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CPD Article

Use of Laboratory Diagnostics in Dermatology for Veterinary Clinicians
Part I: Histopathology

Opthalmology

Approach To a Patient with a "Blue Eye"

Critical Care

Managing South African Snake Bite Envenomation in Dogs

Bee Sting and Massive Attack by Bees in Dogs

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Editor's Note

This edition is full of content by local authors which is my aim as I believe we have enough skilled specialists in this country to create our own content for our own unique situation.

In this issue we concentrate on toxicities - mainly biological toxins and then a short article on the MDR1 gene mutation which causes the sensitivity of collie breeds to ivermectin. This gene affects multiple drugs and it is certainly worth noting which are affected.

The snake bite article has endeavoured to answer specific questions like when is antivenom essential, when can you still give it post admission, compartment syndrome and manual ventilation of patients. The bee sting article provides a nice background to the syndrome and highlights aspects of the anaphylaxis and haemolysis which develops and management options. We also have a nice article on export to EU and Ireland. As vets we need to get these processes accurately administered and websites have been included to allow you to access updated information.

I hope you enjoy your 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.

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Using Technology to Meet Client Expectations

Eric Garcia

For me, technology is a career. But for almost everyone else, it's a way of life. Whereas we might have once considered certain areas of technology, like smartphones, online shopping and FaceTime, to be generation-specific, the data shows this is no longer the reality.

We all now shop online, with 2017's $2.3 trillion in global e-commerce sales expected to grow to $4.5 trillion by 2021. We binge-watch TV via Netflix, send text messages and request Uber rides. Hands reach for the ceiling without hesitation during my lectures when I ask, "How many of you use this type of technology each day?"

Still, despite this growing consensus and our love of tech conveniences, veterinary practices are hesitant to adopt the latest technology solutions. Some practice owners I've met tell me that "mostly millennials use tech" and that millennials are generally broke, so why focus on them?

Well, some of the sentiment is true. Many millennials are broke, having pursued higher education at a much greater cost than what average wages can pay off. We've all read about the rising student debt.

But take note of this: When it comes to pets and pet care, millennials are doling out big bucks. Statistically, millennials are the largest pet-owning demographic, spending an average of $1,285 a year on pet care, with the majority of that figure designated for veterinary care. Millennials play a larger role in the veterinary economy than many practice owners readily acknowledge.

We live in a world where technology is accelerating into our daily lives. We're simply too far along in the process to reverse course, even while governments learn to appropriately regulate behemoths like Google and Facebook. Whether it's Uber, Netflix, Airbnb or another tech solution, pet owners leverage conveniences and apps each day because they make life easier.

I would be remiss not to mention Amazon, perhaps the most notable example of the new technology revolution. Amazon simply provided everything that other retailers didn't, whether it was a better experience, better service or greater convenience. Only when brick-and-mortar retailers started losing revenue did they react, but their responses were too little, too late for many of us.

The Golden Rule

That takes us to the essence of this article: Veterinary practices deprive pet owners of the same conveniences and experiences we demand in our daily lives.

We expect to fetch a quick ride through a ride-hailing app, never dialing a phone number, but we force pet owners to call us when they need something. We also expect them to call us and then drive to our clinics to pick up essentials like food and heartworm and flea medications while we — practice owners, consultants and staff alike — do our personal shopping online.

Similarly, we leverage telemedicine by consulting with our physicians online, but we make pet owners take time off work for simple rechecks and evaluations. We receive texts from businesses of all types, but seldom do we provide the same functionality for pet owners.

Even though there's room for improvement, we need to give our profession credit where it's due. The veterinary industry is outpacing industries once known to excel in client communications. My analysis, experience and research tell me that we're outpacing human dentistry, which used to set a standard for communication. Our industry is changing at a much more rapid pace than even a decade ago.

It's an Online World

Work remains to be done, specifically in areas like telemedicine, online pharmacies and text communication. Many argue that online pharmacy sales pale in comparison to in-person purchases, but we're seeing more online scripts being filled than ever before from places like Chewy and 1-800-PetMeds. Some practices drag their feet amid such change, while others leap toward it. I urge practices to partner with an online pharmacy and promote the option.

Additionally, I urge you to begin texting clients, giving you more intimate, convenient access to the 277 million American adults and teenagers who actively text. (See my article "Keeping in Touch: Texting Pet Owners" at http://bit.ly/2mMCBr6.)

We also need to use more mobile apps — many clients would like instant access to their pet's medical records — and we need to leverage telemedicine, a veterinary advent that is just around the bend.

Let's learn our lesson from Netflix predecessor Blockbuster, the taxi companies and smaller retailers that refused to move toward online solutions and e-commerce. Change is something that can't be prevented or stopped. Instead, it must be embraced.

So, I'll ask one final question to that effect: Would you rather try something once it's too late, or would you rather be an innovator, embracing the new world we live in and offering the best of 21st-century technology solutions to your patients?

To me, the answer is all too clear.

Use of Laboratory Diagnostics in Dermatology for Veterinary Clinicians

Part I: Histopathology

Dr Rick Last – Consulting Specialist Veterinary Pathology

Introduction

In companion animal practice, diseases involving the integument account for around 40% of consultations. In dogs, pruritus is the most common presenting sign, while in cats, cutaneous swellings account for most presentations. Ectoparasites and bacterial pyoderma are the most common diagnoses in companion animals, while hypersensitivity dermatitis and cutaneous neoplasia are also frequently diagnoses in dogs.

The skin acts as a primary protective barrier between the body and its environment. Essential to the maintenance of this skin barrier function is the lipid matrix in the stratum corneum. This lipid matrix prevents excessive water loss through the epidermis and prevents environmental compounds permeating into the viable epidermal and dermal layers provoking an immune response which leads to inflammatory skin disease. The composition of the stratum corneum lipid matrix is dominated by three lipid classes: cholesterol, free fatty acids and ceramides. These lipids adopt a highly ordered, 3-dimensional structure of stacked densely packed lipid layers (lipid lamellae). Arrangement of these lipids depends on the composition of the lipids. Changes in the stratum corneum lipid barrier with loss of epidermal barrier function is a central pathogenic mechanism in the development of atopic dermatitis.

Skin pH is vital to protection of the skin from surface environmental antigens, infectious agents (bacteria, fungi), contact irritants and excessive moisture loss (dehydration). If skin's pH rises into the alkaline range, its natural balance is disturbed. Essential epidermal lipids (cholesterol, free fatty acids and ceramides) are no longer formed leading to damage to the lipid matrix with loss of epidermal barrier function, the skin loses water and dehydrates. Under these circumstances the skin becomes less resilient and more sensitive to environmental triggers (antigens, allergens, irritants) and more prone to development of infections, hypersensitivity diseases and seborrheic skin conditions.

Histopathology

Histopathological evaluation of skin biopsies forms a crucial and essential part of any dermatological evaluation and is one of the most powerful tools in dermatology, if used correctly. With a close relationship between the clinician and pathologist as regards clinical dermatological features, specimen collection and evaluation, skin biopsies can correctly determine the dermatological diagnosis in a very large percentage of cases. Skin histopathology has a limited range of responses (pattern analysis), so if read in isolation has limitations. If combined with clinical features of age, breed, lesion distribution, seasonality, environment, diet etc, plus ancillary diagnostic analyses, dermatohistopathology becomes highly diagnostic.

Indications for performing cutaneous histopathology

- As part of the diagnostic workup of hypersensitivity dermatoses including ectoparasitic hypersensitivity, environmental allergens both inhaled and contact (atopic dermatitis) and adverse food reaction.
- Ulcerative skin conditions can be associated with infectious agents, vasculopathies, immune mediated skin conditions, "drug reactions", venomous bites or stings or cutaneous neoplasia. In many instances, ulcerative skin lesions are high-risk lesions due to destruction of the protective skin barrier. Secondary infection, septicaemia and toxaemia are common complications, and so the disease process needs to be established to enable immediate therapeutic intervention.
- Where the gross skin lesions are similar for very different aetiologies. For example, crusting and depigmenting lesions restricted to the nasal planum maybe observed with discoid lupus erythematosis, nasal mucocutaneous pyoderma, nasal dermatophytosis, uveodermatological syndrome or epitheliotropic lymphoma.
- When a disease condition with pathognomonic histopathology is suspected. For example, feline plasma cell pododermatitis, canine leproid granuloma, pigmented viral plaques.
- Dermatoses that are not responding to therapy.
- Vesicular dermatoses including pemphigoid skin conditions, autoantibodies damage intercellular desmosomes resulting in formation of acantholytic keratinocytes. Be careful of

an unusual distribution of lesions (pemphigus impersonators with acantholytic keratinocytes) namely bacterial pustular pyoderma (exotoxins damage intercellular junctions), pustular dermatophytosis (proteases damage intercellular junctions), drugs reactions (direct damage intercellular desmosomes) and paraneoplastic pemphigus (cell antigens released from tumour cells induce autoantibody production).
- Where the skin condition suspected requires expensive or dangerous therapy (uveodermatological syndrome).
- Unusual skin conditions (cutaneous protothecosis, juvenile ischemic dermatopathy etc) which are not easily recognized at clinical examination.
- Possible neoplastic lesions. Histopathology allows distinction of inflammatory from neoplastic conditions (eg: feline rodent ulcer from feline sarcoid), classification of neoplastic process as benign or malignant, allows for evaluation of surgical skin margins and can establish evidence of metastasis.

Lesions to be Submitted for Histopathology

Primary lesions (nodules, pustules, papules, bullae, alopecia, depigmentation, scaling, erythema) must be included together with a few selected secondary lesions (crusts, lichenification, exudation, ulceration) and all cutaneous nodules need to be biopsied. If the distribution of lesions for a suspected condition is unusual, they should be biopsied (eg: drug associated pemphigus). Multiple samples of a variety of lesions allow the pathologist to examine the full spectrum of lesions and provide more meaningful interpretive comment.

Types of skin biopsies used for histopathology

- Punch biopsies.
- Full thickness ellipse biopsies.
- Incisional full thickness biopsies.
- Excisional full thickness biopsies.

Indications for punch biopsies

- Skin infection (superficial / deep pyoderma).
- Inflammatory skin disease (allergy, immune mediated, nutritional).
- Hyperkeratotic (crusting) skin disorders.
- Skin pustules and vesicles (pemphigus, bacterial).
- Skin conditions affecting mucocutaneous junctions (oral, eyelid, rectal, vagina, prepuce).
- Nail-bed conditions.

Indications for full thickness ellipse biopsies

- Skin conditions associated with hair loss (alopecia).
- Skin conditions with excessive pigmentation (hyperpigmentation) or loss of pigmentation (hypopigmentation).

Indications for incisional or excisional biopsies

- Large and/or ulcerative skin lesions to allow comparison between normal and affected skin.
- Cutaneous neoplasia / nodules.

Special Precautions

Superficial and Deep pyoderma: histopathology should always be done in conjunction with bacterial culture (aerobic / anaerobic) and fungal culture (dermatophytes, deep mycoses). If an underlying condition is suspected, consider re-biopsy after 3 weeks on antibiotic therapy, as then the histological features of pyoderma would have been removed unmasking possible underlying pathology. A similar approach would apply to a "deep pyoderma" which is not responding to antimicrobial therapy based on an antibiotic sensitivity profile achieved against bacterial pathogens isolated. Punch or ellipse biopsies are suitable.

Inflammatory skin disease: Collection of primary as well as secondary lesions (5 punches) and a single punch from unaffected skin for comparison should always collected. Punch produces a standardized sample and so is the best sample for inflammatory dermatoses. Punch biopsies must only include abnormal tissue, due to the way the tissue is prepared. Clinically normal skin is submitted as a separate punch. Advise drawing blood at the first consult and separating serum for freezing and storage, should further serum tests be considered following histopathology results (allergy serology, clinical chemistry, food reaction test).

Hyperkeratotic (crusting) skin disorders: All of the crust should be included, even the crumbled surface crust, which is not tightly adhered to the underlying skin (Fig. 1). Often acantholytic keratinocytes, dermatophytes or bacteria are restricted to this superficial keratin zone and if this surface crust is not

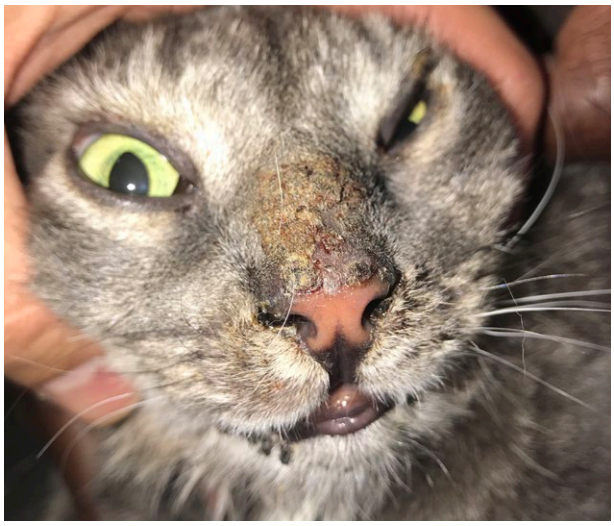

Fig. 1 - Pemphigus foliaceous in a cat with severe crusting of the nasal planum and bridge of the nose. It is important that all of the crust, including crumbling surface, is placed in formalin to give the best possible chance of identifying diagnostic acantholytic keratinocytes (Image courtesy of Sandown Veterinary Clinic Cc, Johannesburg).

