Proceedings

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EERVC

EASTERN EUROPEAN REGIONAL VETERINARY CONFERENCE

Advancing the veterinary profession in Eastern Europe

Welcome to Bucharest for the 2nd Annual Eastern European Regional Veterinary Conference (EERVC)

I am delighted to welcome all new and returning delegates, speakers and exhibitors to the second EERVC. The conference will offer cutting-edge international speakers, the largest regional and international trade exhibition and unparalleled opportunities for professional networking during an affordable, high-quality, 3-day annual meeting.

As for the first conference, the speakers have been selected by recommendation and their expertise. Last year we received fantastic feedback from delegates and we tried to follow your suggestions when considering the speakers and their topics. Lectures cover all aspects of small animal medicine and surgery, as well as topics of current interest for the profession.

The trade exhibition at EERVC 2017 is even larger than last year. It is a fantastic place to discover the newest products from industry, including the very latest innovations and services focused on the needs of the small animal veterinarian. We recommend that you plan a trip to explore this area and make the most of the veterinary industry expertise and their products.

The EERVC Organising Team have prepared lots of fantastic opportunities for networking including the Boehringer Ingelheim Welcome Reception on Friday and the EERVC Party on Saturday Night with the Perpetuum Mobile Band – we hope you can join us and dance until the very late hours!

I thank every individual delegate, coming from nearly 40 countries across the globe; we hope to see you again in the following years of EERVC.

On behalf of the EERVC Board, I would like to thank our exhibitors and sponsors, whose support ensures we can continue to deliver high quality and affordable CPD in this region.

And last but not least, my personal thanks go to the EERVC Project Board and the founding partners from the Small Animal Veterinary Associations of Croatia, Romania and Serbia, working together with the support of the British Small Animal Veterinary Association (BSAVA). The team all work in a voluntary capacity and have worked extremely hard to continue this ambitious mission of advancing the veterinary profession in Eastern Europe.

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Table of Contents

Table of Contents continued

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Pruritic cat – is it always allergy?

MVDr. Lucia Panakova
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Pruritus is undoubtedly he number one dermatologic problem in cats.[1,2] Often these cats have allergic skin disease. Most commonly associated with flea allergy dermatitis if in a flea endemic area, then atopic dermatitis (Non-flea, Non-food allergy), followed by undetermined or food-induced allergic dermatitis. The allergic diseases accounted for 75.6% of 502 chronic pruritic cats.[3] The major non-allergic differentials for chronic, pruritic cats are *Demodex gatoi* infestation, *Cheyletiella* infestation, otocariosis (especially if ear canals are involved) and psychogenic problem. Certainly, *Notoedres cati* infestation is a possible differential diagnosis in cats with pruritus localized on the head/pinnae, but the incidence of this disease dropped down markedly or was completely diminished thanks to a broad use of anti-parasitic drugs in most developed parts of the world. Also the incidence of *Demodex gatoi* is very low, except in regional areas. In these areas, skin scrapings, fecal examinations or even trial therapy with lime sulfur dips or sprays are indicated. Pyoderma is now recognized to also be a problem in pruritic cats, but will be covered in another session. Malassezia overgrowth or dermatitis is a pruritic, secondary disorder in cats. As like as with pyoderma, also here, primary underlying problem should be suspected.

Flea Allergy or Flea-Related Diseases

Fleas need to be ruled out initially in areas where fleas may be present. Flea can induce a variety of lesions in the cat and all these may not really be allergy mediated.

The most classic flea lesion associated with allergy has been milliary dermatitis. This lesion is typified by focal, small 1–5 mm, crusted papules. Depending on the stage, the lesions may appear as erythematous or even hyperpigmented papules. Dorsal involvement raises the probability of fleas but is not required.[3] Cats with flea allergic dermatitis may show clinical signs of head and neck pruritus, eosinophilic granuloma complex (EGC), and also symmetric self- induced alopecia. Other cats may develop symmetric alopecia just from „chasing down" fleas while removing them. This condition might not be real flea allergy, but only flea- related disease.

The advent of newer, more effective, flea control products has been both beneficial and detrimental to clinical practice. The new products have greatly minimized the impact of flea-allergy dermatitis in many practices and allowed for much more effective flea control. The flea control may be good for controlling infestations in the house, but not necessarily effective at stopping flea-allergy dermatitis. Yet, because clients use flea control and do not see fleas, they believe fleas cannot be the cause of disease, making the client acceptance of the diagnosis more problematic. However, especially in cats that spend a significant amount of time outside, this may not be the case.

