weeks).

4. **Intravenous lipid emulsion therapy (IVLE):**
   IVLE is used to treat many lipophilic drug intoxications.. IVLE has been shown to reduce ionised calcium concentrations in cholecalciferol toxicity. IVLE is a 20% solution and is dosed at 1.5 ml/kg IV over one minute followed by 15 ml/kg CRI over 1 hour. Consecutive treatments with IVLE are often performed 2-4 days after initial administration if the ionised hypercalcaemia worsens. Side effects of IVLE include hypersensitivity reactions, bacterial infection secondary to product contamination, acute lung injury, corneal lipidosis, and fat overload syndrome, characterised by pancreatitis, fat embolism, icterus, or haemolysis.

5. **Bisphosphonate treatment:**
   In a severely affected animal where hypercalcaemia does not respond to treatment, or relapses when fluid therapy is discontinued, treatment with a bisphosphonate is indicated. Bisphosphonates bind strongly to bone mineral, inhibiting osteoclastic-induced bone resorption by blocking the dissolution of hydroxyapatite. They are very effective in decreasing plasma calcium concentrations within 24 to 48 hours, allowing further treatment on an outpatient basis instead of being hospitalised and receiving intravenous saline solution for two to four weeks. Bisphosphonates are very expensive, can be difficult to obtain, and some have been discontinued (such as pamidronate disodium, Aredia, Novartis).

Zoledronate/zoledronic acid (ZA) is reported to have a greater potency and efficacy and can be safely infused over a shorter time in humans. ZA has been used to treat hypercalcaemia in dogs and was reported to be effective, safe and well-tolerated.

ZA is administered at a dose range of 0.13-0.32 mg/kg (typically 0.25 mg/kg) diluted in a 100mL bag of sterile 0.9% saline and administered IV over 15 minutes at intervals of 4 - 6 weeks. Side effects reported are hypocalcaemia, hypophosphataemia, and hypokalaemia. Acute renal insufficiency has been reported in humans at an incidence of 10-15%.

Clodronate has also been used to lower serum calcium concentrations in dogs with experimentally induced Vit. D_3 toxicity. Dogs were given an infusion of 4mg/kg clodronate diluted in 150ml 0.9% saline solution 24 hours after Vit. D_3 administration and compared to the control group of dogs who only received 150ml saline infusion. The serum calcium, phosphorous, urea, and Ca to Phos values were significantly lower compared to the control group on days 4, 7, and 12 after administration.

General care:

Cholecalciferol toxicity also causes gastrointestinal signs and antiemetics (maropitant, metoclopramide) and gastric protectants (H2-blockers, omeprazole, sucralfate) should be administered as needed.

Continuous monitoring:

Frequent monitoring of renal parameters and electrolytes are required. Serum calcium, phosphorus, blood urea nitrogen, creatinine, and ionized calcium should be

evaluated every 12-24 hours during hospitalisation, and then every 2-3 days thereafter for the next 2-4 weeks to allow for appropriate treatment adjustment. Even with intensive decontamination and therapy, chronic kidney disease can occur.

Treatment of cholecalciferol toxicity is expensive and requires hospitalisation for an extended period (e.g., 2-7 days to weeks). In hypercalcaemic patients threatment with furosemide and prednisone may to be continued for several weeks following discharge from the hospital.

Cholecalciferol rodenticide toxicity carries a poor prognosis once clinical signs and azotaemia develop due to the high risk of chronic kidney disease.

Acute toxicity with decontaminated and prompt treatment has a more favourable prognosis as soft tissue mineralisation hasn't occurred yet.

Soft tissue mineralisation is minimally reversible and leads to structural damage and decreased function of the renal, cardiovascular, gastrointestinal, and musculoskeletal systems.

Clients should be informed of the guarded prognosis for cholecalciferol toxicosis, as well as the financial implications and commitment required for treatment.

Table 4 – Differentials for hypercalcaemia

Causes:
Laboratory error
Young dogs
Latrogenic (Thiazide Diuretics)
Humoral Hypercalcaemia of malignancy (Lymphoma, multiple myeloma, anal sac adenocarcinoma, certain carcinomas e.g. thyroid, thymoma, rarely bone tumours).
Hypoadrenocorticism
Chronic renal failure
Primary hyperparathyroidism
Pseudohyperparathyroidism
Feline idiopathic hypercalcaemia
Fungal disease
Osteomyelitis
Ingestion of human prescription skin products containing Vit. D analogues (calcipotriene, tacalcitol)

Practical tips on management of suspected rodenticiade toxicity

Dr Liesel vd Merwe BVSc MMedVet(Med)

Many Vitamin K antagonism cases may present afterhours or you are unable to send off blood to test PT/PTT function. Typically these patients present with hypvolaemic shock and evidence of internal haemorrhage - if you look for it.

1. Perform a diagnostic centesis:

   Even though the patient has a bleeding tendency, it is essential that you perform a diagnostic abdominocentesis or thoracocentesis to confirm your suspicion of intra-cavitatory bleeding. You will not worsen the bleed as long as you avoid caudal rib borders where the costal blood vessels run.
2. Blood drawn from central circulation will not clot: Draw blood from the cephalic vein for PCV and to evaluation clotting time. You can leave the blood in the syringe or place it in a serum tube. If the blood you have drawn has not clotted within 5 minutes – generally by the time I have drips in and PCVs done - it is likely you have a coagulopathy. Remember that blood drawn from a haemothorax will not clot
3. Compare the PCV of both the fluid as well as the central circulation.
   - If the PCV of the fluid and the blood is almost the same you have active acute haemorrhage.
   - If the PCV is higher in the fluid than in the blood, the bleeding is subacute as the plasma component is already being reabsorbed into the vasculature to maintain volume. These patients are generally more stable.
   - If the PCV of the fluid is lower than the blood PCV, then you have a fluid effusion with additional haemorrhage. Think neoplasia .
4. If you suspect poisoning but are not sure or you have caused emesis but still want to ensure the patient is not at risk, perform a PT test about 3-4 days post exposure. The citrate sample has to be collected carefully and at the correct ratio.
5. You cannot, as a safety net, start the animal on Vitamin K BEFORE testing the PT /PTT as this medication will normalise the results.
6. You HAVE to be PRO-ACTIVE if you suspect cholecalciferol poisoning. the effects are immediate and appropriate treatment is essential. Even if you are not sure, rather err on the side of caution and start the treatment with saline , prednisolone and furosemide until you can confirm or exclude the diagnosis.

References avaialbe on request

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1. Which one of the procedures below from the management of Vit K antagonism toxicity is CORRECT?

a. If the ingestion is unconfirmed and may have occurred during the night, induce emesis.
b. If unsure if ingestion has occurred, but is suspected from night before, send blood away for PT/PTT test.
c. If unsure if ingestion has occurred but is suspected from the night before, wait another 2-3 days before sending blood away for PT/PTT test.
d. If unsure if ingestion has occurred but is suspected from the night before, wait another 2-3 days before sending blood away for PT/PTT test, but initiate Vit K_1 treatment in case.
e. If unsure if ingestion has occurred but is suspected from the night before, start treating with Vitamin K without testing PT/PTT.