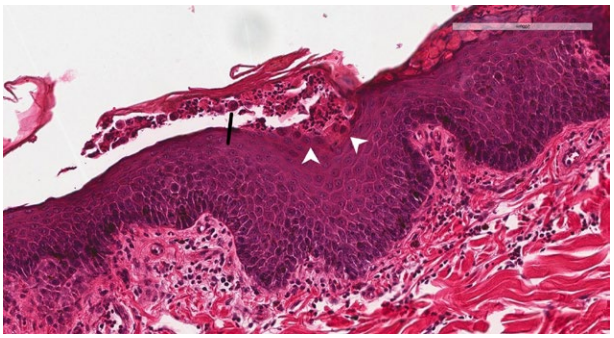

Fig. 2 - Histopathology of a classical pemphigus foliaceous pustule located sub-corneal with acantholytic keratinocytes forming at the base (arrowheads) and exfoliating into the pustule lumen (black line) accompanied by exclusively with neutrophils.

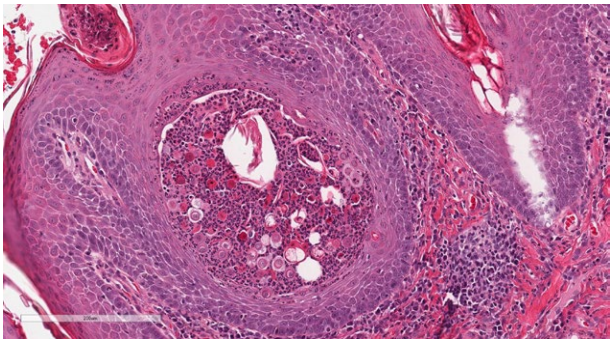

Fig. 3 - Follicular pemphigous with acantholytic keratinocytes restricted to the stratum corneum of the follicular ostia. No acantholytic keratinocytes evident in the surface crust.

processed onto the histological section they can be missed. Punch or ellipse biopsies are suitable.

Skin pustules and vesicles: Intact pustules provide the most meaningful diagnostic histopathology, so wherever possible entire pustules / vesicles should be collected within the punch specimen. Histopathology of sub-corneal pustules containing acantholytic keratinocytes is highly diagnostic and supportive of a diagnosis of pemphigus (Fig. 2). Remember the stratum corneum extends down into follicular ostia so intact sub-corneal pustules containing acantholytic keratinocytes can be observed in superficial follicles (Fig. 3). Acantholytic keratinocytes are formed in waves and usually present in the same area of the crust in biopsy samples, so depending at what level they are at, they can be missed in histological sections as well as cytological smears. In cases where pustules with acanthocytes are restricted to follicles cytological examination of the surface crust is non-diagnostic.

Collection of intact vesicles, bullae or pustules provides the best opportunity for diagnosis, otherwise diagnostic lesions can be easily missed histologically. Since the lesions are so fragile (with rupture and loss of acantholytic keratinocytes), it may be necessary to hospitalize animals for biopsies, so that they can be closely monitored for 2 to 4 hours for the presence of primary intact vesicular lesions. Immunohistochemical staining for anti-species IgG on formalin fixed tissue biopsies is know the preferred diagnostic technique for the diagnosis of auto-immune skin conditions. As IHC labelled slides are counterstained with haematoxylin, the exact anatomical cellular location of positive labelling can be determined. Positive labelling associated with the lesions gives a high level of confidence in the diagnosis.

Mucocutaneous conditions: Histopathology of the of the primary mucocutaneous skin diseases (discoid lupoid conditions, mucocutaneous pyoderma, nasal dermatophytosis, pemphigus conditions, hepato-cutaneous syndromes, uveodermatological syndrome, epitheliotropic lymphoma) can be similar with interface inflammatory infiltrates of lymphocytes and plasma cells at the junction of epidermis and dermis being a consistent feature across the board, except with the hepatocutaneous syndromes (metabolic epidermal necrosis). Therefore, combinations of which mucocutaneous junctions are affected, whether there are any nasal planal lesions and / or pododermatitis (footpad involvement or any other skin lesions, is critical to interpreting the significance of the histological changes and arriving at the correct diagnosis. Depending on the condition/s suspected ensure standard mucocutaneous sites (lip, conjunctiva, rectal, genital) are collected in conjunction with nasal planum (discoid lupoid conditions, mucocutaneous pyoderma, nasal dermatophytosis, pemphigus erythematosus, epitheliotropic lymphoma) and interdigital skin biopsies (mucocutaneous DLE, hepatocutaneous pyoderma).

Nailbed conditions: Collection of biopsies that includes the subungual site provides the best diagnostic information for both the pathologist and clinician. Examining subungual sites is crucial to the identification and diagnosis of many conditions including lupoid onychodystrophy syndromes, bacterial / fungal paronychia and neoplasia arising from the subungual site (squamous cell carcinoma, melanoma). Collection of nailbed biopsies needs to be performed under general anaesthesia with appropriate post-surgical pain management. The technique involves driving the biopsy punch along the dorsal surface of the nail into the nail bed to include

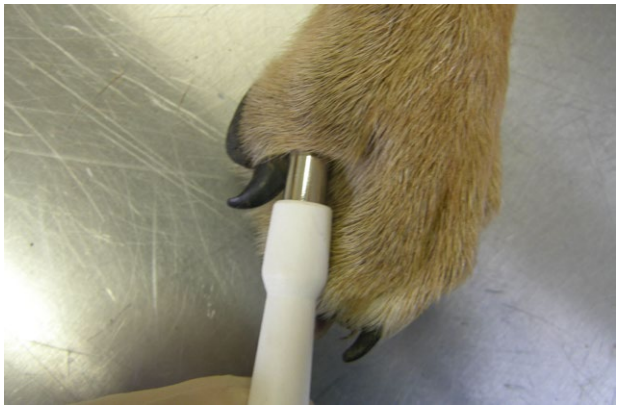

Fig. 4 - Nailbed biopsy technique involving driving the biopsy punch along the dorsal surface of the nail into the nail bed.

the dorsal nail matrix and the point where the dorsal epidermis folds back on the nail and links with the epidermis of the skin (Fig. 4).

Alopecia: Ellipse biopsies should be collected in the direction of hair growth so that hair follicles can be lined up in histological sections, to allow for full examination of at least 8 entire hair follicles from hair bulb to ostia.

Pigmentation disorders: As pigmentation disorders maybe centred on the epidermis or hair follicles (leukoderma, leukotrichia, colour dilution alopecias, follicular dysplasias, immune mediated pigmentary incontinence disorders, endocrine disorders, metabolic skin disease), ellipse biopsies are indicated for histological investigation.

Ulcerative conditions: The requirement for ulcerative lesions is that the entire lesion with transition edge to "normal" tissue be included. Wherever possible, excisional biopsies with margins should be submitted, punch biopsies frequently do not provide adequate tissue to establish a diagnosis. Ellipse excisional biopsies are preferred and should be full thickness to include the entire ulcerative surface, dermis, subcutis and subcutaneous fat. Excision of ulcerative lesions, where possible, is an important part of therapy.

Cutaneous neoplasia: The minimum requirement for a diagnostic biopsy is an adequate amount of tissue that is representative of the neoplastic process and sufficiently free of artifacts to permit a definitive evaluation. Incisional or excisional biopsies are the preferred samples as they meet these minimum requirements. In addition, with excisional biopsies treatment is incorporated in the same procedure and so considered a first-choice technique. Excisional biopsies also allow for examination of surgical margins for the completeness of excision. It is important to remember that tissue can shrink up to ± 40% following formalin fixation and so measurement of formalin-fixed tissue margins is not a true reflection of the actual tumour free distance. Decisions about whether further expansion is required should be based on measurement of fresh tissue margins at surgery and not the measurement of margins in formalin-fixed tissue sections.

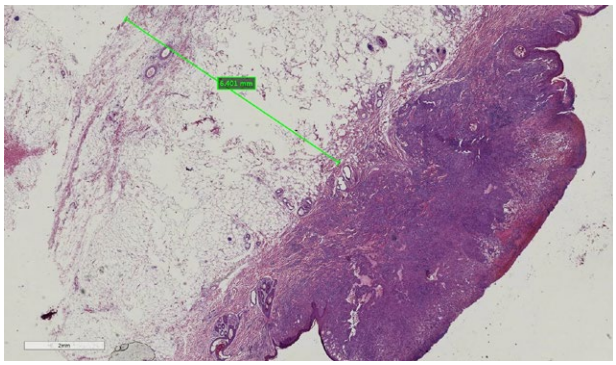

Fig. 5 - Measuring tumour free distance in histological sections (green scalebar) should not be used for decisions as whether to expand surgical margins, due to shrinkage of tissue fixed in formalin.

The only effective way to use histological margin measurement is to submit a clearly marked margin that has been measured in the unfixed state at surgery before it is placed in formalin. This measurement can then be correlated to the measurement of the same margin in the histological section (Fig. 5). Inclusion of regional lymph node/s in formalin enables assessment for possible lymph node metastasis. In general carcinomas metastasize via lymphatics so appearing in lymph nodes, while sarcomas usually metastasize via the blood vascular system and so frequently do not appear in lymph nodes, but rather at distant visceral sites.

Key Clinical Elements for the Submission Form

To ensure the best possible outcomes for patients it is paramount to ensure that the pathologist be provided with all the relevant clinical and patient data. In dermatopathology more than any other field, a complete history and signalment are the most important factors ensuring a correct diagnosis. Simple patient data such as breed, sex, age and distribution of lesions can have significant impact on the interpretation of the histological changes. For example, hereditary parakeratosis of Labrador Retrievers grossly resembles other nasal planum conditions, but the histopathology unique. English Bulldogs have a breed predisposition for flank alopecia and with German Shepherd dog pyoderma, it is the lesion distribution in the breed, that enables the link of the histopathology to the diagnosis, as the histopathology itself is non-specific plasma cell rich pyoderma.

When combining age of onset, distribution of lesions, presence, or absence of pruritis then histopathology can allude to underlying atopic dermatitis. Lesion distribution in classical atopic dermatitis includes the face (peri-ocular), ears, paws, limbs, ventral aspects of the body and perineum. However, not all these sites are affected in all animals. Distribution of lesions can also vary in complicated atopic dermatitis with involvement of concurrent flare factors such as ectoparasites, bacterial / yeast infections and adverse food reaction. What is important to remember is that atopic patients also exhibit suggestive histopathology at skin sites with no grossly visible lesions, and so always ensure biopsies from non-lesional skin are submitted together with biopsies from skin sites with lesions.

Histopathology facilitates inclusion or exclusion of clinical differentials. For example, pedal pruritis is a common and important clinical sign of atopic

dermatitis. Histological examination of pedal skin biopsies enables exclusion of differentials such as hookworm dermatitis and demodectic pododermatitis which are also cutaneous conditions more commonly encountered in juvenile animals.
Histopathology of hypersensitivity dermatitis can appear very similar irrespective of the primary trigger. Marrying the histopathology features to lesion distribution, seasonality, environmental factors and diet composition, provide very important links to distinguish between ectoparasitic, environmental or food associated skin conditions (some atopics react to certain food items). Therefore, it is vital for successful outcome that this background information is provided to the pathologist on your submission form.

Any other previous illnesses suffered by the animal can have significant implications for the development of certain skin conditions. Therefore, it is important that all such information from the patient record be provided on the submission form. For example, canine severe eosinophilic dermatitis is a condition which usually develops within 7 days following institution of multidrug treatment of gastrointestinal disease.

On completion of the submission form please ensure you have **given your pathologist what for!**

1. What did you see? (your clinical observations and findings of your dermatological examination including type of lesions, distribution etc)
2. What did you think? (What are your differential diagnoses for the condition).
3. What did you do? (What treatments, if any, have been attempted and the success thereof, what samples have been collected i.e., biopsies, fresh tissue for culture etc).
4. What do you want? (Histopathology, immunohistochemistry, aerobic culture, fungal culture, allergy serology etc).

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1. Skin pH and epidermal barrier function has minimal protection against which of the following?

a. Bacterial infection.
b. Fungal infection.
c. Environmental allergens.
d. Attachment of ectoparasites.
e. Sebborhea.

2. What would be considered the most important clinical reason to biopsy ulcerative skin lesions?

a. They are high risk lesions for septicaemia and toxaemia.
b. They are strongly associated with cutaneous neoplasia.
c. They are frequently associated with cutaneous vasculitis.
d. Their association with arthropod bites.
e. Their association with drug reactions.

3. Which of the following conditions are usually not associated with depigmenting lesions of the nasal planum of dogs?

a. Discoid lupus erythematosis.
b. Nasal mucocutaneous pyoderma.
c. Pemphigus foliaceus.
d. Uveodermatological syndrome.
e. Nasal dermatophytosis.

4. What is responsible for the formation of acantholytic keratinocytes in bacterial pustular pyoderma?

a. Autoantibodies.
b. Exotoxins.
c. Proteases.
d. Drugs.
e. Cell antigens.

5. Which of the following would be considered a secondary skin lesion?

a. Nodule.
b. Pustule.
c. Papules.
d. Scale.
e. Crust.

6. Punch skin biopsies for histopathology would be contraindicated which of the following skin conditions?

a. Superficial or deep pyoderma.
b. Hypersensitivity dermatitis.
c. Alopecia.
d. Hyperkeratosis.
e. Vesicular skin disease.

7. In which of the following skin conditions would excisional skin biopsies be indicated?

a. Depigmentation.
b. Mucocutaneous conditions.
c. Nailbed conditions.
d. Ulcerative skin lesions.
e. Alopecia.

8. Which of the following cutaneous conditions are consistently associated with lesions on the paws?

a. Facial discoid lupus erythematosis.
b. Hepatocutaneous syndromes (metabolic epidermal necrosis).
c. Pemphigus erythematosis.
d. Mucocutaneous pyoderma.
e. Uveodermatological syndrome.

9. For cutaneous neoplasia histopathology which is considered the most clinically valuable type of biopsy?

a. True Biopsy.
b. Punch biopsy.
c. Ellipse biopsy.
d. Incisional biopsy
e. Excisional biopsy.

10. Which of the following anatomical sites is not considered one of the classical sites of atopic dermatitis in the dog?

a. Periocular.
b. Ear pinnae.
c. Paws.
d. Lumbosacral.
e. Ventral abdomen.