Treatment for flea-allergy dermatitis and flea-aggravated pruritus is effective control of flea populations and prevention of exposure to adult fleas. Success may be easy in situations where the cat is confined indoors and all cats and dogs in the house are treated with products that both kill fleas and prevent completion of the flea life cycle. A flea treatment trial is best accomplished with cats confined indoors for a one-month trial. It this is impossible, it is even more critical that fast-killing products like, nitenpyram, spinosad, isoxasolines (fluralaner, sarolaner) or the fast-killing topical dinotefuran/pyriproxyfen, imidacloprid. In contrary, what was thought in previous times, pruritus may still continue for several weeks after a good flea control. For this reason, repeated and regular applications of flea products are needed. If pruritus didn´t stop after 2-3 months, further allergy diagnostic is needed.

Allergic Dermatitis (Non-flea, Non-Food Allergic Dermatitis)

The term atopic dermatitis is being debated as to whether it is appropriate in cats or not, but that is what we will use for this lecture. According to the previous studies the abdomen, groin and forelegs are the most common pattern, but generalized pruritus, facial and head pruritus, and even pruritic chin with acne have been seen with atopic dermatitis. Unfortunately, some cats with eosinophilic granuloma complex (eosinophilic ulcer, collagenolytic granuloma and eosinophilic plaque) have also atopic dermatitis; in other words, seing a cat with eosinophilic granuloma complex, one should consider atopic dermatitis and other allergies as reasonable differential diagnoses. A recent review of 502 pruritic cats from 9 countries showed that there is much overlap in the lesions and patterns found in all the causes of pruritus. New criteria for the diagnosis were proposed but have not been particularly helpful.[4] Flea control, cytology, possible skin scrapings and diet trials are all necessary to reach a diagnosis of atopic dermatitis in cats.

Diagnosis of food-induced allergic dermatitis requires elimination-diet trials and provocation testing. A new protein and carbohydrate as the only food source is the primary goal. Other option is an ultra-hydrolyzed, or hydrolyzed, as well as limited-ingredient diets for cats. Other options, especially if cats do not find them palatable, are the limited-ingredient diets with one major protein and green peas or potato as the carbohydrate source.

The true confirmation of food adverse reaction requires that feeding the offending diet can induce symptoms (provocation testing). After symptoms have improved significantly or been eliminated, the pet should be challenged with the diet being fed prior to the diet trial. Diet trials should be done in conjunction with the flea-treatment trial, as both trials are most effective when cats are confined totally indoors. In cases, where the animals are highly pruritic, these can be put on anti-pruritic drugs in the beginning of the dietary trial (e.g. short duration of oral or topical glucocorticoids) in the beginning of the elimination diet. For this reason, oral glucocorticoids are preferred and the dosage might be well tapered down. Other possibility is to eliminate the pruritus first with "big guns" like ciclosporin and to put the cat on the diet. After pruritus is eliminated, the cat is left on the diet and one can try to taper down the ciclosporin. This approach might prolong the elimination diet, but may improve the compliance with the owner. If the drug cannot be withdrawn, even though the cat was on the diet already for 6-8 weeks, food allergy is not probable. If the drug (ciclosporin A) can be stopped, elimination diet must continue for at least another 2-4 weeks, before making provocation test. Flea control must be performed also during the elimination diet.

Non-Food, non-Flea Allergic dermatitis

Once the flea control and food trial were performed and were not successful, the diagnosis of Non-food, non-flea allergy is confirmed. This disease actually is compatible with Feline Atopic Dermatitis.

Treatment for Atopic Dermatitis

The role of allergy testing in the diagnosis has not been established, though it is not considered a diagnostic criterion. Intradermal testing has many false negatives based on comparison to serum tests. According to the latest study, changing the allergen concentrations for pollen allergens in the intradermal tests in cat might help to get more reasonable results from intradermal testing.[7] Even though, the patients show response to allergen-specific immunotherapy (ASIT). Ideally, these patients should get a combination of both tests (intradermal and serum allergy test. According to available studies approximately 60-70% of the cats respond to ASIT. In many cats, ASIT ends up being preferred by the owner because they find giving injections at home more convenient than oral medications. In cats that are difficult to medicate, this is often preferred by clients as many find subcutaneous injections tolerated better than oral therapy.