2. Both cholecalciferol and anticoagulant toxins are lipid soluble. Which one of the following statements is thus INCORRECT?

a. Lipid soluble drugs/toxins may go through an enterohepatic cycle.
b. The administration of activated charcoal is not effective in treating lipid soluble drug intoxications.
c. Intravenous lipid emulsion is effective in removing lipid soluble agents from the bloodstream.
d. Administration of a fatty meal will aid in the decontamination of the GIT.
e. Lipid soluble drugs or toxins accumulate in the liver and fat.

3. Which one of the following statements regarding anticoagulant rodenticides (ARs) and cholecalciferol is INCORRECT?

a. Resistance has developed to many first generation ARs.
b. Multiple feed baits decrease the incidence of bait aversion.
c. Second generation ARs are single feed baits.
d. Rodents have a specific sensitivity to the effects of cholecalciferol.
e. Cholecalciferol containing baits have increased negative environmental residual effects.

4. Which one of the clinical findings listed below is most likely to be present in a patient with clinical signs of anticoagulant rodenticide toxicity?

a. Petechial haemorrhages
b. A very low platelet count
c. Evidence of hypovolaemic shock
d. Melaena
e. Haematuria

5. Which one of the following statements is CORRECT?

a. Vitamin K is essential for the production of Vitamin K dependent clotting factors.
b. Vitamin K is essential for the activation of Vitamin K dependent clotting factors.
c. PTT test is very specific for the Vitamin K dependent clotting disorders.
d. A properly filled fluoride sample is required for a PTT test.
e. Circulating blood levels of clotting factors will maintain normal coagulation for 2 days.

6. Which one of the treatment options listed below for AR toxicity is INCORRECT?

a. Emergency rapid IV injection of Vitamin K_1
b. Whole blood transfusion if haemorrhage is occurring into a body cavity
c. Fresh frozen or frozen plasma transfusion if less severe signs of bleeding are present.
d. SQ Vitamin K injections in addition to the administration of blood products
e. SQ Vitamin K injection if only mild bleeding observed.

7. Cholecalciferol has been added to ARs. Which one of the following effects of cholecalciferol below is INCORRECT?

a. Rodents have a very low tolerance for cholecalciferol.
b. Little feeding on the bait is required.
c. Hypercalcaemia develops if ingested by a dog.
d. Very little effect on raptors eating poisoned rodents.
e. Hyperphosphataemia develops if ingested by a dog.

8. Which statement below regarding cholecalciferol toxicity in dogs is INCORRECT?

a. Soft tissue calcification of proton pump rich organs occurs.
b. Acute kidney failure develops within 48 hours.
c. Poisoning in dogs cannot be effectively treated .
d. Prognosis is guarded once signs of uraemia are evident.
e. Early preventative treatment is essential.

9. Which one of the following medications is NOT used to manage the hypercalcaemia caused by cholecalciferol toxicity?

a. IV fluids
b. Furosemide
c. Glucocorticoids
d. Phosphate binders
e. Insulin

10. Which one of the following listed below is NOT a differential for hypercalcaemia?

a. Malignancy
b. Increased nutritional intake of calcium
c. Fungal infections
d. Hyperparathyroidism
e. Feline idiopathic hypercalcaemia

Corticosteroids, Apoquel and Cytopoint:

All effective for Canine Atopy but not all without side effects

Margaret Gober and Andrew Hillier.(2023) Perception and usage of short-term prednisone and prednisolone in dogs. BMC Medical Research. Vol 19:91
Summarised and worked on with permission from Dr Andrew Hillier
ANDREW HILLIER, BVSc, MANZCVS (Canine Medicine), Diplomate ACVD
Director –Dermatology Medical Strategy & Zoetis Specialists,
Petcare Veterinary Professional Services.andrew.hillier@zoetis.com

We as veterinarians need to make rational treatment plans for our patients. We thus need to ensure our clients have all the information they need to make treatment choices. We also then need to be able to adjust our patient care according to the choices the client make. There is not one recipe which fits all.

Corticosteroids are widely used with a low incidence of reported side effects and a general level of confidence in the hands of most veterinarians. Maybe too much.

With a low side effect reporting level of <5% in the UK and high level of comfort there may be complacency and underestimation of the impact side effects of corticosteroids may have on a pet and pet owner.

This 2023 study by Grober and Hillier hypothesized that dogs receiving anti-inflammatory doses of prednisone or prednisolone (n=45 in this study) would experience an increased incidence of side effects day 14, earlier than was currently reported.

Synthetic glucocorticoids like prednisone/prednisolone are considered to have an intermediate duration of action, based on the concept of their biologic half-life of 12 -36 hours. This refers to the time for the effect, not the plasma half-life, to decrease by half. This characteristic makes these products appropriate for alternate day dosing.

Anti-inflammatory dosages for dogs are recommended at a range of 0.5–1mg/kg one to two times a day with a tapering dosing regimen. Even within 7 days of using anti-inflammatory doses (0.55/kg daily) of prednisone, the hypothalamic–pituitary–adrenal axis is suppressed. Glucocorticoid receptors are found in almost every cell type, thus exogenous steroid administration may affect multiple body systems.

While it is estimated 3–15% of dogs have atopic dermatitis, pruritic dogs represent up to 30% of dogs presenting to the veterinarian for skin disease.

The anti-inflammatory effects of corticosteroids have led to their use in dogs when underlying allergic dermatitis is suspected, but they often have side effects.

While corticosteroids have broad anti-inflammatory effects, many veterinary textbooks.Many of these adverse effects focus on months or years of usage. However, the impact of short-term use is not published. In this study, pet owners were asked about the behaviour and clinical signs of their dogs on day 5 and day 14 after starting anti-inflammatory doses of prednisone/prednisolone. A final questionnaire was completed on D30 post initiation of treatment.

Veterinarians prescribed corticosteroids for the immunomodulation of inflammatory conditions, not immunosuppression. Initial administration of the corticosteroid could be BID or SID and there could be discretionary dose reduction to SID or alternate day treatment. All dogs in the study received some tapering of the dose.: either number of doses per day or mg/kg per day.

- At day 5, 97% of pet owners reported the prednisone/prednisolone controlled the condition for which it was prescribed. Pet owners reported general satisfaction with prednisone/prednisolone. For pet owners who were neutral (5) or unsatisfied (3), 7 of the 8 owners (26% of our total population) reported polyuria and polydipsia, with one also increased aggression and two reporting negative behaviour with other dogs and people.
- Pet owners gave corticosteroids an overall satisfaction rating of 4.5/5 on day 14, showing an overall appreciation of improvement of the clinical and reduction of the associated discomfort
- On day 14, 90% of owners (28/31) also reported at least 1 change in their dog's behaviour, including polyuria, polydipsia, polyphagia, and/or polypnea as the most common changes noted.

This study confirms the hypothesis that usage of prednisone/prednisolone changes the behaviour of the majority (81%) of dogs as soon as day 5 after treatment with an antiinflammatory dose is initiated. Published guidelines have shown the benefits of corticosteroids when used for treatment of canine atopic dermatitis. Adverse effects of oral corticosteroids are normally considered to be proportional to drug dosage and duration of administration. In this study, 39% of dogs received initial doses less than the anti-inflammatory range of 0.5–1.0 mg/kg, but side effects noted by the pet owners were still reported regardless of this low and short duration dose.