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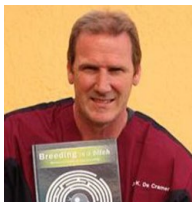
IDEXX

Bee Sting and Massive Attack by Bees in Dogs



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Rand en Dal Veterinary Hospital
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Reviewed by Dr. Liesel van der Merwe
BVSc MMedVet (Med) Small Animals

Introduction

Animals and humans get stung by bees where these species cohabit. Accurate statistics are kept in some countries for bee sting related fatalities in humans and these may approach up to a 100 deaths per year (Brazil). The prevalence of bee sting and related associated deaths in dogs is unknown but it is speculated to be generally underreported. It is likely that dogs are stung more often than humans because most humans take evasive action when attacked by bees whereas dogs are frequently prevented from escape because they are mostly enclosed and are also often combative in response to bee attack. It is therefore also more likely that more dogs than humans die from bee stings in most countries.

Unlike the European honeybee, the African honeybee (*Apis mellifera scutellata*), is an aggressive bee which attacks easily following little and often non-intentional provocation and its tendency to attack in larger numbers resulting in large volumes of venom injected into their victims. Once the first bees start stinging, volatile chemical substances from the bee sting are released which incite the other bees to aggression prompting them to sting as well. Venom from both European and Africanized bees appeared identical when analysed by acid-urea gel electrophoresis and both bees deliver an equal amount of venom.The African honeybee has a larger radius wherein it will pursue its targets. The African honeybee is highly adaptable in warmer climates making it a successful invader species where they have been introduced and readily hybridise with local European bees to form the feared "Africanised killer bees".

Venom properties

Honey bee venom contains many biologically active components such as melittin, phospholipase A_2, apamin, mast cell degranulating peptide, hyaluronidase, histamine, and dopamine (Table 1). The main activity of the toxins is to disrupt cell membranes. This leads to pain and inflammation at the site of envenomation and systemically it causes massive haemolysis and rhabdomyolysis by phospholipase A_2 in concert with mellitin. Apamin is a neurotoxic peptide that may lead to neurological signs following massive bee sting attack.

Five distinct syndromes may be associated with a sting and manifestation of one or more of the syndromes in a patient depends on the victim and the number of bee stings delivered

Patient responses to bee sting envenomation

1. Local reactions

This is the most common reaction to bee sting in humans and dogs and occurs in all stung individuals. There is local pain, erythema and swelling together with some pruritis. It is caused by the components of the venom and not a hypersensitivity response. Most such reactions to bee sting are self-limiting events that require no treatment provided the patient is not allergic to its venom.

2. Regional reactions following bee sting

Some dogs with one or only few bee stings may develop a regional Type 1 hypersensitivity reaction with oedema of the lips, ears eyelids and face. This reaction is usually inconsequential and disappears within hours without special attention or treatment provided the region of the sting does not cause swelling which may obstruct the upper airways. Some cases may require minor symptomatic treatment.

3. Anaphylactic reactions

This reaction occurs in animals which have been sensitised and have specific IgE antibodies to components of the venom. The reaction is rapid and occurs within 10 – 15 minutes of the sting. Signs may vary in severity between individuals and include urticaria, pruritus, angioedema, nausea, and vomiting. More serious signs of anaphylaxis include hypotension and dyspnoea (wheezing) due to bronchoconstriction and possibly also local airway oedema of the larynx, epiglottis and pharyngeal region.

4. Delayed hypersensitivity reactions

Delayed reactions (serum sickness) occurring 2 weeks after the sting are unusual and include polyarthritis, vasculitis, immune mediated anaemia and glomerulonephritis.

5. Systemic envenomation (massive bee sting envenomation)

The venom of bees also has a direct toxic effect. Massive bee sting envenomation in dogs is a dire emergency. The severity of the envenomation depends on the sensitivity of the dog as well as its mass and, most importantly, the number of bee stings received.

The estimated lethal dose for humans and mammals varies widely but some estimates are around 20 stings/kg. In humans as few as 50 stings have caused symptoms of systemic envenomation and deaths have been reported with 125 stings. However deaths usually occur most commonly with 500 or more stings in adults. Patient prognosis is poorer when treatment is delayed. When attacked by large numbers of bees, clinical signs of massive envenomation will commence within an hour.

Systemic envenomation is usually not associated with local or regional swelling from a type 1 hypersensitivity but ears and lips will be oedematous and erythematous due to direct action. These dogs are agitated or depressed, febrile and exhibit facial paralysis, ataxia and seizures. Further progression to vomiting, a very severe diarrhoea, rapid dehydration, haemolysis and rhabdomyolysis characterised by red discoloured urine, trembling, shock, hyperventilation, incoordination and later total collapse ensues.

The mechanism of haemolysis results in a spherocytosis and is direct toxin mediated, caused by mellitin and phospholipase A, both proven in vivo to cause haemolysis and deform the erythrocytes into echinocytes at lower concentrations, and spherocytes at higher concentrations. Severely affected dogs may

Table 1 - The pathophysiology and clinical effect of the components of venom.

Biological components of venom	Mechanism of action	Clinical effect
Melittin Main component (50 60% DMW)	• Acts with Phospholipase A_2 to disrupt cell membranes causing lysis of erythrocytes leukocytes, platelets, myocytes and vascular endothelium • Associated with acute myocardial ischaemia	• Haemolysis • Histamine release and local pain (direct action) • Acts like a detergent – damaging lipid cell membranes
Phospholipase A_2 (11% DMW)	Synergistic with catecholamines	• Haemolysis • Major allergenic component
Hyaluronidase	• Breakdown of connective tissue promoting uptake and spread of venom • Changes is cell permeability	Diffusion of venom, swelling
Apamin	Neurotoxicity	Spinal cord related neurological signs
Peptide 40	Mast cell degranulation release of histamine and vasoactive amines causing vasodilation	Cell swelling and oedema
Other vasoactive amines: histamine, dopamine, noradrenalin		

also develop respiratory distress from acute lung injury and neurological signs such as tremors and epilepsy. Acute kidney injury is a common complication of envenomation due to factors such as myoglobinuria, inflammation and hypotension. The venom also has direct nephrotoxic effects

Therapy for systemic bee sting envenomation

Honeybee venom sacs continue to contract even after torn from the bee's body and all the venom is injected within 60 seconds. There is thus no value in removing the stings to avoid further envenomation. There are no effective first aid measures one can take in dogs following massive bee sting envenomation (unless the owner has an Epipen®). Prevention of further stings is the most important immediate measure that needs to be taken and then seeking immediate veterinary care.

Most anaphylactic reactions occur within 1 hour of the sting.

First line treatment is adrenaline and fluid resuscitation. Immediate treatment with adrenaline (2.5 – 5 µg/kg iv, 10 µg/kg im, 0.05µg/kg/min CRI). Observe the patient during administration for arrhythmias. Adrenalin stabilises mast cell membranes and thus inhibits ongoing anaphylactic response, causes vasoconstriction and increased blood flow and heart rate, causes bronchodilation and also maintains endothelial integrity. Treatment may need to be continued for several hours as clinical signs dictate. Intravenous fluids at titrated shock volumes (maximum dose considered 90ml/kg in the dog and 60 ml/kg in the cat).

Second line treatment is antihistamines and glucocorticoids. Glucocorticoids block the arachidonic acid cascade thus preventing further/biphasic anaphylaxis. Additional treatment involves pain management, anti-histamines and other supportive measures such as transfusion therapy.

Airway management and supplemental oxygen may be indicated if acute lung injury occurs. Continued haemolysis appears to be a poor prognostic indicator. Some dogs may suffer from icterus and anaemia and make a full recovery following a long stay in intensive care. Some dogs may show an apparent recovery and later die from renal failure or other complications. Death from single or few stings is due to Type 1 hypersensitivity and anaphylaxis.

Death from major stings result from three major mechanisms: Direct venom toxicity; intravascular haemolysis due to mellitin and profound hypotension due to massive histamine release. Together these three effects are cumulative and cause multi-organ failure, immune mediated haemolysis, immune mediated thrombocytopaenia and disseminated intravascular coagulopathy.

No specific therapy is currently available and a safe and effective antivenom for the treatment of mass bee attacks is urgently required to safe both human life as well as those of dogs. Experimental work has demonstrated antivenom neutralizing ability using assays for venom phospholipase A_2 and in vivo activities. . Further research on the efficacy of antivenom for mass bee sting attack is required but research institutions with the required expertise need to be convinced about the financial feasibility of such studies and make accurate projections on return on investment. Ultimately, the commercial availability of a specific bee sting antivenom can only become a reality when veterinary practitioners demonstrate an in-practice need.

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The MDR1 Gene in Dogs

Mutations and Increased Susceptibility to Drugs

Unistel Pets

As the public awareness of multi-drug resistance in dogs continues to rise, it has become increasingly important for both pet owners and their veterinarians to understand the mechanisms at play as well as the risks involved with administering certain types of medications. With information so readily available, pet owners may be presented with confusing or misinformation about their dog's condition which in turn could make them wary of administering any form of medication or even taking their pets for routine vaccinations. It is therefore very important that veterinarians be able to provide their clients with accurate information in order to help them make the best decisions for their animal's health.

The multi-drug resistance 1 (*MDR1*) gene is a member of the ATP-binding Casette (ABC) transporter family. This gene encodes for an ATP-dependent P-glycoprotein transporter in the cell membranes of eukaryotes. When this protein functions normally, it serves as a pump within the cell membrane, which transports potentially harmful compounds out of the dog's central nervous system (CNS). It is an important part of the blood brain barrier. A mutation in this gene, reduces the effectiveness of these membrane pumps. Studies into drug sensitivity in dogs have found a 4 base-pair deletion within the *MDR1* gene, which in turn leads to a frameshift that causes a premature stop codon. This mutation creates a truncated (shortened) transporter protein of only 91 amino acids as opposed to the normal 1281 amino acids needed to create a proper functioning membrane pump.

The mutation is found predominantly in the herding dog breeds. Breeds most commonly affected: Border Collies, Rough Collies, Australian Shepherds, Shelties, English Shepherds, German Shepherds and Old English Sheepdogs.

The mutation is genetically passed on from one generation to the next. Dogs who receive a mutant copy of the gene from both mother and sire will be the most severely affected and prone to toxicosis. Dogs who receive one mutated copy will still be affected by the dysfunctional proteins (referred to as an intermediate macrocyclic lactone sensitive phenotype), but to a lesser extent than dogs carrying two mutant copies. The defective P-glycoprotein allows higher levels of medication to cross the blood brain barrier (BBB), leading to an increased sensitivity to certain drugs which increases the toxic neurological side effects of some medications. P-glycoproteins also act in the biliary system, intestine, and kidney to promote drug excretion into bile and urine and out of the body.

Several medications (tabulated below) have been shown to lead to toxicosis in dogs carrying copies of the mutated *MDR1* gene. Some of these medications can still be administered at a reduced dosage, whereas others should be exchanged for a non P-Glycoprotein based drug.

It is important to have at-risk breeds tested for the *MDR1* gene mutation, not only to assist vets in making informed decisions when it comes to the dog's medication management, but also to identify animals in breeding studs that could potentially pass the *MDR1* mutation on to their offspring. Testing for the *MDR1* mutation is now available from several laboratories. Samples can be sent in the form of blood EDTA, buccal swabs, or sufficient hair (with roots), and can be submitted through the veterinarian or by the owners themselves.

Commonly used as:	Medication given:	Dosage adjustment:
Antiparasitic agent	Ivermectin	Average dosage of 6mg/kg used in treating heartworm is safe for dogs with the *MDR1* mutation . However, higher dosages (300-600µg/kg), such as those used to treat mange will cause toxicosis in dogs with 2 mutant copies and could cause toxicosis in dogs with 1 mutant copy.
	Milbemycin, Moxidectin, Selamectin	Average dosage for heartworm treatment is safe for dogs with the *MDR1* mutation, but higher dosages may lead toxicosis.
Anti-diarrheal agent	Loperamide	Normal dosage for treatment of diarrhoea can cause toxicosis. It is advised to rather avoid treatment with this drug.
Pre-anaesthetic/ Tranquilizing agent	Acepromazine	Reduction in dosage required.
Analgesic/ Pre-anaesthetic agent	Butorphanol	Reduction in dosage required.
Chemotherapy agents	Doxorubicin, Paclitaxel, Vincristine and Vinblastine	Reduction in dosage required.
Induce Vomiting after poison/toxin ingestion	Apomorphine	Standard dosage can cause CNS depression in affected dogs.

Plugging the Protein Faucet in Dogs with PLE

On a normal basis, anywhere from 10% to 50% of the total protein loss funnels through the gut; however, more than this amount leads to PLE

Joan Capuzzi, VMD

The key feature of PLE is panhypoproteinemia, due to the loss of both small (albumin) and large (globulin) proteins.

A leaky gut has several potential backstories. But every type of "protein-losing enteropathy" (PLE) shares a common loss: albumin, a vital protein that regulates the oncotic pressure of blood, among other things. Although dogs with PLE are quite sick, advances in diagnostics and treatments have improved outcomes.

Healthy dogs lose regulated amounts of protein through the gastrointestinal (GI) tract and the kidneys, but hypoproteinemia can result from excessive loss secondary to protein-losing enteropathy (PLE). Other conditions that can result in hypoproteinemia include protein-losing nephropathy (PLN) and decreased hepatic synthesis. On a normal basis, anywhere from 10% to 50% of the total protein loss funnels through the gut[1,2]; however, more than this amount leads to PLE.[3]

Hypoproteinemia was once thought to result solely from decreased protein synthesis. But in 1949, investigators discovered increased GI losses to be the major cause. In 1957, radioactive iodine tags were hitched onto albumin so it could be followed along its journey through the intestines.