Glucocorticoid therapy has traditionally been the main treatment for allergic cats, partly because cats are often described as being "resistant" to the adverse effects. Though controlled studies have not been reported, it is accepted that the incidence is probably lower than in dogs and humans, but adverse effects do still occur.

Diabetes mellitus or alterations in glucose metabolism appear to be more common than in dogs. There are also significant numbers of atopic cats that do not respond well, therefore alternative therapies are still being sought. Alternatives to consider include antihistamines, fatty acids, allergen-specific immunotherapy and ciclosporin.

A variety of glucocorticoids and dose regimens have been recommended for cats. Oral methylprednisolone, prednisolone or triamcinolone can be used and are preferred to injectable long term acting steroids, because of lower risk of potential side effects. There are few studies on appropriate doses for allergic disease in cats. One blinded study showed induction doses of methylprednisolone (2 mg/kg daily) and triamcinolone (0.2 mg/kg daily) to be good. Tapering to maintenance of 0.25- 0,5 mg/kg q48h and 0.08 mg/kg EOD is usually possible. If Atopic dermatitis causes a localized disease, topical glucocorticoids e.g. Hydrocortison aceponate are a reasonable treatment option. Along this treatment, ASIT or other GC sparring treatments can be given.

Ciclosporin has been approved for treatment of allergic dermatitis in cats. The liquid formulation comes as 100 mg/ml. It is initially dosed at 7 mg/kg daily until a good response is seen, which is typically in 2–6 weeks in 80% of cases. Generally, it is given with a small amount of meal, but that is not essential. Once a response is seen, the treatment is tapered to EOD, and if remission is maintained (about 70%) for a month, then further tapering may occur. Some cases have tapered to EOD, then lower mg/kg dose even though they could not go to less frequent dosing. Similar to dogs, adverse reactions of vomiting and diarrhea are most common but not a limiting factor in most cases. Gingivitis or gingival hyperplasia may occur. There are rare reports of systemic, fatal toxoplasmosis and it is believed this most likely occurs in cats that have never been exposed to *Toxoplasma* until after being on ciclosporin. In addition, the problem appears to be much more common in male cats (11/13) and the only case to survive was female.[6] Pretesting titers of toxoplasmosis may not be that helpful, but since cases may become positive while on therapy it may be worth monitoring. All systemic cases present with respiratory disease, so owners need to watch for coughing, dyspnea or lethargy. It is strongly recommended to try to limit hunting in cats on ciclosporin, even if seropositive prior to initiating therapy. The incidence in either case appears to be very low. Titers are monitored in cats kept on the drug long term and if IgM titers develop, appropriate therapy is indicated.

Oclacitinib, the JAK-1 kinase inhibitor, is a rather new drug, prescribed for the treatment of pruritus in Canine Atopic Dermatitis. When used for treatment of atopic pruritus in cats at 0,4-0,6mg/kg BID for 14 days and after this, SID pruritus was reduced in less than half of the cats. During the 2017 ESVD-ECVD Conference in Lausanne, Dr. Noli presented a study on atopic cats treated with oclacitinib 1mg/kg BID or methylprednisolon (0,5-1 mg/kg) for 28 days. Both group of patients (oral methylprednisolon or oclacitinib) have shown comparable reduction of pruritus (VAS) and lesions (SCORFAD). In half of the cats treated with oclacitinib 1mg/kg BID, both urea and creatinin have mild increased after 28 days of treatment, even though stayed still within the reference ranges.

Antihistamines may be helpful in treating atopic cats, though I do not see the 50% response rate that some have reported. Examples for antihistamines that seem to work better in cats are: chlorpheniramine at 2 mg q12h and cyproheptadine 2 mg q12h, cetirizine 5 mg q 12–24h (which is considered by some to be more valuable because of its inhibition of eosinophil migration), or clemastine 0.67 mg once a day.

Fatty acids are also indicated for allergic cats. How effective they are is controversial and not well studied. Omega 3 supplementation has been shown to have some benefit.[5] The easiest method of giving this therapy is by selecting diets that have high levels of omega 3 fatty acids in them. My preference is to also select fish oil as a significant source so that eicosapentanoic acid is one of the omega 3s present and fish oil is the best source for it.

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Eosinophilic granuloma complex

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Eosinophilic granuloma complex is actually not a disease, but only a group of symptoms or a group of cutaneous reactive patterns.