Despite being on an "adverse effects" type study, (or maybe because of it) only two (6%) owners contacted their veterinarian during the study period to discuss changes in their dog's behaviour. This means 94% of our pet owners did not report any changes in their dog's behaviour to their veterinary clinic.

As veterinarians we should still want to know what side effects were occuring, how severe they were and how the dog and owner were affected. This reporting and information is dependent on client communication and education. Side effects a client may just accept; weakness or obesity, may really be an unacceptable side effect clinically or medically and require treatment changes.

At the conclusion of the study, 23 pet owners completed a single final 30 day post study question with 70% (16/23) indicating they would select another product with fewer side effects even if the product cost more.

This is an important point as we as veterinarians often take this decision out of our clients hands what they can manage or afford or use what we are familiar with.

Discussion of treatment options with a client is vital. When the prednisone/prednisolone was initially dispensed, 74% (23/31) of the veterinarians recommended prednisone/prednisolone but also discussed other treatment options with the pet owners, allowing the pet owner to make the final selection. For 10% of pet owners (3/31) the veterinarian initially recommended an alternative therapy to prednisone/ prednisolone, but the pet owner specifically requested the use of prednisone/prednisolone and the veterinarian conceded. For the remaining 16% (5/31), the veterinarian made the selection of prednisone/

prednisolone for the pet without discussion with the pet owner.

In a study (Apoquel Chewable US Launch Strategy, Zoetis Services LLC, February 2023) pet owners said that improving their dog's quality of life was #1, while having a reasonable price of medication was only the sixth-ranked factor.

The limitations of this study include a small sample size and the potential bias associated with solicited pet owner reporting including the possibility of more active monitoring of perceived adverse drug effects. In addition, many of these pets had received steroids as treatment in the past and pet owners had their own expectations and comfort level regarding the prior treatment.

We have no alternative but to use corticosteroids.....

Dr L van der Merwe BVSc MMedVet(Med)

So while clients will accept corticosteroids when offered, many are prepared to pay for a medication as effective but with less side effects, even those which occur on the tapered dose. These medications include Apoquel® and Cytopoint® as the most effective product on the market in controlling the clinical signs of canine atopic skin disease.

However, that said, these drugs are only part of a full skin management regimen as these patients are still prone to flare ups and increased incidence of superficial skin infections.

Many pet owners are not financially able to afford the gold standard of medications for their allergic pet. In this situation the veterinarian has to take a practical and holistic look at the patients quality of life. A dog with severe dermatitis is not a happy dog. They experience a decreased quality of life if their clinical signs are moderate to severe and if they also develop otitis externa their discomfort worsens.

Knowing that prednisone/prednisolone treatment causes side effect, it is up to the veterinarian to use the medications together with a holistic treatment plan, in order to minimize the dose and frequency of the medication, thus minimizing side effects .

The following must be considered:

- Diet: Omega-3- fatty acids
- Superficial skin infections:
  - Bacterial: Staph. pseudointermedius
  - Fungal: Malassezia dermatitis
- Flare ups: superficial infection or (seasonal) re-exposure to allergens.

Prednisone/prednisolone:

- Start treatment at 0.5 – 1 mg/kg (20 mg/m2)OID.
- The bigger the dog (> 20 kg) the lower the dose - Body surface area tables - 10 kg = 0.469m^2, 20 kg = .744m^2, 31kg =0.997m^2 and 40 Kg = 1.18m^2 only).
- Large breed dogs may develop rapid severe muscle atrophy - hence the much lower doses used.
- Taper over several weeks to a maintenance dose of 0.2 -0.5 mg/kg every every 2-3 days.
- If this can be achieved then management of the atopic dermatitis should be with minimal side effects. This is however always based on the individual patient.

Shampoos

Most flare-ups do not require a higher dose of prednisone or increased dosing frequency as they are usually due to a secondary superficial infection . Some patients do however get seasonal flareups due to allergen exposure.

Use the best quality shampoo the client can afford. Chlorhexidene shampoos are very effective against S. intermedius. A well formulated shampoo also stays on the skin longer in-between washes. Washing every 3 days with a 10 min leave-in time over 2 weeks will resolve a superficial pyoderma and often control flare-ups. Malaseb® is also effective against malassezia skin infection.

A sellotape test is essential to rule out a concurrent Malassezia dermatitis in atopic patients. If present it requires a short course of antifungal medication to get under control. These superficial skin infections are secondary. There are there are inherent weaknesses in the skin of a patient with atopy so despite effective itch control corticsteroids, Apoquel® or Cytopoint® infections will still occur.

Skin barrier function:

Skin health is best addressed by diet, especially a diet rich in omega -3 fatty acids which will decrease the pro-inflammatory response of the cells in the skin and link the keratinocytes more tightly. This is a long term management approach and the effect will only be seen after 2-3 months. Although less effective, if a client cannot afford a supplemented diet then supplementing the dog with omega-3 capsules (1 x 1000mg/10 kg) daily may be of benefit.

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The Crux of Canine Colour Genetics

Colour genetics is an interesting topic to explore due to the complex interactions which produce various coat colours and patterns. By gaining a better understanding of the mechanisms behind coat colour inheritance and their genetic factors we can see how these coat patterns arise. Equipped with this knowledge, one can better select animals for these coat colours and patterns, without comprising the health of our canine companions.

All observable physical attributes are encoded by genes, which are small DNA segments located at specific places in the DNA sequence, called a locus. These genes have different forms, called alleles, and can be either dominant or recessive. This in turn determines whether one or two copies of the allele are needed to produce a specific outcome. Dominant alleles only need one copy to confer a trait, whereas recessive alleles need two copies.

Several genes are associated with canine coat colour and control the expression of two forms of pigment, eumelanin and phaeomelanin, each of which has a core colour which can be modified by various genes.

The core colour of eumelanin is black, which can be modified to brown or grey, while phaeomelanin is red, which can be modified to cream, yellow or tan. Both eumelanin and phaeomelanin affect coat colour, but eumelanin also affects the colour of the skin, nose, and eyes.

The genes involved in this process can be clustered into three groups, namely:

- genes that affect colour distribution throughout the coat,
- genes that affect colour shade,
- genes controlling coat patterns or markings.

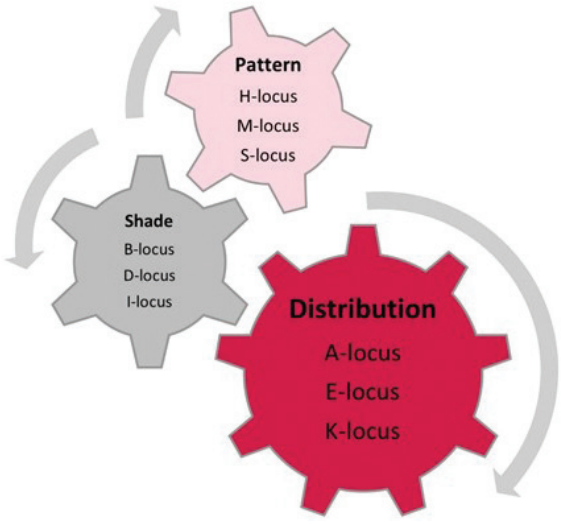

Figure 1 Canine Coat Colour Clusters

Distribution

The E-locus gene controls which areas of the coat produce eumelanin or phaeomelanin, i.e., how far the coat colour "extends". Specific forms of E-locus can produce a normal extension of the coat colour (EE/Ee), masking (EMEM/EMEm), or recessive red/yellow (ee). Normal extension means that the dog can produce eumelanin or phaeomelanin in all areas of the coat, depending on what the A- and K-locus is. Figure 2 shows the mechanisms of the E-locus.