More of a syndrome than a discrete "disease," PLE is now known to have aetiologies that can vary between dogs as well as between species. "PLE is not a diagnosis," Owens said. "It's just a beginning. You need to figure out what the underlying cause is."

In humans, this underlying cause is usually either cardiovascular disease, liver disease, ulcerative colitis or Crohns disease. In cattle, Johne's disease can result in protein loss from the gut, whereas equine PLE is often associated with GI ulcers and infectious diarrhoea (eg, salmonellosis). In dogs, inflammatory bowel disease (IBD) is often the main suspect.

Mammalian species have a conserved microarchitecture that facilitates the absorption of nutrients from ingested food. Millions of raised villi line the intestinal tract. Within each villus, venules, arterioles and capillaries surround a central lacteal, which drains nutrients that cross the mucousa but are too large to be absorbed into the vasculature. The lacteals transport these particles, primarily dietary fats, to larger lymphatic vessels and then to the liver.

The job of the lacteals is to "pick up the mess from all the leftovers that can't be absorbed through the veins." Several different pathologies can disrupt this process. Anything that alters mucousal permeability and integrity can precipitate protein loss. These include erosions, foreign bodies, neoplasia, infection (eg, parvovirus, parasites), and inflammation (IBD).[4]

Protein leakage can also result when lacteals burst despite a healthy intestinal mucousa, a condition called lymphangiectasia.[4] Although lacteals are flexible and can distend easily, Owens noted, "they hit that breaking point and then they all start popping and you get this massive, acute protein loss." In dogs, PLE is typically associated with either lymphangiectasia or lymphoplasmacytic enteritis.[5]

Several breeds are predisposed to PLE. In Yorkshire terriers, lymphangiectasia can be primary (defective, dilated lacteals) or secondary (lymphatic obstruction due to portal hypertension).[6] In either case, Owens explained, "they ooze tons and tons of protein." These yorkies often arrive acutely ill, with albumin levels as low as 8 g/L (normal range, 24-40 g/L).

Other breeds that are genetically predisposed to PLE include soft-coated wheaten terriers (specifically, a PLE/PLN complex possibly associated with food allergy[7]) and Norwegian lundehunds (a severe chronic enteropathy seen in virtually all dogs in this breed due to genetic bottleneck[8]), as well as Rottweilers, German shepherds, and shar-peis (all prone to chronic enteropathies).

Dogs with PLE often present with small- or mixed-bowel diarrhea, although some 30% of them do not have diarrhoea as part of their clinical picture. Weight loss is typical and vomiting is rare. Some dogs can present with oedema and pleural effusion due to severe protein loss. Blood work may show hypoalbuminaemia or panhypoproteinaemia, hypocholesterolemia, hypercholesterolaemia, lymphopenia, hypocalcaemia and hypomagnesemia. Hepatic or renal protein loss should be investigated with a urinalysis, as well as an evaluation of serum liver enzymes and bile acids.

Coagulopathies (hyper- and hypocoagulopathies) may also be present due to associated vitamin D or K deficits or loss of antithrombin. In a study of 138 dogs with PLE, 15% had evidence of thrombus formation. Lacteals, normally microscopic, may be visible on an ultrasound when dilated. During an endoscopy, lymphangiectasia appears as a speckled white pattern within the small intestine. IBD, on the other hand, features cobbly intestinal mucousa.[9] Owens recommends administering corn oil 3 to 4 hours prior to endoscopy, thereby making the lacteals swell so they are easier to visualize. Endoscopic versus surgical biopsies are preferred in patients with severe hypoalbuminaemia due to the postoperative risk for dehiscence in these compromised tissues.

The presence of hypoalbuminaemia is a major factor informing treatment and prognosis.[10] In a study involving 80 dogs with IBD, 20% had hypoalbuminaemia. Of the 10 dogs that were euthanased, 7 had hypoalbuminaemia.

Another study found shorter survival times (701 days) in dogs with hypoalbuminaemia compared with those with normal albumin. Approximately 75% of dogs survived the acute period, the crucial first month. Additionally, the extent of the hypoalbuminaemia did not correlate with outcome: A dog with mild hypoproteinemia did not necessarily fare better than one with severe protein loss.

For dogs whose hypoalbuminaemia was caused by intestinal lymphoma—rarer and more serious in dogs than in cats—survival times ranged from 2 weeks to 2 months, according to results from one study.

The main goal of therapy is to harness albumin loss. Acute cases require more aggressive treatment than those that have been festering for a while. If the albumin level is less than 15 g/L, fluids are likely to shift into pleural and paritoneal spaces causing effusions because there is not enough albumin to trap them in the vasculature. To avoid third-spacing of fluids, Owens recommends using half crystalloids and half colloids. If colloids are not available, fresh frozen plasma can be used; however, large amounts may be needed to substantially raise oncotic pressure.

(*If mild pleural effusion is present but the patient is eupneic, Owens cautions against thoracocentesis because it could lead to further albumin loss. Antibiotics should be used only if diarrhoea is present. Intravenous metronidazole can be given in the hospital, and then the patient can be sent home with oral metronidazole; tylosin can be used as an alternative.[11]*)
- Both editors disagree with the above statements

Editors comment: PP, VDM - Draining the effusions caused by hypoalbuminaemia should not be necessary unless pleural effusion is causing dyspnoea. Fluid therapy in the face of low albumin can often cause iatrogenic effusions. Decreasing the fluid rates will generally correct the situation without adding colloids.

If biopsies confirm that the PLE has an inflammatory component, as is usually the case, immunosuppressants play a huge role. According to Owens, most dogs respond to prednisilone. Budesonide may also be effective,[12] and result in less systemic absorption, particularly important if heart disease is also present. For those whose hypoalbuminemia is severe or refractory to steroids, a second immunosuppressant can be considered. Cyclosporine is the gold standard for additive therapy,[13] but chlorambucil or azathioprine can also be used.[14]

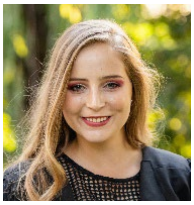
Anticoagulants are used in dogs with hypercoagulopathies, *as patients lose antithrombin which is similar in size to albumin* (*Ed.*), and vitamin supplementation with cobalamin (vitamin B12) can be used in case of cobalamin deficiency.

In the case of lymphangiectasia, low-fat diets such as Royal Canin Gastrointestinal Low Fat dog food, Hill's Prescription Diet i/d Sensitive and home-cooked chicken or potato-type meals should be implemented.[15] For patients with food allergies or IBD, hypoallergenic diets can help to seal the gut.

References available online: www.vet360.co.za

Successful Pet Export to EU and Northern Ireland

The private veterinarian's role with new regulations



Dr Anika Aline de Witt (BVSc)
Compulsory Community Service Veterinarian
Export Control, Western Cape Government

Introduction

The amended regulations for commercial pet export to the European Union and Northern Ireland will be implemented as from 21 August 2021. The understanding of the new regulations will assist the private veterinarian and subsequently the client to avoid common mistakes due to inadequate knowledge of the process.

Timeline overview

First time travelers: Minimum 4-month process. (Fig. 1)

Transponder and Rabies vaccination

Vaccinations are only considered valid after implantation/reading of the transponder by a registered veterinarian.

The animal must be older than 12 weeks of age at the time of vaccination against rabies.

Vaccinations are only accepted if done by a SAVC registered veterinarian. It is important to sign and write "BVSc" or equivalent qualification under the signature for the official veterinarian to verify that a veterinarian administered the vaccine.

Rabies Neutralizing Antibody Titre Test

Blood sample collection for rabies neutralizing antibody titre test (RNATT) must be done by a registered veterinarian not less than 30 days after the preceding primary vaccination within the current valid vaccination series. Day zero is the day the vaccination was administered and therefore days should be counted from the date after the administration date.

A sample of 1 ml blood can be collected in a serum tube. The rabies antibody titration must be performed by an approved laboratory and the result of the rabies antibody titer should be equal to or greater than 0,5 IU/ml.

A minimum period of 90 days after the date of blood collection must lapse and the result of the RNAT must be >0.5 IU/ml before a health certificate can issued by the private vet.

The original RNATT certificate must be presented to the export office upon application.

The RNATT will continue to be valid, provided that the pet is revaccinated against rabies within the period of validity of the previous vaccination according to the manufacturer's recommendations.

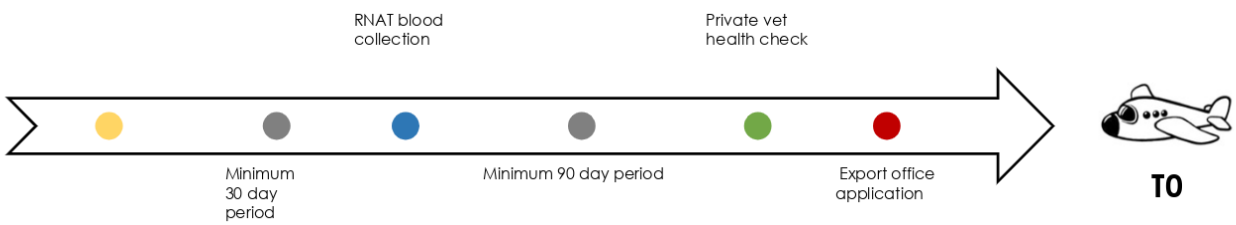


Fig. 1 - First time traveler 4-month process

Private Veterinarian Health Check

Health certificate

Completion of the Western Cape specific health certificate must be done before application at the state office is done. The template used in the W Cape is valid for Gauteng as well . see template on digital version)

https://www.elsenburg.com/services-and-programmes/veterinary-services

Echinococcus multilocularis treatment for dogs

For pets traveling to Malta, Finland, Ireland, and Northern Ireland must receive treatment against Echinococcus multilocularis within a period of not more than 48 hours end ending not less than 24 hours before scheduled time of entry of the dog into the member states. The private veterinarian should record the name, date and time of treatment administered.

Declaration for cleaning and disinfection of transport crates

Cleaning and disinfection of crates is essential should occur for the commercial exportation of dogs and cats to the EU and Northern Ireland. An approved surface disinfectant must be used, and disinfection should be carried out at the veterinary clinic or animal travel agency premises prior to loading.

The written declaration form must be complete, and the weight of the crate and pet must be recorded.

Veterinary Health Certificate

The application for a veterinary health certificate is done by the client , by appointment, at a state veterinary export office.

Type of applications are described in the table below.

Non-commercial travel	Commercial travel
The pet is traveling with the owner/authorized natural person	The pet is traveling without the owner/authorized natural person
The pet is traveling on or within 5 days of the owner/ authorized natural person	The pet is traveling more than 5 days from when an owner/authorized natural person
Veterinary Health Certificate (VHC) Validity	
Issued not more than 5 days before arrival at Malta, Finland, Northern Ireland and Ireland. VHC valid for 10 days from date of issue for other EU member states.	Health certificate issued within 48 hours before departure

For export via Cape Town International Airport:
Milnerton Export Office, Western Cape:
vetexport@elsenburg.com

Boland State Vet Office:
svboland@elsenburg.com

Via OR TAMBO
Gauteng: Pretoria or Germiston State Vet:
VetPermits@daff.gov.za
https://www.nda.agric.za/vetweb/Contacts will also give you some contact information

The client should present all the relevant documentation on the day of application.

Client Checklist

- ☑ Schedule appointment and verify registration of holding place
- ☑ Original and copy of RNATT certificate
- ☑ Original and copy of vaccination booklet incl. ID and rabies vaccination page
- ☑ Transponder certificate or letter
- ☑ Completed Health Check
- ☑ Completed writter declaration form
- ☑ Flight details

Email addresses for the Departments dealing with pet import

New Zealand and Australia have extremely helpful web pages to assist the vet / owner in going through all the documentation - Ed.

New Zealand
https://www.govt.nz/browse/immigration-and-visas/bringing-things-into-new-zealand/bringing-pets-into-new-zealand/

Australia
https://www.agriculture.gov.au/cats-dogs

UK (no longer EU)
http://www.defra.gov.uk/animalh/quarantine/pets/procedures/owners
http://apha.defra.gov.uk/documents/bip/iin/bllv-5b.pdf (March 2021 Documentation)

USA
https://www.cdc.gov/importation/bringing-an-animal-into-the-united-states/dogs.html

Canada
https://inspection.canada.ca/animal-health/terrestrial-animals/imports/import-policies/live-animals/pet-imports/eng

Approach To a Patient with a "Blue Eye"

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Introduction

A blue appearing cornea is the result of corneal oedema. This is a common presenting clinical sign and is often an indication of a sight threatening ocular disease.

A transparent cornea is essential for normal vision. Transparency of the cornea is the result ofseveral features, including the uniform small diameter and a lamellar arrangement of the corneal collagen fibres, lack of blood vessels and pigment and the relative state of dehydration (80 % hydrated).

Anatomic integrity of the corneal epithelium and endothelium provide two-way, physical barriers against the influx of tears and aqueous humor. Loss of the corneal epithelium may result in a 200% increase of corneal thickness whereas a loss of the endothelium which may result in a 500% increase of corneal thickness. Apart from the role as physical barrier the endothelium also functions as a Na – K ATPase pump removing water from the corneal stroma.

If the corneal epithelium is lost water enters the stroma from the precorneal film and a bluish "fluffy" discoloration occurs until a new layer of epithelium has covered the area. Endothelial cell loss tends to cause more severe and diffuse corneal oedema. Endothelial corneal oedema is reversible If the underlying cause is removed and if enough functional corneal endothelial cells are be present to re-establish the natural dehydrated state of the cornea.