For simplification, here I will cover three controversially similar/overlapping patterns (best referred to as 'eosinophilic disease'): Indolent ulcer ("rodent ulcer") (IU); eosinophilic plaque (EP); eosinophilic granuloma (EG).

Background - presence of eosinophilic degranulation products coat but not alter the collagen. Inflammatory reactions of the skin are often associated with hypersensitivity/allergic diseases, but always look for underlying cause. One newer theory on eosinophilic granuloma complex suspect, that eosinophilic granulomas are mainly bacterial infections in a hypersensitive cat. Recently hypothesized that the *Felis domesticus* allergen I (Feld I), the cat allergen contained in feline saliva and hair, may → auto-sensitize cats.

Eosinophils are present in many other feline cutaneous conditions, like pemphigus, neoplasia, viral diseases and pyoderma, so further tests might be necessary (biopsy, viral PCR..).

Indolent Ulcer

Clinical signs - ulcerative mucocutaneous lesion, well circumscribed shiny red-brown-yellow, glistening, painless, **non**-pruritic lesion +/- submandibular lymphadenopathy, upper lip ("dished-out"), near midline, +/- lower lip.

Etiology: Pyoderma in hypersensitive cat, refractory atopy, food hypersensitivity, flea hypersensitivity, genetic defect of eosinophils.

Differential diagnoses – neoplasia (SCC), infectious ulcers (bacterial, fungal, FeLV, FIP), trauma.

Eosinophilic Plaque

Clinical signs - moist raised erythematous papules (coalesce) → orange to yellow plaques, sharply demarcated lesions, cobblestone appearance most/glistening surface (full of eosinophils and mast cells), notice white collagen specks; **intensely** pruritic +/- peripheral lymphadenopathy, ventral abdomen or flank, medial thigh (sometimes)

Etiology – Pyoderma in hypersensitive cats, 90% have underlying allergic (i.e., FAD) hypersensitivity (younger cats).

Differential diagnoses - infectious ulcers (bacterial, fungal, FeLV, FIP), trauma, neoplasia

Eosinophilic Granuloma

Clinical signs

- linear (2–4 mm wide, up to 5 cm long) raised yellow lesion on caudal aspect of hind/ less often front extremities (thighs); sometimes papular to nodular configuration; **non**-pruritic, not uncomfortable
- chin – swelling, not ulcerated, haired
- mouth- verrucous or plaque (surface or tongue/oral cavity/palate)
- pinnae/ perianal- papules
- foot pads/paws (e.g., cat litter reaction???) 1 or 2 feet with ulcerative, sometimes papules, crust or even pustule, nodules). In a subset of this disease: associated with mosquito bites (nodules +/- ulceration

Etiology - in hypersensitive cat, refractory atopy, food hypersensitivity, flea hypersensitivity, genetic defect of eosinophils, not well understood, often in young animals.

Differential diagnoses: Dependent on the location and appearance: infectious ulcers (bacterial, fungal, FeLV, FIP), trauma (SCC, fibrosarcoma in oral cavity), neoplasia, plasma cell pododermatitis (feet), xanthoma (feet); chin acne (chin)

Diagnostics for Eosinophilic Disease

- Cytology: determine bacteria burden (swab, aspirate, impression) vs. eosinophils and in case of pyoderma, treat to rule out poyderma
- Punch biopsy: to differentiate from neoplasias, infections
- Culture and sensitivity might be necessary in case of pyoderma

- Blood work (CBC): look at blood composition (blood and tissue eosinophilia common in granulomas and plaques)
- Dermatopathology: normally with flame figures formation; **Granuloma:** palisading granulomatous dermatitis with multifocal areas of collagen **coated with the released substances from degranulated eosinophils** (formerly known as 'collagen degeneration' or 'collagenolysis'); eosinophils common in facial/oral biopsies

Management of eosinophilic disease

- Treat secondary infection first (systemic antibiotics)!
- Control/manage allergies (flea preventatives, food trial, immunotherapy)!!!

- If the treatment of pyoderma didn't heal the lesion completely, additional treatment is needed ➔
  - ➔ Glucocorticoids (2mg/kg prednisolone) initially, later taper down
  - ➔ Ciclosporine (Atopica®)-25 mg PO (7 mg/kg PO/day) can be applied in recurrent cases; rule out toxoplasmosis (IgG or IgM titers); Semiannual CBC, chemistry, and urinalysis
  - ➔ Laser, laser surgery, photodynamic therapy, INF-alpha...