The A-locus gene is responsible for the regulation of melanin production and the distribution thereof throughout the coat. Distribution is controlled by genetically switching E-locus on and off, alternating between activating eumelanin and phaeomelanin production. This results in the agouti coat type, distinguished by hair strands having bands of two or more colours, and can also produce fawn/sable coat; wild-type agouti; tan-points or recessive black. Figure 3 shows the mechanisms of the A-locus on the E-locus.

The K-locus gene controls the expression of "dominant black", with two confirmed alleles, namely KB (dominant) and KY (recessive). The KB allele prevents the A-locus from activating the E-locus, blocking phaeomelanin production, thus only eumelanin can be produced. However, if two copies of the KY allele are present, A-locus functions normally. Figure 4 shows the mechanisms of the A- & K-loci on the E-locus.

Shade

The B-locus gene is responsible for black and brown pigment (eumelanin) expression, and the B (dominant) and b (recessive) alleles determine the colour. Dogs with either one or two copies of the dominant allele (BB or Bb), will have black coats, but dogs with two copies of the recessive allele (bb) will have brown coats. The same will apply for the nose, eyes, and paw pads. The D- (dilution) and I-locus (intensity) genes are responsible for diluting or reducing the intensity of eumelanin or phaeomelanin, respectively. Only dogs which inherit two copies of the recessive allele for D- and/or I-locus (dd or ii) respectively, will produce coats with a diluted/lighter base colour. The D-locus will also affect the colour of the nose, eyes, and paw pads. Figure 5 shows an example of how the D-locus interacts with the B-locus to produce various shades of colour.

Patterning

The M-locus (merle) gene is responsible for merle coat patterning, observed as irregularly shaped patches of diluted and solid pigment, affecting the eumelanin. Interestingly, Harlequin, a coat pattern in Great Danes, which presents similarly to merle, is not controlled by the M-locus but the H locus. The S-locus gene determines the distribution of white spotting, with several variations on the degree of spotting. The dominant form of the S-locus allows full expression of colour, whereas recessive forms of S-locus

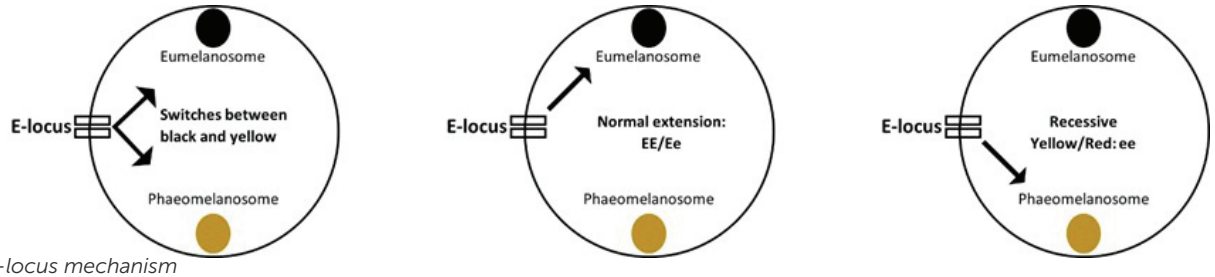

Figure 2 E-locus mechanism

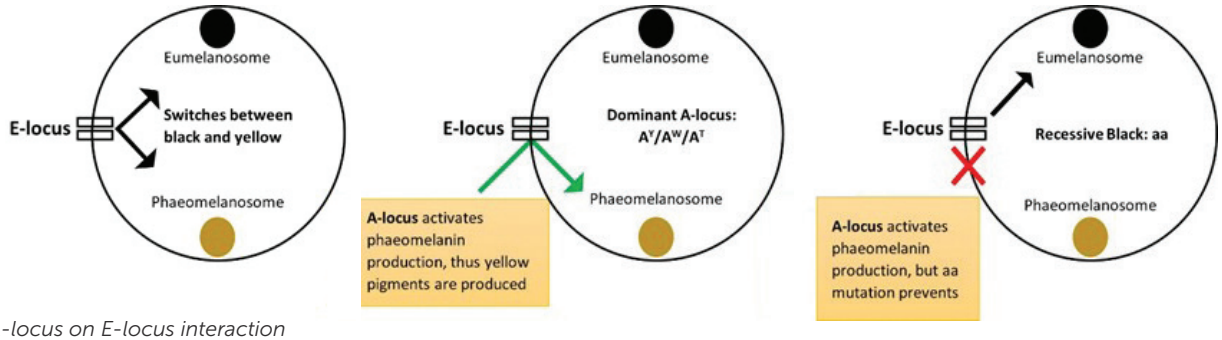

Figure 3 A-locus on E-locus interaction

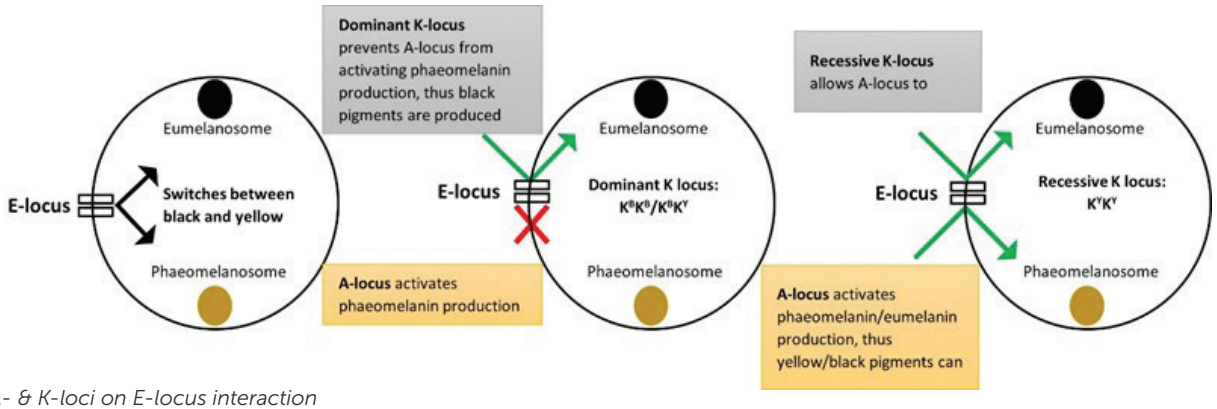

Figure 4 A- & K-loci on E-locus interaction

allow for degrees of white spotting. The most extreme form produces extremely large white areas or completely white coats. The S-locus should not be confused with C-locus, which is responsible for a form of albinism, resulting in a lack of pigment production, and can be distinguished from S-locus by their nose, eyes, and paw pads.