Clinical approach

It is important to differentiate an epithelial cause from an endothelial cause. It is very easy to differentiate

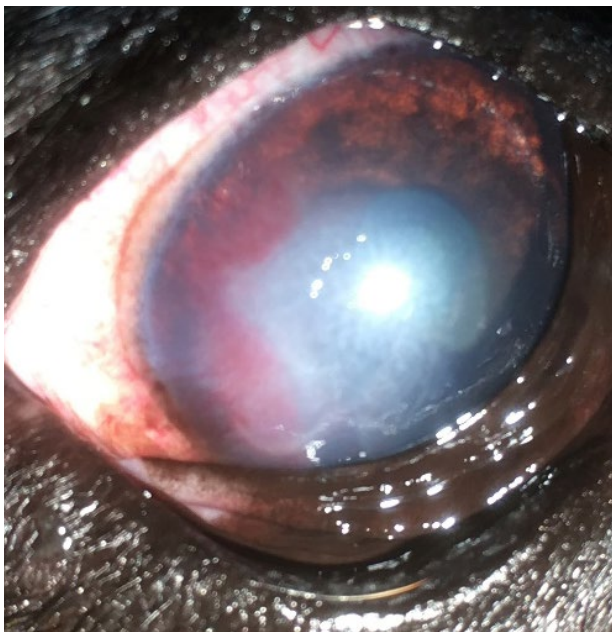


Fig. 1 - Corneal oedema associated with epithelial cell loss. It is more localized to the area of corneal ulceration

with the use of fluorescein. A fluorescein positive cornea is an indication of the loss of corneal epithelial cells. In cases with a corneal erosion / ulcer It is critical to try and identify the underlying cause for the loss of epithelial cells and address the underlying issue.

Conditions that may lead to loss of epithelial cells include the following: distichiasis, trichiasis, ectopic cilia, eyelid neoplasia, entropion, epithelial dystrophy / degeneration, mechanical abrasions, keratoconjunctivitis sicca, lagophthalmos, chemical burns, exposure keratitis, endothelial disease leading to bullae formation and primary corneal pathogens [Feline Herpes virus].

The diagnostic approach to a patient with a corneal ulcer should include the following:

- Assessment of corneal and palpebral reflexes.
- Schirmer tear test.
- Examination of eyelids, conjunctiva as well as under the third eyelid with good light source and magnification.

Bacterial / fungal cultures if the ulcer is believed to be infected. This is not done routinely but is indicated in non-healing / complicated ulcers

If the cornea stains fluorescein negative the most likely cause for the corneal oedema is endothelial cell loss/ malfunction. Endothelial disease include endothelial dystrophy and age-related degeneration. Endothelial cells can further be damaged by numerous factors including persistent pupillary membranes, mechanical trauma, toxic reactions, anterior uveitis, endothelitis and glaucoma.

Primary endothelial disease is typically non painful, the eye is not inflamed, there is no aqueous flare and the intraocular pressure is normal. (will highlight this statement somehow)

1. Endothelial dystophy

A breed predisposition for endothelial dystrophy occurs in a number of breeds. The most common breed affected is the Boston Terrier, but other breeds include Chihuahuas, Boxers and Dachshunds. It occurs more frequently in females. Canine endothelial cell dystrophy may represent a disease process similar to Fuchs dystrophy in humans. Fuchs dystrophy is inherited as an autosomal-dominant inherited disease with incomplete penetrance in humans. The condition is characterized by progressive, bilateral degeneration of corneal endothelial cells.

The typical clinical signs include corneal oedema affecting the lateral cornea and progressing slowly over months to years to involve the entire cornea. Severe oedema leads to stromal bullae with consequent rupture and secondary corneal ulcers. This is typically a nonpainful condition unless bullae form and rupture.

2. Age related endothelial cell degeneration.

The clinical appearance is very similar to endothelial degeneration with the exception that it occurs in older patients. The endothelial cells in the mature animal have a very limited mitotic ability and once clinical signs appear the condition is always progressive.

3. Endothelial degeneration

Intraocular inflammation [uveitis] results in corneal oedema via an increase in endothelial permeability and a decrease in Na+/K+-ATPase pump activity. There are numerous potential causes.

- Anterior lens luxation causes trauma to the

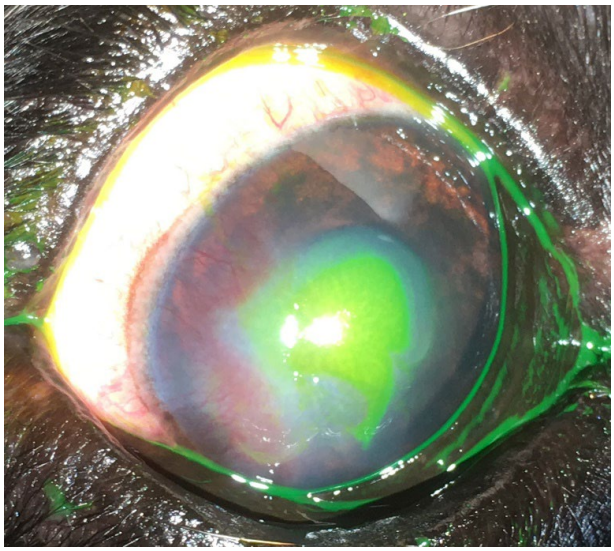


Fig. 2 - Same patient as in Fig. 1 showing the extent of the epithelial cell loss after the application of fluorescein.

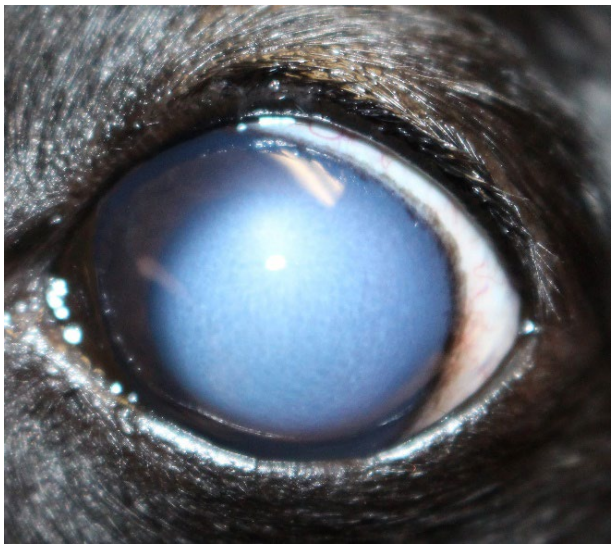


Fig. 3 - Typical diffuse corneal oedema associated with endothelial cell loss/ malfunction.

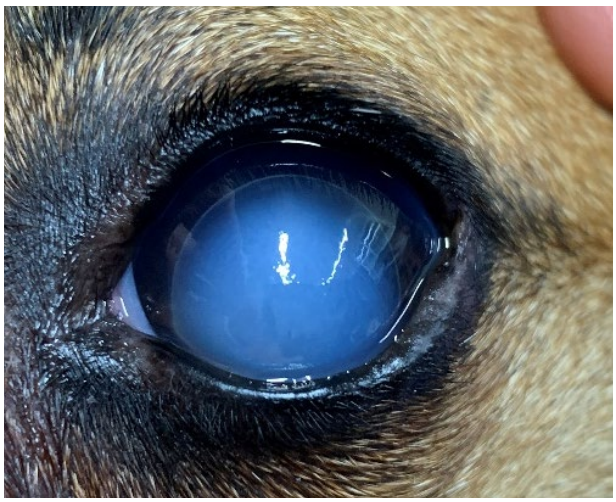


Fig. 4 - Patient with anterior lens luxation. The corneal oedema is more or less limited to the area of possible direct contact between the luxated lens and the corneal endothelial cells.

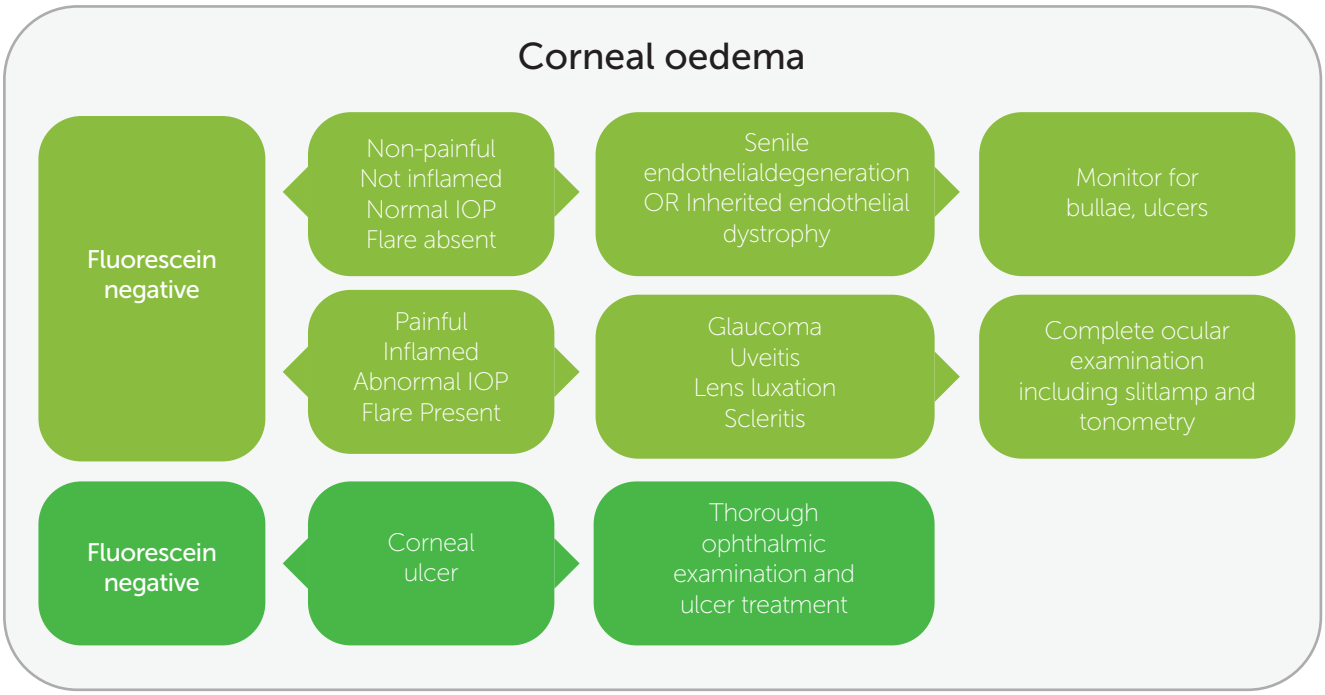
endothelium by physically damaging the endothelial cells due to the abnormally located lens as well as due to a possible secondary glaucoma.

Certain drugs may have side effects resulting in corneal and oedema in dogs. These include chlorpromazine, lortalamine and tocainide.

Another important condition to keep in mind whenever a patient with corneal oedema is presented is glaucoma. Intra-ocular pressure exceeding 40 mmHg leads to damage of corneal endothelial cells.

Treatment

The treatment of patients with a blue cornea obviously depends on the underlying cause. The examination of all patients with corneal oedema should ideally include tonometry, examination with a slit light for the presence of aqueous flare as well as fluorescein staining. The treatment of each of the possible causes for a patient with corneal oedema falls outside the scope of the article.



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Managing South African Snake Bite Envenomation in Dogs

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Introduction

Snake bite is a common South African veterinary emergency in pet dogs but very rare in cats. Dogs are by nature inquisitive and lack the fear factor than most human beings have for snakes. This is the reason that the incidence of snake bite in dogs is likely much higher than it is in human beings. In humans the most common bite sites are below the knee (as people usually stand on the snake that bites them) or below the elbow (especially in snake handlers). In dogs, the most common bite site is the face and neck region. This is a consequence of them either sniffing at or biting and attacking the snake. The affected anatomical region has implications for the way bites (especially from cytotoxic venoms) are managed (see below). Most snake bites occur in summer and autumn. There is a fairly typical annual autumn peak in incidence and this may be due to increased pre-hibernation snake activity.

South African snakes are grouped according the effect the venom has on the envenomed host. The most common bite in dogs is probably from the puff adder which has a cytotoxic venom and causes tissue destruction. The Berg adder is an exception in that it produces a neurotoxin as well as a cytotoxin. It is a rare cause of envenomation in humans and bites have not been recorded in the veterinary literature. Neurotoxic venoms from cobras, with the Mozambique spitting cobra a notable exception, cause generalized muscle paresis/paralysis. Many neurotoxic snake venoms also have cytotoxic effects and hence are mixed toxins. The Mozambican spitting cobra has cytotoxic venom (see Table 1).

Venoms that cause a primary coagulopathy with massive spontaneous haemorrhage are the third venom class and these are represented by the Boomslang and the Twig snake. Bites from these species are probably more common in dogs than in humans.

Pathophysiology and clinical signs

In the greatest majority of cases, snake bite is suspected based on presenting clinical signs and circumstantial evidence (such as where the dog lives and time of year). Many snakebites are not witnessed by the owner. This requires the clinician to be aware of the typical presenting clinical signs and have a suspicion index of the likelihood of envenomation. Not all snake bites result in envenomation as in some cases snakes will inflict so called 'dry bites' (no venom is injected with a bite).

Fig. 1 - Typical uncomplicated cytotoxic snake bite envenomation in two dogs. The facial swelling is due to interstitial haemorrhage. These dogs require minimal treatment and make uneventful recoveries.

Cytotoxic envenomation

The mechanism of action of these venoms is reflected by the nature of the fangs of the snakes that carry them. These snakes have long hinged front fangs which means the venom is deposited deep into tissues. There is no significant systemic circulation of the venom – all the action is local at the bite site. Any systemic effects seen as a result of a bite are a consequence of the potentially massive local tissue injury. Although morbidity is high, mortality is typically low.