Summary

This lecture brings a common symptom in feline dermatology (Eosinophilic Granulom Complex), in which etiology, secondary infections and allergies play the major role. This lecture has provided a large spectrum of clinical appearances of so called Eosinophilic Granulom Complex, with theirs differential diagnoses. Etiology is until today not fully elucidated.

Feline pyoderma

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Overview of the Issue

Other than abscesses, pyoderma has been considered uncommon in cats. Lesions such as eosinophilic plaques, eosinophilic ulcers and miliary crusts are often attributed to allergic skin disease. Though these lesions do occur most commonly in allergic cats, the actual lesion often represents a pyoderma. The opinion of different authors on the topic of eosinophilic dermatitis and pyoderma differs markedly.

Key Etiologic and Pathophysiologic Points

Staphylococcus pseudintermedius is the most common bacteria associated with these lesions. The lesions often occur from licking or scratching the skin, which occurs from pruritus associated with allergic disease. Similar to dogs, the Pyoderma in cats is most often secondary. If the underlying disease is put in remission the Pyoderma may resolve without specific treatment. The incidence of this is unknown, but appears to be much more common than in dogs. If the pyoderma becomes severe enough or long term glucocorticoids are used, then this may change so that the pyoderma will require specific therapy.

Pyoderma in cats often occurs secondary to immunosuppression e.g. in cats treated for pruritus or any other reasons with steroids. Feline viral diseases e.g. herpes-viral dermatitis might also result in secondary pyoderma.

Rather rare cause for pyoderma in cats is a keratinization disorder. Severe cases of feline acne or idiopathic facial dermatitis and might develop secondary pyoderma and need systemic treatment.

Key Clinical Diagnostic Points

The main feature of pyoderma lesions are moist or exudative surface. In areas where the cat can lick, erythematous erosions are often seen in linear patterns with oval shapes. Self- induced alopecia at or adjacent to these lesions are common. These appear as eosinophilic plaques which should always be assumed are pyoderma and only considered sterile allergic lesions, if cytology reveals no bacteria with neutrophils. Pyoderma lesion cytology will usually reveal a mixture of neutrophils, eosinophils and a few mononuclear cells or macrophages. Intracellular bacteria may be seen but generally the cocci will be extracellular. In areas where licking is difficult, scratching generally results in excoriations. When a cat licks excessively, the tongue may result in areas of cheilitis which can become infected. Once the lip becomes infected it may swell and then be more prone to trauma from licking. According to an Australian retrospective study, superficial pyodermas in cats were presented as multifocal lesions affecting the face (62%), neck (37%), limbs (33%) and ventral abdomen (29%). Crusting (83%), alopecia (67%), ulceration / erosions (54%) and erythema (46%) were common lesion types.

Making a diagnosis

Cytology is the key to making this diagnosis. Bacteria are often seen intracellulary and also extracellulary. Eosinophiles, neutrophiles and often macrophages are present. Treatment response will confirm the suspect diagnosis.

Key Therapeutic Points

The pyoderma lesions respond to systemic antimicrobial therapy, though lip lesions are slow to resolve and may require 4–8 weeks of therapy.

Key Drugs, Dosages and Indications

Key drug	Dose range	Route/ frequency	Comments
Amoxicillin/ clavulanate	62.5 mg/cat < 7kg 125 mg/cat > 7kg	PO Q12h	Well tolerated, bid dose effects compliance
Cefalexin	25mg/kg	PO Q12h	Well tolerated, bid dose effects compliance
Cefovecin	8 mg/kg	Subq Q14 days	Improves compliance, two treatments preferred
Clindamycin HCL	11mg/kg	PO BID	Sometimes better in thick scarred lesions
Doxycycline HCL	10 mg/kg	PO Q24h	Make liquid or give water following dose
Marbofloxacin	2.75–5.5 mg/kg	PO Q24h	Preferred fluoroquinolone with lower potential retinal concerns

Key Prognostic Points

Control of underlying causes of pruritus is key to long term remission of pyoderma in cats with allergic dermatitis. In immunosuppressive treatment induced pyoderma, pyoderma will have a good prognosis, as long as steroids withdrawal is possible.

Summary

Eosinophilic plaques and eosinophilic lip ulcers often represent pyoderma in the cat and respond to appropriate antibiotic therapy. Allergy is a common underlying primary disease and led to the belief these lesions represented allergy in cats. Imunosuppression with steroids is certainly another reason for pyoderma in cats. These patients often need and respond very well on systemic antibiotics.