It is important to note that some coat colour genes, namely D-, M- & C-locus, are associated with potential health complications, such as dilution alopecia, or ocular and auditory abnormalities. The consequences of these complications must be considered when breeding with dogs who carry or express these coat colours. It is essential to understand the basic mechanisms and interactions of the genes which control coat colour and pattern, as this allows one to make strategic breeding choices and combat potential associated health concerns, ensuring a long and happy life for our canine companions.

Where can I get genetic health tests done?

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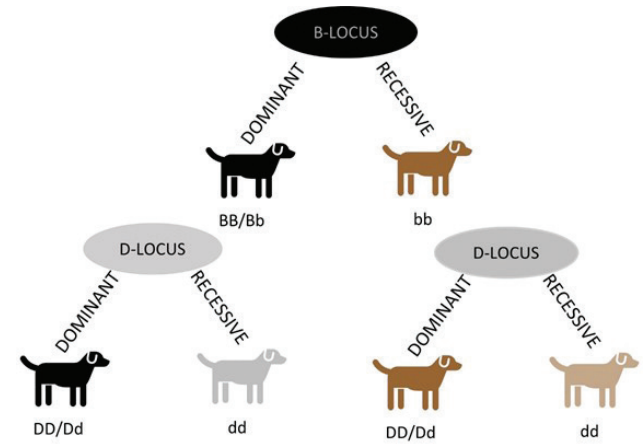

Figure 5 B & D locus interaction

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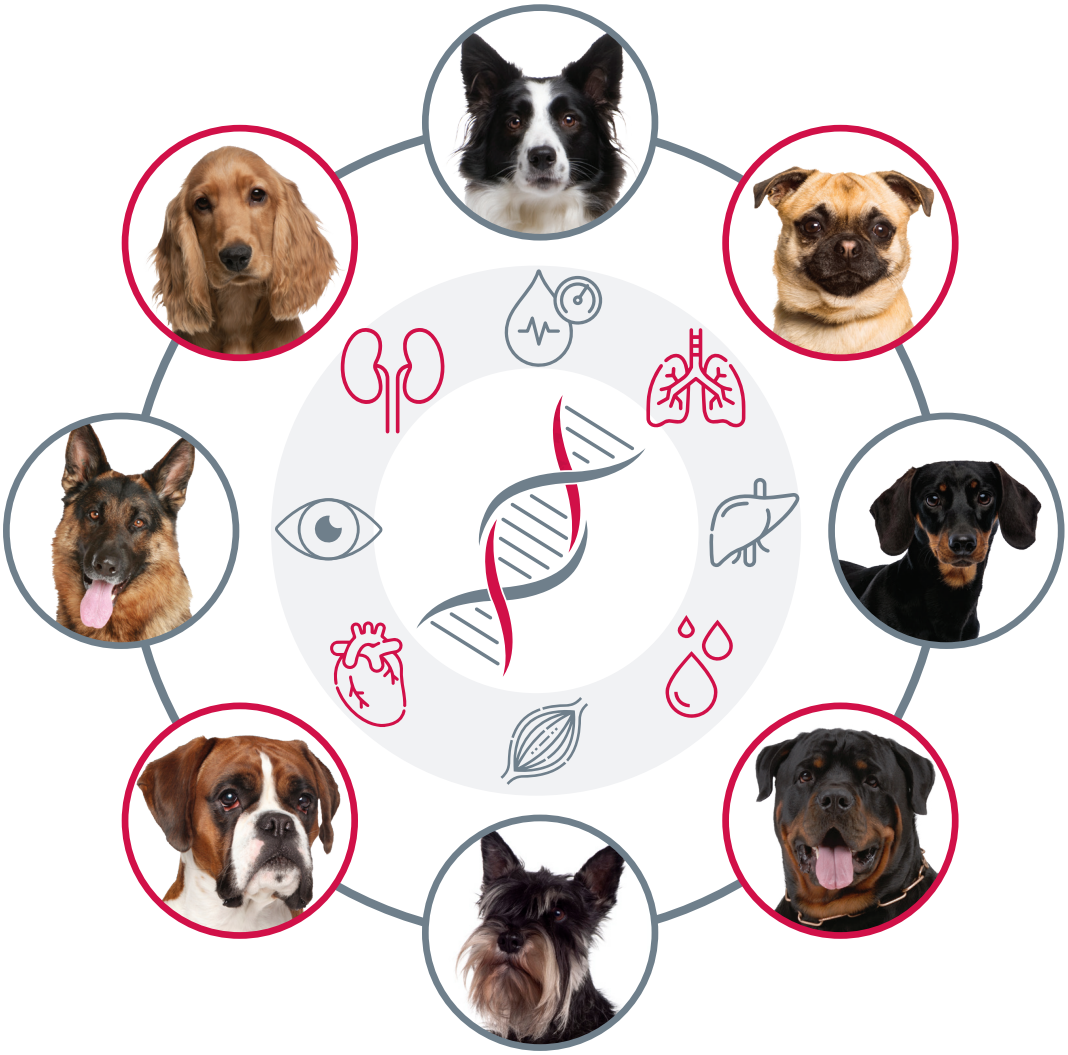

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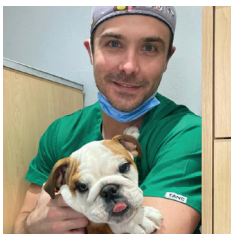
Surgical Oncology – Essential Guidelines for General Practitioners



Dr Christiaan F Triegaardt
Small Animal Surgeon (Cape Specialist Surgical Vet)
christiaan@cssv.co.za

We are treating the individual, not the population. By adhering to sound principles we can offer our patients the most effective and compassionate care in their fight against cancer.

Cancer remains a leading cause of death in dogs over 10 years old, accounting for 40-50% of deaths. As a general practitioner managing oncological cases, adhering to fundamental principles in surgical oncology is paramount for effective tumour management. In this article we outline key steps to approach surgical oncology cases more effectively.

There are three questions we need to address:

1. What is it?
2. Where is it?
3. How bad is it?

1. WHAT IS IT?

The cornerstone of any oncological case begins with a biopsy. Obtaining a definitive diagnosis is crucial as it guides treatment aggressiveness, prognosis, and client costs. Biopsy techniques include fine needle aspirates, incisional biopsies (such as punch, Tru-cut, wedge, and endoscopic), and excisional biopsies. Each method serves specific purposes:

- **Fine Needle Aspirates (FNAs):** This is the least invasive and cheapest method and is suitable for most and cheapest, suitable for example in mast cell tumors (MCTs), lipomas, and histiocytomas. FNAs can be challenging with soft tissue sarcomas (STS) due to their less exfoliative nature. Cytology sets the platform for further investigation.
- **Punch Biopsies:** Effective for superficial lesions but ineffective with deeper tissues. Suitable for intestinal biopsies. Recommended punch sizes: # 2 for cats, #3 for dogs.
- **Wedge Biopsies:** This is considered the optimal choice among biopsy techniques.
- **Excisional Biopsies:** Should be reserved for specific cases to avoid compromising curative intent surgery. Specific cases include:
  a. Disease conditions where all differentials are treated similarly (e.g., bleeding splenic mass, lung mass, bone tumor).
  b. A mass on a digit where a marginal excisional biopsy can be performed. If malignant, the digit can be amputated with clean margins; if benign, the issue is resolved.
  c. Areas with ample free skin where the owner simply wants the mass removed.