The dominant clinical sign is sudden onset of relatively pain free facial swelling. Most cases are otherwise bright alert and responsive. Swelling usually begins with a few hours of the bite and will typically continues worsening, peaking at 12 – 24 hours after the bite (Fig. 1). Although the swelling is uncomfortable, the severe pain and haemorrhagic blistering seen in humans (typically bitten below the knee or elbow

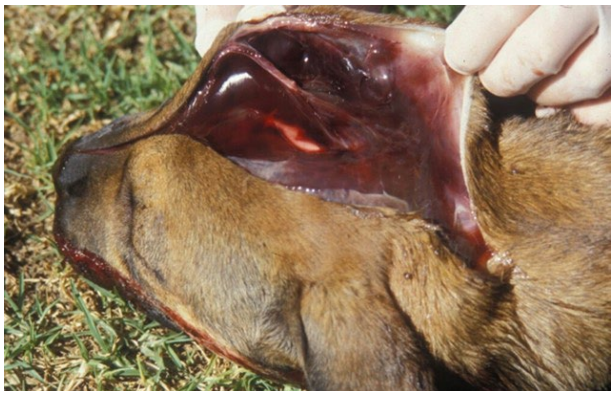

Fig. 2 - A puppy that died as a result of pPuff adder envenomation. Note that the swelling is due to frank blood loss around the bite site.

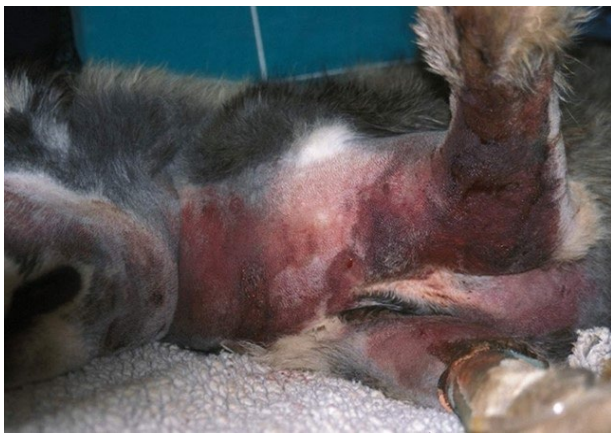

Fig. 3 - Very severe pPuff adder envenomation demonstrating prostration and profound blood loss into massive interstitial haemorrhage. Note the significant swelling around the head neck and forelimbs and the obvious bruising.

where there is very little loose tissue) is not a feature in dogs.

Compartment syndrome

Because venom deposition in humans usually occurs in muscle bellies or in tissue spaces confined by fascia, the severe swelling caused by the venom will often result in compartment syndrome. Compartment syndrome is rare in veterinary medicine and us usually seen as a result of crush injury, surgery and recently some reports of neoplasia of the muscle. This syndrome causes a massive increase in interstitial pressure with the resultant collapse of veins, lymphatics and sometimes arterial vessels. The consequences are severe pain and ischaemic tissue loss through necrosis. Recovery will often be accompanied by large tissue defects as a result of tissue loss. The amount of loose skin and substantial potential space in these loose tissues of a dogs face and neck mean that compartment syndrome in this bite site is not a feature.

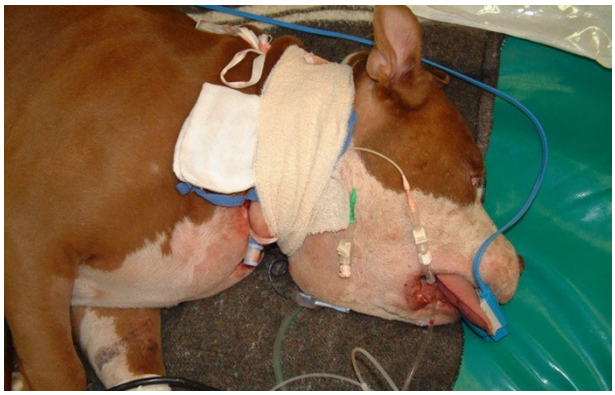

Fig. 4 - A case of severe soft tissue swelling following a Puff adder bite causing obstruction of the upper airway that necessitated the use of an endotracheal tube as a tracheostomy tube.

On the rare occasion when a dog is bitten in a muscle belly or into tissues bounded by restrictive facial planes, compartment syndrome and swelling with pain disproportionate to the injury pain is a feature of the clinical presentation. Although cytotoxic bites are common emergencies in the Onderstepoort Veterinary Academic Hospital (OVAH), this syndrome has been observed infrequently and only when bites have occurred on limbs.

The swelling associated with Puff Adder bites is caused

Table 1 – Summary of snakes and associated toxicity type

Common Name	Scientific name	Venom effect
Puff Adder	*Bitis arietans*	Cytotoxic
Berg adder	*Bitis atropos*	Cytotoxic and neurotoxic
Rhombic night adder	*Causus rhombeatus*	Cytotoxic
Mozambique spitting cobra	*Naja mossambica*	Cytotoxic (spitting)
Cape cobra	*Naja nivea*	Neurotoxic
Snouted cobra	*Naja annulifera*	Neurotoxic and cytotoxic
Black mamba	*Dendroaspis polylepis*	Neurotoxic
Rinkhals	*Hemachatus haemachatus*	Neurotoxic and partially cytotoxic (spitting)
Boomslang	*Dispholidus typus*	Haemotoxic
Twig Snake	*Thelotornis capensis*	Haemotoxic

by frank interstitial haemorrhage (loss of whole blood into the interstitial space) (Fig. 2). Severe swelling can result in clinically significant blood loss within the first 12 – 24 hours following the bite (Fig. 3). Whole blood loss will not reflect in a lower haematocrit until days after the event. This loss of circulating volume can result in classic hypovolaemic shock with all of its presenting clinical signs such as weakness or collapse, tachycardia, reduced body temperature, pale mucous membranes, reduced urine production and sometimes frank haemorrhagic faeces as a result of loss of the intestinal mucousa as a result of circulatory shock.

Massive envenomation can result in secondary immune mediated haemolytic anaemia, thrombocytopaenia and disseminated intravascular coagulation with a bleeding tendency. Haemolysis may be evidenced by haemoglobinaemia and haemoglobinuria. In some cases, the severity of the facial swelling may result in occlusion of the airway, especially if the patient was bitten in the tongue or pharyngeal area (Fig. 4).

Signs of Deterioration

The signs of deterioration that would indicate a need for an increased level of care would include: massive swelling (Fig. 3), progressive weakness, and upward trend in pulse rate which is usually due to due to blood volume depletion, the development of polypnoea or dyspnoea which may be due to acute respiratory distress syndrome or acute lung injury, mucous membranes becoming pale due to hypoperfusion, swelling that progresses and becomes severe, increased effort to breathe due to impingement by swelling on the upper airway, a drop in blood pressure, reduced urine production due to reduced renal perfusion, the development of haemolytic anaemia, the presence of haemoglobinaemia or haemoglobinuria indicating intravascular haemolysis (Fig. 4), signs of DIC such as spontaneous bleeding, clinical pathology evidence of a coagulopathy or the development of haemorrhagic stools.

The Rhombic night adder may also inflict bites that cause significant tissue loss and even death.

The Mozambique spitting cobra is a very common resident in the areas where it does occur and inflicts a very painful cytotoxic bite. It is also the most common cause of ocular envenomation in South Africa and this is not an uncommon occurrence in dogs. The venom can cause a very severe keratitis and conjunctivitis if untreated (Fig. 5). The clinical presentation of this cytotoxin is quite different from that seen with the Puff adder. Swelling is minor but pain is extreme (reaching levels similar to those seen with Hyalomma tick bites) and large geographically irregular zones of partial to full thickness skin necrosis become evident within days of the bite (Fig. 6).

It is not uncommon to see significant body surface areas necrose following bites. The delineation of the extent of the skin loss is not usually seen for 4 or 5 days after the bite. Although the most obvious presentation of bites from the Snouted cobra are neurotoxic, within a week of the bite (if the dog survives the initial stages), there will very often be a moderately sized well circumscribed zone of full thickness necrosis at the bite site (Fig. 7)

Differentials to consider with severe swelling of the head and neck region:

- Intermandibular abscesses – sub acute swelling on history as well as an inflammatory swelling

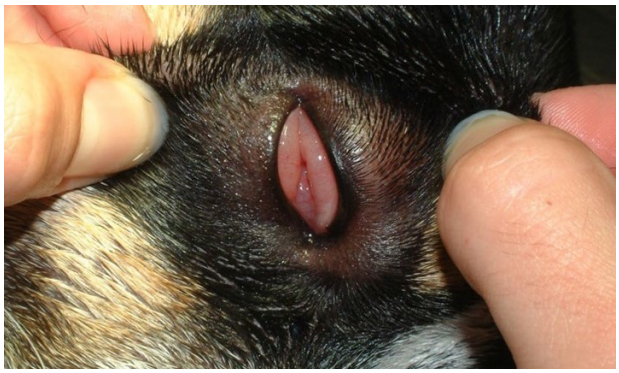

Fig. 5 - Ocular envenomation due a Spitting cobra. Note the very severe conjunctival swelling. This condition is very painful and requires deep sedation to allow proper irrigation of the eye to rinse remaining venom off the tissues.

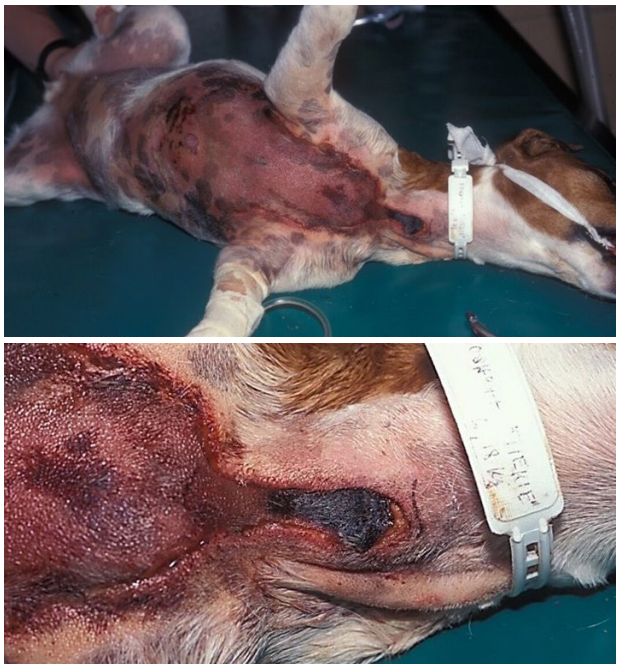

Fig. 6 - Large areas of skin necrosis and intense pain associated with a Spitting cobra bite.

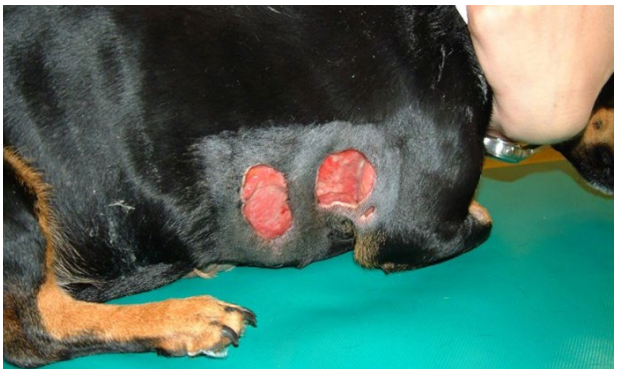

Fig. 7 - Tissue loss as a result of the cytotoxic effect of a Snouted cobra bite. This dog survived the neurotoxic phase of the envenomation (with the aid of polyvalent antivenom and ventilation) and then went on to develop this skin loss at the bite site.

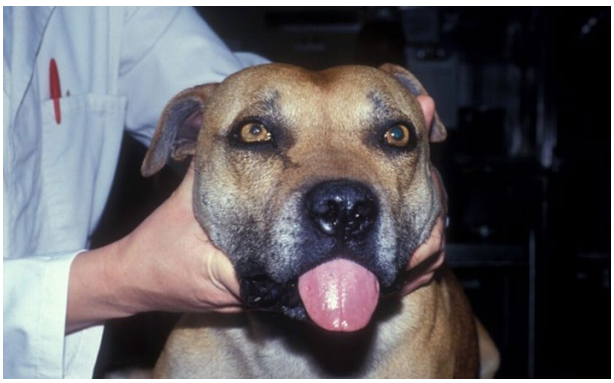

Fig. 8 - Typical bulbar paralysis (note the cycloplegia and loss of tongue tone – the dog was already quadriplegic) seen with a neurotoxic snake bite (a Snouted cobra in this case).

with heat and pain in the region.

- Sialocoele - More unilateral and slower to progress. History tends to be chronic. Acute swelling if it becomes infected.
- Acute urticaria or angioedema due to acute type I hypersensitivities (as seen with stings and insect bites).
- Anasarca ("bottle jaw")
- Soft tissue mass resulting in lymphatic or venous drainage of the head/neck ("Cranial vena cava syndrome")
- Neoplastic mass – History tend to be chronic

Neurotoxic envenomation

The snakes that carry these venoms have short front fixed fangs. The venom generally has little local effect (except for the Snouted cobra where tissue loss is often seen) and must be absorbed and circulated systemically to reach the target (which is the neuromuscular synapse). These venoms (as represented most commonly by the Snouted cobra , Cape cobra and Black mamba cause a very significant junctionopathy by blocking neuromuscular transmission through either a pre- or a post synaptic mechanism. This results in a progressive flaccid paralysis that ultimately results in respiratory muscle failure and asphyxiation.

The clinical signs can progress very rapidly with bites by Black mamba causing death within 30 minutes. It is more typical for muscular weakness to begin to occur within an hour of a bite and progress within the next hour or two to the point of death. The progression of muscle failure is usually fairly typical and begins with hind quarter weakness, progresses to fore quarter weakness and then bulbar paralysis, a cranial nerve failure manifesting as an inability to retract the tongue, loss of the swallowing reflex, saliva overflow and mydriasis due to paralysis of the ciliary muscle (cycloplegia) (Fig. 8). Dogs that survive the acute phase of the disease may well develop transient megaoesophagus with dysphagia.