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Dermatophytosis : an update

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The World Association of Veterinary Dermatology has produced clinical practice guidelines on dermatophytosis. These have recently been published in Veterinary Dermatology. Recommendations from these guidelines are made on the basis of evidence based information. This lecture aims to summarize the findings and recommendations of this paper.

Dermatophytosis in companion animals is a common skin disease caused by a superficial fungal infection of keratinized skin structures. Fungal organisms may be zoophilic, geophilic, or anthrophilic, most commonly *Microsporum canis, Microsporum gypseum*, and *Trichophyton mentagrophytes*.

Clinical Signs: The clinical signs of dermatophytosis reflect the follicular pathogenesis of the disease. Lesions though can present with any combination of hair loss, papules, scales, crusts, erythema, follicular plugging, and hyperpigmentation. Typically lesions are asymmetrical. Pruritus is variable and self-trauma can mimic areas of pyotraumatic dermatitis or ulcerative eosinophilic lesions in cats. Feline dermatophytosis most commonly affects the face, ears and muzzle of cats before progressing to other body areas (1). Dermatophytosis is transmitted via direct contact with another infected host or contaminated fomite. Hunting dogs especially Jack Russel terriers may develop lesions on the muzzle and paws (2). Nail involvement characterized by onychogryphosis on one to multiple digits may occur.

Diagnosis: In all cases of dermatophytosis, the question is not "what is the gold standard test", but rather what test(s) confirm the **presence** of an active infection i.e. the animal needs treatment and what test or tests confirm the **absence** of an active infection, i.e. the animal poses no infection risk and that the animal is cured? In order to make a diagnosis a range of complementary diagnostic tests should be employed which includes Wood's lamp of infected material; direct examination of hairs in potassium hydroxide or lactophenol cotton blue to document active infection; dermoscopy, dermatophyte culture by hair coat brushings (toothbrush or carpet square technique), hair plucks or tape strips to diagnose the fungal species involved and monitor response to therapy and biopsy with special fungal stains for nodular or atypical infections. PCR detection of dermatophyte DNA can be helpful, however a positive PCR does not necessarily indicate active infection, as dead fungal organisms from a successfully treated infection will still be picked up on PCR, as will non-infected fomite carriers. Monitoring of response to therapy includes clinical response, use of Wood's lamp if possible, and fungal culture. Mycological cure is normally defined as two negative cultures 7 days apart.

Treatment

Topical therapy decreases the infectious, contagious, and zoonotic risks associated with dermatophytosis by disinfecting the hair coat and minimizing contamination of the environment. Twice weekly application of lime sulphur, enilconazole or a miconazole/chlorhexidine shampoo are currently recommended as effective topical therapies in the treatment of generalized dermatophytosis in cats and dogs (3-6).

Systemic therapy is important in generalised disease. The most commonly used systemic antifungal drugs for dermatophytosis in veterinary medicine are itraconazole, ketoconazole, terbinafine, and griseofulvin. Itraconazole and terbinafine are the most effective and safe treatments for dermatophytosis. Numerous studies have documented the use of itraconazole in cats to treat *M canis* either as sole therapy or in combination with topical drugs (5-9). Two commonly used protocols are those of Carlotti (2010) who recommended 5mg/kg orally for a week on and week off until mycological cure was obtained. In his study this was 56 days (9). A second protocol is one recommended by Newbury (2011) who used Itraconazole at a dose 0f 10mg/kg daily for 21 days with either lime sulphur or a chlorhexidine/miconazole product twice weekly. Mycological cure was achieved in 36 days (6). Terbinafine is not licensed for the treatment of dermatophytosis in dogs and cats however numerous studies have detailed its efficacy (10-14). The most recent study by Newbury (2015) suggests 20-40mg/kg orally daily for 21 days with lime sulphur twice weekly as an effective way to resolve infection in a shelter environment (15). Ketoconazole, fluconazole are less effective treatment options. Griseofulvin is effective but also has more potential side effects compared to itraconazole and terbinafine. Lufenuron has no in vitro efficacy against dermatophytes and has no place in the treatment of dermatophytosis (16, 17). Antifungal vaccines do not protect against challenge exposure but may be a useful adjunct therapy (18-20).