Don't be that that vet...

A very dangerous logic circulating in surgical oncology is: "Remove as much as I can and see."

If you don't have a diagnosis, remove as little as possible and avoid disrupting the surrounding tissue. The surrounding tissue may contain tumour cells and the peripheral tumour cells are often the most aggressive and active.

Disrupting these cells can lead to a significantly larger resection due to a contaminated scar (Fig. 1). The scar effectively becomes the new tumor, necessitating clear margins that may require a much wider and deeper excision, possibly reaching the body wall. Additionally, radiation therapy might be required to "mop up" the residual disease.

Dirty margins increase patient morbidity, can be very costly for the client, and might compromise the ability to cure the patient.

"NEVER BE SURPRISED BY YOUR RESULTS; EXPECT THEM."

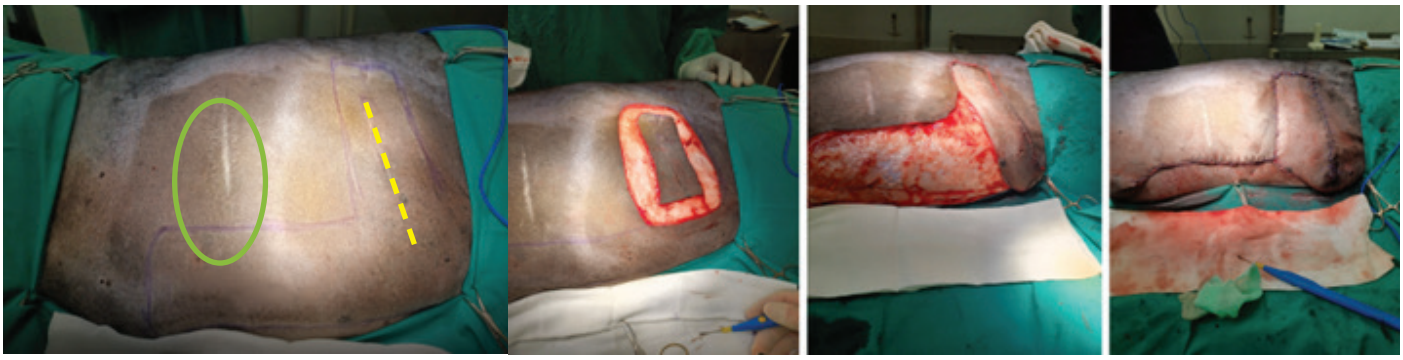


Fig 1: Example of a scar) with clean- (green oval, -and-dirty (dashed yellow line) margins and the extent of the surgery requiring a caudal superficial epigastric axial pattern flap, which follows (Photo credit: Dr CF Triegaardt, Johannesburg Specialist Veterinary Centre 2022.)

2. WHERE IS IT?

Staging is important to determine if the tumour is local, regional, or distant using the TNM system (Tumour, Lymph Node, Metastasis). The tumour type generally provides insights into its behaviour (or = american; our = UK) characteristics.

i. Lymph nodes

Regional Lymph Nodes (RLN) drain a certain region. Sentinel Lymph Nodes (SLN) are the first lymph node to which cancer spreads. Normal palpation of size and shape does not guarantee the absence of metastasis. Up to 50% of normal sized lymph nodes have neoplastic metastases. Fine needle Aspirates: Negative fine needle aspirates do not rule out metastasis.

ii. Sentinel Lymph Node Mapping:

The reginal Lymph node (RLN) is not the sentinel lymph node (SLN) in up to 60% of cases. Mapping is a crucial but underutilized concept in practice. The lymphatic drainage regions are called lymphosomes and are relatively typical per species. See Figure 2 below for more anatomical and flow direction details.[4] These maps are utilised to estimate to which lymph node region drainage is occurring.

Methods of finding /mapping the SLN include radiographic, CT lymphography, Lymphoscintigraphy and the use of dyes (Methylene blue) or Indocyanine green (ICG).

ICG has multiple applications in medicine but with regards to mapping it is a technique where a medical dye is injected peri-tumourally and the SLN and draining pathways can be visualised with a special lens as it fluoresces green under a certain light spectrum. This is, however, a very costly procedure due to special equipment required.

There is a cheaper in-house alternative that can be utilized, called indirect lymphography.[1]

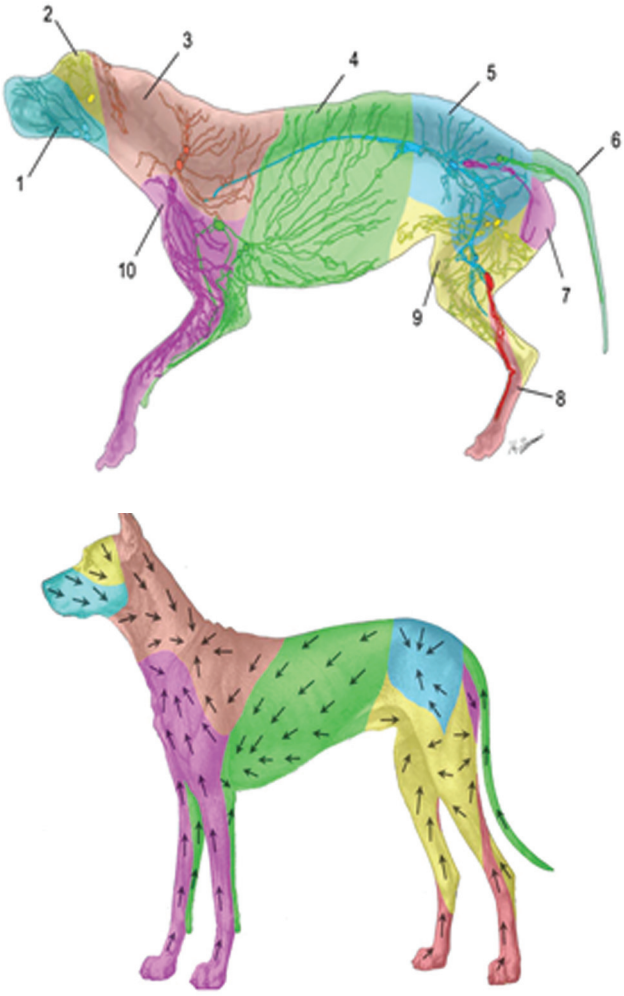


Fig 2: Lymphosomes in a canine.
Colour coded diagram of the lymphatic territories (lymphosomes) with lymphatic vessels shown distally from their corresponding lymphnodes: 1 – submandibular, 2 – Parotid, 3 – dorsal superficial cervical, 4 – Axillary, 5 – medial Iliac, 6 – lateral sacral, 7 – hypogastric, 8 – popliteal, 9 – superficial inguinal, 10 – ventral superficial inguinal. (Image source: Suami, H., Yamashita, S., Soto-Miranda, M.A. and Chang, D.W., 2013. Lymphatic territories (lymphosomes) in a canine: an animal model for investigation of postoperative lymphatic alterations. PLoS One, 8(7), p.e69222.)