Although morbidity is typically low (cases that recover generally do so without significant sequelae), the mortality rate is high with these venoms. The progression of signs is quick and the outward clinical presentation unremarkable, otherwise apparently healthy dogs may well just be found dead by owners. If there is uncertainty about whether envenomation actually occurred (so called 'dry bites' do occur when a snake bites but does not inject venom) , it is important to check the animal every 10 minutes or so for several hours by taking it out of its cage to check for any signs of muscle weakness. Onset can be delayed and quite rapid when it does occur.

Coagulopathic envenomation

These bites are inflicted by the Boomslang and the Twig snake in South Africa. These snakes carry fixed back fangs and this means they are inefficient biters. They require much longer than other venomous snakes to inflict a proper bite as the snake needs to chew its victim briefly to deposit the venom. Dogs bitten by these snakes usually present at least a day after the bite and the typical presentation is a collapsed dog with spontaneous haemorrhage from the oral mucousae, haematuria and haematochezia.

Dogs are almost always very pale as a result of significant blood loss. The venom is a potent procoagulant resulting is a massive consumptive coagulopathy. This mechanism means that petechial and ecchymotic haemorrhage is not typical. The nature of the bleed tends to be massive and spontaneous. Laboratory evaluation of coagulation is characterised by very prolonged partial thromboplastin (PTT) and prothrombin times (PT). Without very specific treatment in affected dogs, mortality is very high.

Treatment

The treatment applied depends heavily on the type of venom responsible for the presentation.

There is a single polyvalent antivenom manufactured for use in South Africa. It is produced by the South African Vaccine Producers in Sandringham, Johannesburg (http://www.savp.co.za/). It is made

from venom from the Puff Adder, Gaboon Adder, Rinkhals, Green Mamba, Jameson's Mamba, Black Mamba, Cape Cobra, Forest Cobra, Snouted Cobra and Mozambique Spitting Cobra.

The dose of antivenom cannot be calculated on a mg/kg basis as for a traditional drug. In essence what is required is sufficient antivenom to neutralize the amount of venom injected by the snake. This is not known and as such dosing in veterinary patients is usually governed mostly by cost. Even 1 vial given timeously and intravenously can make a critical difference. Up to 8 or more vials may be required to reverse severe envenomation. Administration should be slow (at around 1 vial over 10-15 minutes).

Prognosis is influenced by size (smaller dogs get relatively more venom per kilogram than larger dogs and hence tend have a poorer prognosis). Mild type 1 hypersensitivity reactions to the antivenom may occur. This usually manifest as acute urticaria or angioedema, vomiting or diarrhoea. These reactions are not uncommon but are almost always clinically unimportant.

Cytotoxic envenomation

The majority of dogs presented for Puff adder bites do not require intensive treatment. Most dogs will present with clinically obvious facial and ventral neck swelling (Fig. 1). It is important to remember that the severity of this swelling may increase in the first 24 hours following a bite, so although these dogs may appear minimally affected when presented, their condition may deteriorate after the initial assessment. It is usually prudent to admit these dogs to the hospital, place an intravenous catheter through which a crystalloid fluid (such as Ringers lactate) can be provided at maintenance rates

Additional treatments such as steroids, antibiotics, analgesics and antihistamines are NOT indicated.

The oral cavities of snakes are populated by multiple species of bacteria leading to recommendations for prophylactic antibiotics but studies in people showed a very low incidence of wound infection, leading to the recommendation that antimicrobials be reserved for wounds developing necrosis and abscessation. A study of 102 patients with rattlesnake envenomation showed only one patient which developed an infection, which was secondary to suspected compartment syndrome.

The swelling of the region affected in cytotoxic snake bites is not inflamed, thus anti – inflammatories are not indicated and in fact, NSAIDs may cause acute kidney injury if there is systemic hypotension.

Intra-arterial envenomation in people have been reported to result in rapid deterioration and cardiac arrest.

As long as there are no systemic signs antivenom is not indicated in this patient category. These dogs are usually safe to discharge after 24 – 36 hours of observation.

Deterioration is an indication for the immediate use of the polyvalent antivenom. Antivenom has a beneficial effect even if given after complications begin to develop. This is an expensive treatment and as such many owners will not be able to afford many vials – traditionally we will use as many as the owner can afford to buy (anything from 1 – 8 vials). The antivenom must be given slowly (1 vial over approximately around

Treatment of ulcerative keratitis secondary to snake envenomation

by Dr Izak Venter

Snake venom will lead to severe corneal ulcerations affecting the entire corneal surface in most cases. Fortunately, unless the cornea becomes secondary infected with bacteria it very seldom involves the stroma.

The clinical approach to a patient with a fresh lesion should include application of topical anaesthetic eyedrops and rinsing the cornea with physiological saline to ensure all venom is removed. The patient should then be treated for the resulting corneal ulcer and in most cases secondary uveitis.

Broad spectrum antibiotic eyedrops [ofloxacin q4h] should be started to prevent bacterial adhesion to the exposed cornea stroma. The lesion is typically very painful and 1 % Atropine is administered q12h for no longer than 8 days. Due to the size of the exposed stroma and high risk of melting anti collagenolytic drugs should form part of the treatment protocol. The most commonly used products are topical serum / plasma [q6h] and / or oral Doxycycline 10mg/kg daily. Oral NSAID's should be prescribed for the secondary reflex uveitis.

These cases typically respond well to treatment, but it may take weeks for the cornea to become completely fluorescein negative. If the cornea is still fluorescein positive one week after the incident continue with antibiotic and serum, but topical hyaluronic acid drops can be added at this time as lubrication.

The end result is usually a fluorescein negative cornea, but with moderate to severe corneal vascularization. Once the cornea is fluorescein negative topical prednisilone acetate or dexamethazone should be prescribed to clear the vascularisation.

10-15 minutes) by intravenous injection. Additional supportive measures must be employed as needed.

Severe envenomation requires intensive care support.

1. If upper airway obstruction is present, an endotracheal tube will need to be placed. This may well need to be changed to a tracheostomy tube as the airway obstruction may last for days. A traditional tracheostomy tube will be too short to extend from the skin, through the very swollen neck tissues and into the trachea and as such an ET tube may need to substitute as a tracheostomy tube (Fig. 5).
2. The severe swelling is due to whole blood loss and as such these dogs may become oligaemic and require whole blood transfusions to prevent circulatory collapse. Blood should be administered until heart rate and signs of peripheral perfusion show improvement. If whole blood is not available a synthetic colloid could be used. Crystalloids are the last fluid of choice as much of the infused volume will end up extravascularly within hours of administration and thus not provide sustained volume support.
3. Fluid support may be required to ensure adequate urine production and pressors may be required for blood pressure support.
4. Oxygen supplementation may be helpful
5. Gastro-protectants and prokinetic agents may be considered.
6. If immune mediate haemolysis occurs, prednisolone should be used to induce immune suppression.
7. As with any critically ill patient, 'Kirby's rule of 20' should be considered in providing care.

If compartment syndrome is present then open fasciotomy by incision of the skin and fascia is the most reliable technique for releasing the pressure. An insufficient fasciotomy is as ineffective as a delayed fasciotomy. Complications do include bleeding and sepsis but these are manageable, tissue necrosis is not. Administration of antivenom is also recommended with this complication.

Neurotoxic envenomation:

Any dog bitten by a neurotoxic snake must be admitted for at least 24 hours of observation. Although clinical signs of weakness usually occur with an hour of the bite, some will suddenly show signs hours after a bite. Antivenom is an absolute requirement for dogs showing neurological signs as long-term mechanical ventilation alone is not a practical option. The management of a typical case would follow the following course:

1. The envenomed dog becomes weak (usually in its hind quarters first). At the first sign of this occurring the clinician must prepare for the administration of an intravenous induction anaesthetic (propofol is a reasonable agent) and the endotracheal intubation of the patient. Once the dog is anesthetised an AMBU bag (with or without oxygen) can be used to support ventilation.
2. If the use of antivenom is not an option - observe if the patient does in fact progress to the point where respiratory paralysis occurs. In some patients where smaller amounts of venom were injected, only partial paresis may occur.
3. Maintenance of anaesthesia by means of continuous intravenous anaesthesia is required as the patient is not breathing voluntarily. Propofol is ideal for this purpose (but can be expensive and requires an infusion pump). Pentobarbitone, which is much cheaper and can be titrated in small boluses rather than used as a continuous rate infusion, may also be used.
4. As much polyvalent antivenom must be administered (slowly IV) as what the owner can afford. Administration of antivenom reduces the duration required for ventilation. Even 1 vial makes a difference
5. A plan should be made to supply mechanical ventilation. Depending on the severity of the envenomation and the dose of antivenom given, most dogs will require 8 – 12 hours of ventilation until they can maintain their own spontaneous breathing. Attempts to wean these dogs off ventilation can be attempted from around 8 hours after antivenom administration.
6. Diligent care must be given to airway care. Excessive cuff inflation must be avoided, ET suctioning and cleaning to prevent mucous plugs forming and keeping the oral cavity clean and moist (soaked swabs) must be attended to.
7. Regular turning to prevent pulmonary hypostasis is important.

Manual ventilation with the ambubag or an anaesthetic machine is an option.

1. Care must be taken not to overinflate the lungs and cause barotrauma. The thoracic wall must only slightly expand with each breath. If willing/necessary the owners can be utilised to assist with this.
2. Positioning of the patient is important here to prevent aspiration. Regularly turn from left to sternal to right side, or maintain in sternal of possible. IV Fluids at maintenance rates and keep body temperature stable.
3. Weaning from ventilation, even manual, is essential. Once the patient start trying to breathe on its own ventilation can be slowed down. Patients are generally not able to expand the chest fully to ventilate properly however and also tire easily, so it is inevitable that they will need assisted ventilation again. This cycle will be repeated a few times with longer intervals where the animal maintains by itself This is normal, don't despair.

Prognosis is usually good as long as sufficient antivenom and respiratory support can be provided.
It is a good idea to place these patients on nil per os until 100% recovered to avoid aspiration in the recovery phase.

Coagulopathic envenomation

Bites from the Boomslang can usually be managed very successfully (even in cases of advanced envenomation) by employing the only species-specific monovalent antivenom made in South Africa. A single vial given IV is all that is required,and it is an absolute necessity if the dog is to survive. Blood transfusion support may be necessary but transfusions alone are inadequate. Bites from the Twig snake are fortunately thought to be very rare. There is one report of a bite in a dog and the clinical signs were mild and the dog recovered without any interventions. In serious envenomation's, the only means of treatment would be supportive in the form of blood transfusions.

Take home messages

- Puff adder bites result in blood loss and hypovolaemia. Treatment is aimed at volume replacement. In most cases, especially large dogs, progression of the condition is uneventful and requires minimal intervention.
- Smaller size dogs often present with more severe clinical signs.
- If the case shows severe envenomation with shock and blood transfusion is required or other organ involvement is present, antivenom is required.
- Compartment syndrome can occur if the bite penetrated a muscle belly - usually on the limbs.
- For neurotoxic snake bite cases - first see if neurological signs develop. If they do then only should one start antivenom treatment.
- Any amount of antivenom is helpful in neurotoxic snakebites but you may not be able to use enough to prevent the need for mechanical ventilation. It does however reduce the time during which ventilatory support is needed.
- Monovalent antivenom treatment is essential in clinical Boomslang bites. There is no antivenom effective against Twig snake venom.
- When ventilating - be careful to not cause barotrauma.
- Not all cases with neurological signs progress to paralysis.

A very helpful App for smartphone use is available for free download:
www.africansnakebiteinstitute.com

It is very informative and provides excellent guides of snakes of South Africa, snake identification, first aid and many helpful contacts.

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Reference:
1. Data on file. Pet Owner Submissions, August 2016. Zoetis LLC.

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The ABCs of Veterinary Dentistry

"Y" is for your dental operatory—you've got to take care of it, and it will take care of your patient

An expert in veterinary dentistry outlines the importance of caring for your machines and accessories to facilitate safe, effective dental procedures.

Jan Bellows, DVM, DAVDC, DABVP, FAVD

The dental operatory is an area with special equipment and facilities to perform dental diagnostics and treatment. Care and maintenance of the equipment and materials in this room are essential for successful dental procedures and safe delivery of anaesthesia. This article focuses on getting consistent, accurate results from your anaesthesia monitors and machines through careful inspection and maintenance. The second half of the "Y" is for... series will cover preventive care for your dental delivery system, handpieces, dental surgical instruments, and intraoral imaging sensor(s).

Anaesthesia safety

Anaesthesia safety is probably the top concern on clients' minds regarding dentistry. Your clients may think that because they are not anesthetized when they go to the dentist, why should their dog or cat need general anaesthesia for a case of halitosis? The answer is "to thoroughly examine and treat (any) subgingival disease, which is not possible without anaesthesia." Safely performed, professional oral prevention, assessment, and treatment rely on choosing the right patient, the right anaesthetic protocol, and proper monitoring during and after anaesthesia.

The monitor

Purchasing the best anaesthetic units and monitoring systems is not enough to ensure consistently positive experiences. Out of the box, monitors generally work flawlessly with large and bright electrocardiogram (ECG) tracings, loud and regular audible beeps, the pulse oximeter, and consistent measurements of blood pressure and end tidal CO2. Sometimes, months after the purchase, you look up at the screen and can barely read the ECG, the CO2 end tidal volume alarm is constantly being triggered, and there is no evidence of blood pressure measurements. What is wrong with these units? Generally, the problem with the units is not the manufacturer. It is the way we maintain the peripheral attachments, and how we train our surgical assistants to perform anaesthetic monitoring.

The monitor's accessories are considered consumable items and, based on their direct contact with patients, are subject to wear and tear. They need to be replaced from time to time. This is a harsh reality for many who are used to paying for the highest quality equipment and expecting everything to work seamlessly without much additional effort or expense.