Environmental therapy helps to minimize the risk of disease transmission to people and other animals. Disinfection of non-porous surfaces involves three steps. The first is the mechanical removal of all debris via vacuuming or sweeping. Disinfectants will not work in the presence of organic debris. The second is the washing of the target surface with a detergent until the area if visibly clean. Detergents must be rinsed from the target surface because some may inactivate disinfectants. These two steps are the most important and in many

cases alone will decontaminate a surface as has been shown in shelter situation. The final step is the application of a disinfectant to kill any residual spores. Disinfectants that have been shown to be useful include sodium hypochlorite, enilconazole and accelerated hydrogen peroxide (21-23).

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Differential diagnoses of feline facial dermatoses

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Aetiology and Pathogenesis

Many different feline skin problems can affect the face. There are some dermatological problems that tend to affect this area more commonly than others. Table 1 is not an exhaustive list but gives an indication of the most common and the breadth of different problems that should be considered for this disease presentation.

Category of disease	Disease
Infectious dermatoses– viral, bacterial, fungal disease	Pox virus infection, herpes virus infection, calici-virus infection, erythema multiforme associated with herpes infection, papillomavirus infection, bite abscess, acne, atypical mycobacterial infection, dermatophytosis, Malassezia dermatitis
Parasitic dermatoses	Demodicosis, trombiculiasis, otodectic mange, notoedric mange, cheyletiella
Allergic dermatoses	Atopic dermatitis, food intolerance, allergic contact dermatitis
Eosinophilic granuloma complex	Indolent/ rodent ulcer, eosinophilic granulomas
Autoimmune disease / immune mediated dermatoses	Pemphigus foliaceus, pemphigus erythematosus, discoid lupus erythematosus, systemic lupus erythematosus, drug eruptions, vasculitis, auricular polychondritis, sebaceous adenitis
Pigmentary dermatoses	Vitiligo
Environmental dermatoses	Burns, frostbite, actinic dermatitis
Neoplastic dermatoses	Squamous cell carcinoma, basal cell tumour, fibrosarcoma, epitheliotropic lymphoma, mast cell tumours
Facial disease associated with internal disease	Necrolytic migratory erythema, paraneoplastic exfoliative dermatitis
Idiopathic diseases	Idiopathic facial dermatitis of the Persian, Feline orofacial pain syndrome (FOPS)

History

A thorough history is important and can give useful clues. Autoimmune skin disease usually progresses rapidly, allergy and ectoparasites are often more insidious in onset. Breeds such as the Persian are predisposed to dermatophytosis, they are also affected by the poorly understood disease idiopathic facial dermatoses of the Persian. Feline orofacial pain syndrome is a dramatic disease which manifests as facial discomfort. The disease is usually unilateral and affected young cats, who will typically paw at their mouth and cheeks. It is most commonly seen in Burmese and cats of Burmese ancestry such as the Tonkinese. Young cats are prone to dermatophytosis and parasitic diseases; older cats are more likely to develop neoplastic diseases such as squamous cell carcinoma or epitheliotropic lymphoma. Hair and skin colour also predispose to some diseases. White cats are more likely to develop sun light induced diseases such as actinic dermatoses, squamous cell carcinoma, haemangiosarcoma and discoid lupus erythematosus.. Where there is evidence of contagion to other animals and humans then such diseases as dermatophytosis and cheyletiella should be investigated. Animals with food intolerances often have associated gastrointestinal diseases. Cats with signs of debilitation and weight loss with facial lesions should be assessed for pancreatic or hepatic disease.

Clinical examination

In addition to a dermatological examination, a full clinical examination should be undertaken for every case. Pruritic facial dermatoses leading to self-inflicted trauma to the face are most commonly associated with allergy and ectoparasitic diseases. Although both atopic dermatitis and food intolerance can affect the face of a cat the latter is the more common clinical presentation. Viral disease can also be pruritic especially pox virus

infections, where the initial presenting sign can be a wound on the face through a rodent bite leading to more generalised maculopapular lesions within a few days. None pruritic diseases that often have erythema and scale associated with them includes dermatophytosis, demodicosis and neoplasia especially epithelitropic lymphoma as well as some of the rarer diseases such as paraneoplastic exfoliative dermatitis and lymphocytic mural folliculitis. None pruritic disease that can present with ulceration and crusting includes autoimmune skin disease such as discoid lupus erythematosus, pemphigus foliaceus and drug eruptions. Involvement of the lips is suggestive of indolent or rodent ulcer; squamous cell carcinoma or cryptococcosis. Nodular lesions are most consistent with neoplasia especially mast cell tumours or squamous cell carcinomas, deep fungal infection especially cryptococcosis; or bacterial disease.