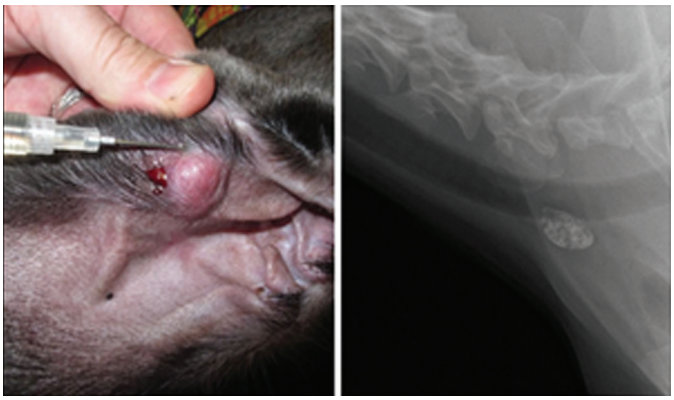


*Figure 3: Peri-tumoural injection with Omnipaque®;Contrast visible in SNL. * note that this is for illustrative purposes only and that the lymphnode does not drain the injected area. Photo credit - Dr Julius M Liptak.animalcancersurgeon.com/principles-clinical-staging.*

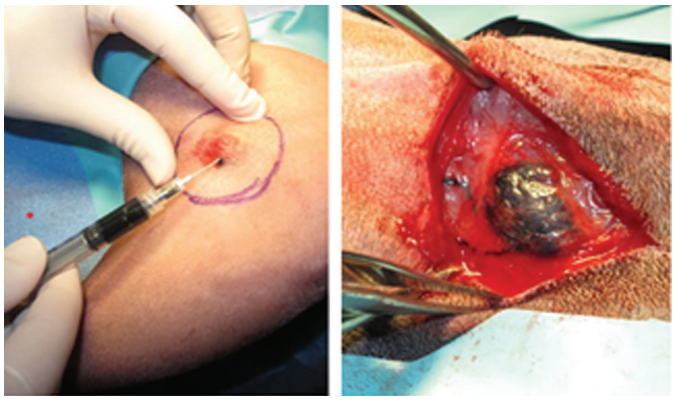


Figure 4: Peri-tumoural injection with methylene blue, land intra-operative appearance of the SLN) Photo credit - Dr Julius M Liptak.animalcancersurgeon.com/principles-clinical-staging.

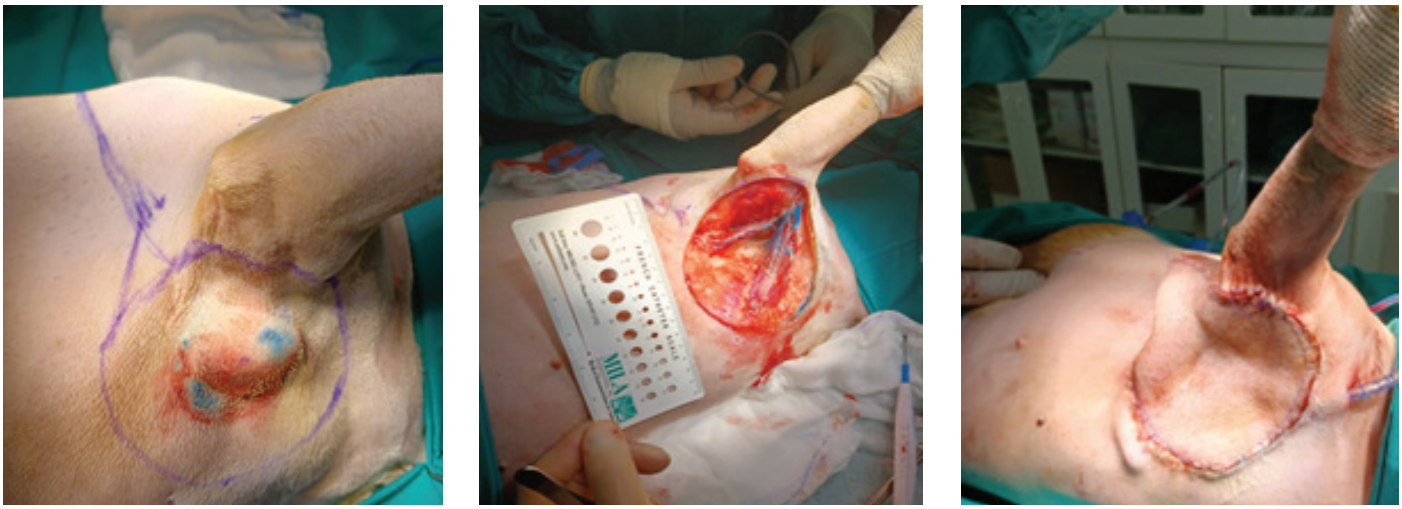


Figure 5: A dog with a mast cell tumour in the axillary region showing the Methylene blue staining the lymphatic drainage. (Photo credit: Dr CF Triegaardt, Johannesburg Specialist Veterinary Centre 2022.

- **Indirect Lymphography (In-House) for radiographic localization:**
  Iohexol (Omnipaque®) water soluble contrast agent is injected in 4 quadrants peri-tumourally under sedation. (3-4ml total/patient). Take regional radiographs immediately and at fixed intervals of 1 min, 3 min, 5 min and 10 min after injection. (Fig 3) .The SLN will take up contrast in 78% of cases and will be visible on radiographs. Intraoperative identification of the lymph node will require good anatomical knowledge.
- **Methylene blue injection for intraoperative detection:**
  Methylene blue is injected peri-tumourally in 4 quadrant technique at a volume of approx. 0.25 - 0.5ml/quadrant (Fig 4,5). Methylene blue can be messy, therefore mark your planned excision margins before with a surgical marker. [1]

Distant metastatic disease is evaluated by taking into account the primary tumour type and their typical patterns of metastases. Imaging plays a pivotal role. Thoracic radiographs can be utilized if no CT is available, but a LR, RL and VD view must be taken (3-view metastatic views) as 10-15% of metastases are missed with only 2 views. The uppermost lung lobe is more inflated with air and masses will show more clearly in that lobe. A CT scan is more sensitive, being able to identify nodules 1mm in diameter versus a minimum size of 7 mm on radiographs. (Fig 5)

3. HOW BAD IS IT?

The malignancy of the neoplasia is determined by grading. Grading is expressed as I, II, III or High vs Low grade depending on the tumour type and grading system. This is where the pathologist plays an integral part in the approach to oncological cases. The accuracy of the information