Fig. 1a - SpO2 tip-clip sensor. Not using the tension clip will cause the sensor to fray where the lead meets the sensor. Using a sensor that is frayed can cause injury to the patient and possible burns to the area at which the sensor is applied. It also can cause inaccurate readings and dropped measurements. This sensor is dirty with dried blood; daily cleaning is necessary.

Fig. 1b - Stationary table mount.

How the accessories are cared for directly affects the frequency and expense of replacement. For example, the rate at which we (at All Pets Dental) were replacing pulse oximeter sensors seemed exceedingly high. At $250 each, we needed to do better, and made sure that every staff member was trained and held responsible for their proper care.

What we have learned

- We have learned the hard way to buy monitors manufactured in the United States. For us, buying cheaper monitors from overseas resulted in poor unit life span, inaccurate results, and impossible service. Tip: Buy from US manufacturers that ensure 3- to 5-day repair turnaround.
- Much of the damage to handheld units came after the instruments were accidentally dropped on the floor. Consider options for mounting and protection and opt for table mounts and rubber covers. The cost of 1 repair could easily cover that expense.
- For us, the best approach to monitor responsibility and care was to designate the role of anaesthesia unit and monitor equipment specialist to one of our technician assistants. In addition to meeting with and training all those who handle the monitors, the monitor equipment specialist (on a nonsurgical day) completes a weekly checklist to ensure that each unit is fully functioning. If a unit is not fully functioning, the specialist would bring

Fig. 2 - Training staff on the operation and care of each monitor.

MONITORING EQUIPMENT FUNCTIONALITY CHECKLIST

Date: _____

	Inspect Housing	Powers On	Temperature	CO2	SPO2	Blood Pressure	ECG & Heartrate	Notes
Cardell Touch								
Cardell Max12 HD								
Digicare Lifewindow8 Lite								
PetMap + ECG + ETCO2								
Sentier Vetcorder								
Masimo Rad-57								
Masimo Rad-97								
EMMA								

- **Temperature**: Hold the probe in hand. If the value on the screen rises, the probe is functional.
- **CO2**: With adapter of choice on sensor, allow for 2 minutes of warming once monitor is powered on. If no errors display, take several breaths into the patient side of adapter to ensure breaths display on screen.
- **SPO2**: Place sensor on finger or earlobe to ensure detection of pulsatile blood flow.
- **Blood Pressure**: Ensure cuff size setting on monitor is set to SMALL. Place #2 cuff on thumb and raise cuffed hand to heart level. Blood pressure is obtained while in a seated position with feet touching the floor.
- **ECG:** Hold the white lead between the right thumb and index finger, black lead between the left thumb and index finger, and red lead between the left pinky and ring finger.

Storage & Care Best Practices

✓ Accessories should be wiped down with soap and water between uses. Never submerge accessories in fluid and avoid caustic cleaning solutions.

✓ Replace consumable parts, such as sampling lines, ECG clips, CO2 airway adapters, or blood pressure cuffs, as recommended, or if cracked, damaged or leaking.

Fig. 3 - Weekly monitor checklist after damage/leaking.

this to the attention of the practice manager after calling the vendor for advice.
- Because most practices have monitors from multiple suppliers, consider keeping additional peripherals, blood pressure cuffs, and repair items associated with a particular monitor together in a large transparent plastic container. Care should be taken to avoid any interchange of the accessories from one monitor to another unless they are 100% compatible. This is especially true for power supply/charging units. Even if the connector is compatible, severe damage can occur with a power overload.

Fig. 4 - Transparent plastic containers to keep peripherals for each monitor separate.

Care and maintenance of peripheral accessories

Blood pressure cuffs

When the Velcro on the blood pressure cuff is worn out, it is time for a replacement. Taping the cuff will yield inaccurate readings. In addition, pay close attention to how the blood pressure cuffs are stored after use. They should be disconnected from the unit and stored flat. Storing them on the unit often creates abnormal stress on plastic surfaces.

Tips:
- Keep blood pressure cuffs clean by wiping with alcohol after each use.
- Replace cuffs if damaged, worn, or unable to be thoroughly cleaned or disinfected.
- Replace cuffs if the 2 sides of Velcro do not properly adhere to each other—never tape a cuff.
- Be sure to follow manufacturer guidelines for cuff size and placement.
- Always use the cuffs provided by the manufacturer.
- Never place a cuff on the same limb as the Sp02 sensor orcatheter.

Fig. 5 - Noninvasive blood pressure cuff with hair on the Velcro, making it unable to hold continuous contact. This cuff is dirty and, even after being cleaned, remains discolored. In addition, the hose to this cuff is loose, causing it to have a possible air leak and deliver inaccurate measurements.

CO_2 sampling lines

A good rule of thumb: as soon as an occlusion alarm is triggered, replace the sampling line. If visible moisture is evident beyond the neoprene filter, hang the line to dry. Failure to do so would increase the chances that the moisture will reach the internal module and burn out the circuitry.

Tips:
- Monitors equipped with side stream CO_2 sample lines should be disconnected and the mouthpiece hung downward to dry.
- Never force air through the line to expedite drying, because it can destroy the filter.

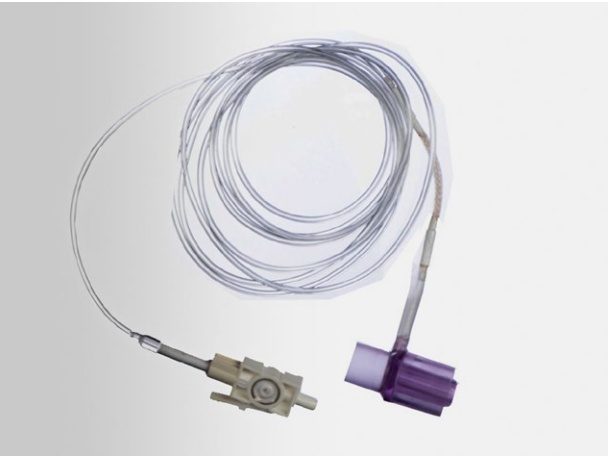

Fig. 6a - The sample line was damaged and then taped. This causes inaccurate measurements. The filter is yellow and dirty because of excessive moisture and use, which will cause inaccurate readings and occlusion in the sample line.

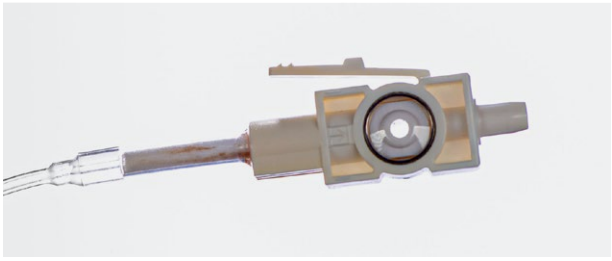

Fig. 6b - Close-up of filter damage.

Fig. 7 - Electrocardiogram (ECG) clip damage secondary to excessive use without cleaning between patients and using alcohol instead of ECG gel, which causes faster corrosion of the metal. This clip is rusted and so is the spring, which can cause damage to the patient's skin as well as inaccurate readings.

ECG leads and clips

Green or black buildup on copper clips can be part of normal oxidation that should be regularly cleaned, or it can mean your staff is mistakenly using ultrasound gel (acoustic gel) rather than electrode paste (conductive) to maintain good contact. Ultrasound gel will not only block the electrical signal but will also quickly degrade the clips.

Tips:

- Clean clips daily by gently wiping with alcohol or by using an instrument brush.
- Never wind tightly to store, and never use ultrasound gel instead of electrode gel.

Pulse oximeter sensor

When the infrared light on the pulse oximeter sensor is intermittent, it is because the thin internal wires are usually frayed or are being pulled apart at the junction, a precursor to complete sensor failure. Always have a spare pulse oximeter sensor. Having a working spare will also allow you to troubleshoot the parameter to eliminate the possibility that the problem resides in the monitor itself.

Tips:

- Wipe the sensor clean with alcohol daily; never use other cleaners that may damage plastic. Never wind tightly or pull clips off when changing them.
- Be sure to unclip before moving the patient.
- Use the tension clip on the cable to avoid damage to the patient's skin and avoid fraying.
- Avoid using the lingual sensor in the mouth during dental procedures; tail sensors are ideal for use during dental procedures.
- To avoid pressure damage, the sensor should be repositioned every 20 minutes.

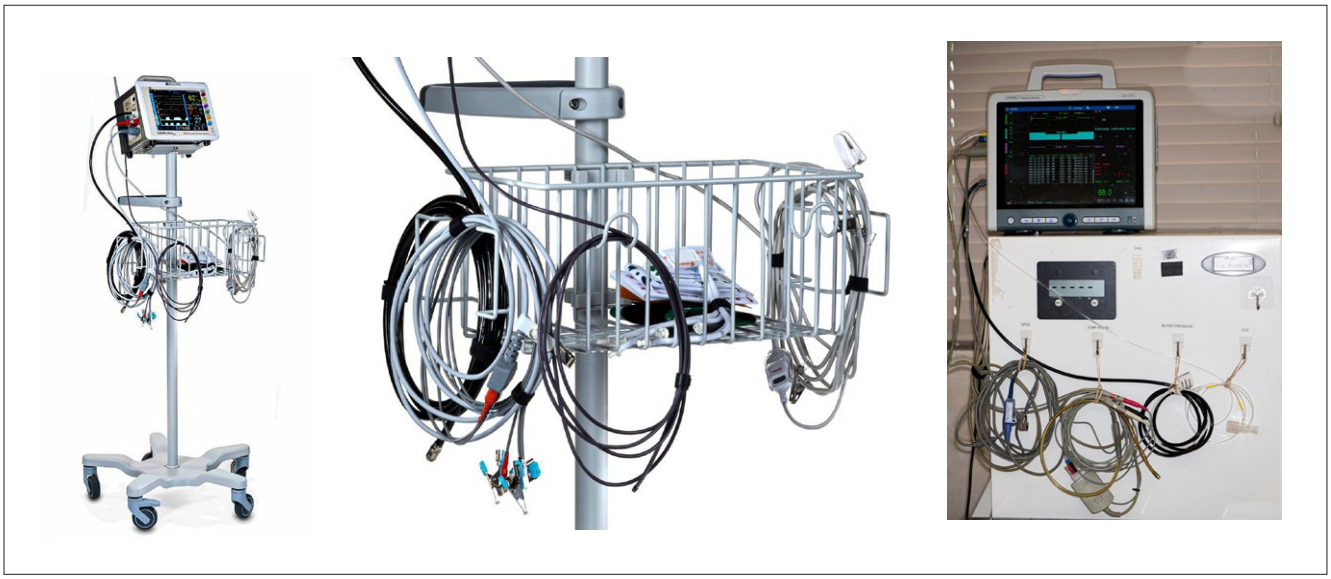

Fig. 8 - Management of peripheral wires after use; a) rolling stand with coiled wire storage (Digicare); b) close-up showing hooks; c) wires attached with wraps on lift table.

Anaesthesia machine

Similar to a pilot examining a plane and completing a checklist, consider adding an anaesthesia unit checkup before the patient is anesthetized. A recently released monitor unit would have the checklist incorporated and allows a manual checkoff before monitoring commences.

The leak test

A leak test should be performed before every procedure. By verifying that there are no leaks in the machine, you allow the technician/doctor to administer the correct flow rate of oxygen and percentage of inhalant drug to the patient. Leaks are not only wasteful but can compromise the safety of the patient and staff.

Tips:

- Perform a leak test between each procedure.
- Replace the maintenance kit at least annually, including the black rubber tubing, domes, gaskets, flutter, and discs.
- The rubber parts should be cleaned daily with mild soap and water.

Breathing bag and breathing circuit

The breathing bag and breathing circuit should be washed and hung to dry after each procedure. If a bag/circuit is used for successive procedures without being cleaned, infections can be passed between patients.

Tips:

- If a tear is evident in a bag or breathing circuit, that particular bag/circuit should be replaced.
- Taping the circuit or bag may continue to allow a leak in the system and waste oxygen and the inhalant drug.

CO_2 absorber

CO2 absorbent granules are designed to "scrub" the expired gases of CO2, and the reaction causes the granular absorbent material to change color (bluish gray or purple) as a visual guide. The absorbent material can be depleted after a matter of hours, and will return almost to its original color overnight, a phenomenon known as regeneration. Depleted granules are hard to the touch and not easily crushed when pressed between the fingers, as compared to fresh absorbent. Channeling of gases may occur, leaving the visible granules against the sides of the canister fresh, while the gases pass through the expired granules toward the center, sending increased levels of CO2 back to the patient.

Tip:

- Keep a log sheet next to the machine and replace the absorbent every 6 to 8 hours of use.

Vaporizer

To ensure the accuracy of anaesthesia delivery, the vaporizer should be professionally serviced and certified on an annual basis by the manufacturer. Some companies have an exchange program through which they send you a reconditioned vaporizer in exchange for the one in current use. Failure to make this exchange could result in patients being too "light" (and waking up in midprocedure) or too "deep," which could compromise vital organs.

Handling the costs of quality maintenance

On all our invoices, there is a line item for hourly anaesthesia monitoring. This allows our practice to update and maintain our anaesthesia and monitoring equipment and accessories. Paying close attention to the care of your monitors makes sense for the patient, staff, and practice.

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[1]Wernimont, S.M., *et al.* Food with Specialised Dietary Fibre Sources Improves Clinical Outcomes in Adult Cats with Constipation or Diarrhoea (in a 2 Month Study). *FASEB J.* 2020;34(1). Some cats may require multimodal management. [2]Fritsch, D.A., *et al.* Food with Novel Fibre Blend Improves Clinical Outcomes and Changes Gastrointestinal Microbiome Metabolism in Dogs (in a 2 Month Study). *J Vet Intern Med.* 2019;33(5):2513.

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