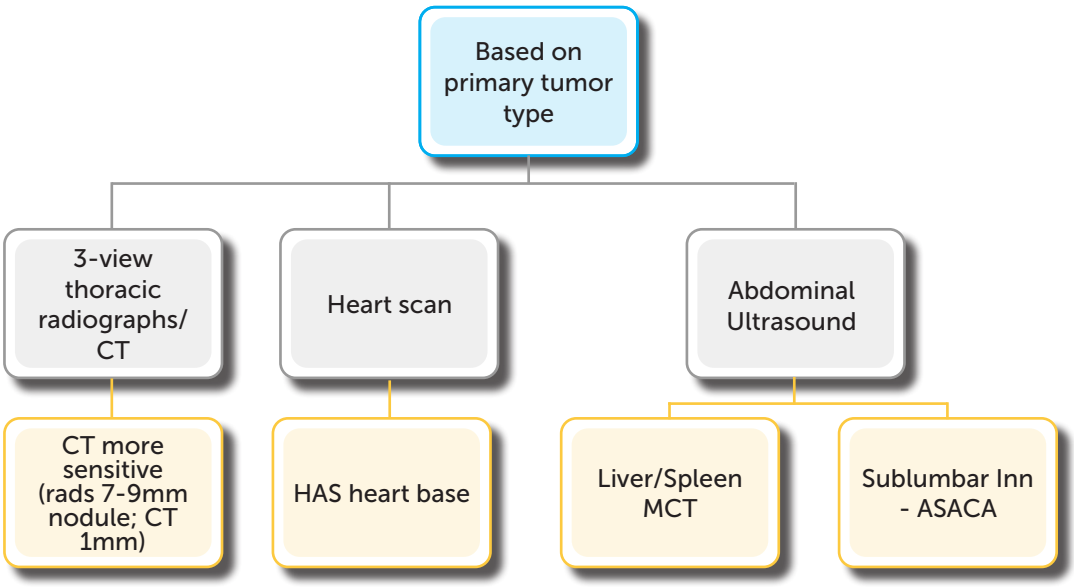


Figure 5: Organogram outlining evaluation of a patient (staging) for distant neoplastic disease.
Key: CT = computed tomography; HAS = haemangiosarcoma; MCT = mast cell tumour; ASACA = anal sac adenocarcinoma

submitted to the pathologist as well as clear inking of margins will improve the information gained. There are many criteria used to evaluate aggressiveness of the neoplasia and more and more tumour markers are being utilized

Treatment Plan:

After answering the three key questions, devise the appropriate treatment plan. The goals are:

- Curative Intent
- Palliative Care
- Euthanasia (considering the quality of life)

The primary aim with surgical resection is to remove all cancer, It is not about making sure the wound can be closed – a wound is rarely fatal, but cancer is (Fig 7). Make sure about your margins on the surface and deep to the tumour. The old saying is still the best saying - Your best chance for full resection is your first chance! Stay away from the tumour - do not incise – you will contaminate your surgical site.

Deep surgical margins are dependent upon tissue layers and fascial planes, not actual measured depth.[3]

Figure 7: Illustration of surgical principles for tumour mass resection.

Surgical dose is a term used to describe the type of resection which will be performed. They are classified as (Fig 7) :

- intracapsular/ intralesional (basically debulking/ palliative) - incomplete resection inside surrounding pseudo capsule (black line)
- marginal resection - incision is exterior to the surrounding pseudo capsule but still within the reactive zone (yellow).
- wide resection - incision removes both microscopic and macroscopic disease, including at least 1 fascial layer
- radical resection - *en bloc* – of the entire muscle or bone compartment with removal of affected lymphnodes as well

Things to Think About

- **Aggressiveness:** Cells at the periphery of the tumor are the most aggressive. If left behind, the tumor will regrow with a more aggressive population of cells.
- **Reconstruction Considerations:** Plan how the defect will be reconstructed. Consider how the patient needs to be shaved and prepped for a skin flap. Be aware that the hair will regrow in different directions.
- **Avoid "Coning Down":** When you incise deep tissue, address it with the same importance, don't angle the blade inwards, otherwise, the large skin defect is in vain.
- **Sterile Markers:** Use sterile markers to draw margins in a circle, not an oval.
- **Tumour Cell Movement:** Tumour cells move along the path of least resistance, often laterally, stopping at the facial plane or muscle.
- **Changing Gloves/Clean Instruments:** Always change gloves and use clean instruments when tumour margins are penetrated or when elevating a skin flap.
- **Drains:** Avoid Penrose drains due to the risk of tunneling and seeding. Instead, use closed suction drains and exit them very close to the wound margin to prevent far seeding of potential cancer cells.

Sample Handling

- **Mark the Margins:** Use sutures or tissue ink (yellow or black preferred) to mark the margins. Tack the overlying skin to the subcutaneous tumor in the original orientation so that it doesn't twist on its own.
- **Inking Concerns:** Ink the margin you are concerned about and clearly note this in the submission form.
- **Sectioning the tumour:** Section the tumor to partial thickness to improve fixation and reduce necrosis.
- **Formalin Ratio:** Use a 10:1 formalin ratio for proper fixation.

One study estimated that pathologists only examine between 0.01% and 0.1% of the total tumour to decide on the margin.[2] Therefore, it is crucial to guide the pathologists by providing the best quality samples and the most information. The histological margin is the only one that matters, not the surgical margin.

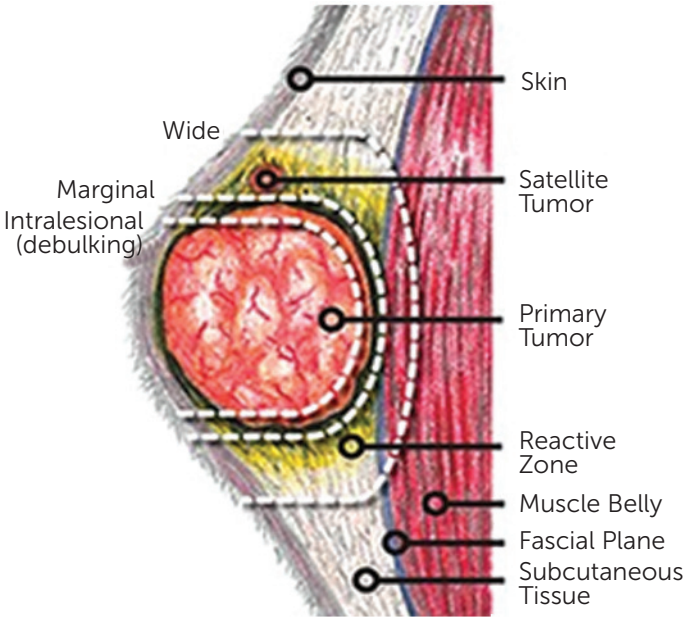


Figure 6: Illustration demonstrating the different tumour resection levels - "Surgical Dose "
Image source: Orencole, M.J. and Butler, R., 2013. Fundamentals of Surgical Oncology in Small Animals. Today's Veterinary Practice, 3(6), pp.14-18.)

In conclusion, for oncological cases, it is important, it is essential to ensure that all options have been thoroughly explored and (Fig 6) that we have provided our patients with the best possible treatment. Expensive equipment is not a prerequisite for high-quality care; instead, a sound approach and dedication to each individual patient are paramount.

Remember, we are treating the individual, not the population. By adhering to these principles, we can offer our patients the most effective and compassionate care in their fight against cancer.

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