Diagnostic tests

Although *Notoedres cati* is rare in the UK, skin scrapes should be performed to look for this as well as demodex spp. and *Otodectes cynotis* mites. Wood's lamp examination will reveal signs of apple green fluorescent hairs if *Microsporum canis* is present. Rodent ringworm *Trichophyton mentagrophytes* will not fluoresce, so direct microscopy of hairs and fungal culture should be performed. Cytology from lesions can help identify infection or abnormal cells such as acanthocytes in pemphigus foliaceus or neoplastic cells. Fine needle aspirates of nodular lesions may help give a clue to their aetiology, however skin biopsy is necessary in most cases to give a definitive diagnosis. Where infection is suspected special stains can help to identify specific organism or samples submitted in sterile saline can be taken for tissue culture. Other diagnostic tests that may be useful include FeLV, FIV testing, viral culture abdominal ultrasound or pulmonary radiographs.

Therapy

The specific therapy of the different diseases will of course depend on the diagnosis. A prolonged discussion on therapy is beyond the remit of this paper.

Differential diagnoses of feline pododermatitis

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Aetiology and pathogenesis

Almost any feline skin problem can affect the feet; however there are some diseases which tend to affect this area more commonly than others. Table 1 is not an exhaustive list but gives an indication of the most common and the breadth of different problems that should be considered for this disease presentation.

Table 1 Aetiology of feline pododermatitis

Category of disease	Disease
Infectious dermatoses – viral, bacterial, fungal disease	Pox virus infection, herpes virus infection, calici-virus infection, erythema multiforme associated with herpes infection, bacterial paronychia, atypical mycobacterial infection, dermatophytosis, Malassezia dermatitis, candidiasis.
Parasitic dermatoses	Demodicosis, trombiculiasis
Allergic dermatoses	Atopic dermatitis, food intolerance, allergic contact dermatitis
Eosinophilic granuloma complex	Eosinophilic plaques and granulomas
Autoimmune disease / immune mediated dermatoses	Pemphigus foliaceus, discoid lupus erythematosus, systemic lupus erythematosus, drug eruptions, vasculitis, plasma cell pododermatitis, mucinous lymphocytic mural folliculitis.
Pigmentary dermatoses	Vitiligo
Environmental dermatoses	Burns, frostbite, irritant contact dermatitis
Neoplastic dermatoses	Squamous cell carcinoma, fibrosarcoma, metastatic bronchial carcinoma, epitheliotropic lymphoma
Pododermatitis associated with internal disease	Necrolytic migratory erythema, paraneoplastic exfoliative dermatitis,

History

A thorough history is important and factors such as breed, age of onset, life style, presence of systemic illness, response to previous therapy and contagion to other animals and people can give useful clues. Breeds such as the Persian are predisposed to dermatophytosis, vitiligo is most commonly seen in the Siamese. Young animals are prone to viral, fungal and parasitic diseases as well as allergy and congenital problems. Hunting cats have a greater tendency to pick up dermatophytosis and pox virus through contact with rodents. Where the cat has a history of gastro-intestinal signs, weight loss or respiratory disease then systemic disease may be underlying the pedal lesions.

Clinical examination

A full clinical examination should be undertaken for every case. In addition the distribution and types of lesions may help narrow down the potential list of differential diagnoses. Infections, neoplastic disease, trauma and burns tend to affect a single foot; whereas multiple feet are involved with autoimmune disease, allergy and where systemic disease is a cause of the pododermatitis. When the condition is pruritic allergy, including components of the eosinophilic granuloma complex should be considered; together with ectoparasites and viral disease. Diseases that present with erythema and scaling include dermatophytosis, allergy and demodicosis. Ulcerative lesions are seen with autoimmune dermatosis, deep bacterial infections, viral infection and neoplasia. Crusts are commonly associated with pemphigus foliaceus and erythema multiforme. Nodular disease is seen with neoplasia, eosinophilic granuloma complex and deep infectious lesions with bacteria or fungi.

Diagnostic tests

As both ectoparasites and dermatophytosis are common causes of skin disease in the cat, skin scrapings and

mycological investigations should be undertaken in every case. Skin scrapes should be taken from the nail beds to look for demodex cati or gatoi mites. Wood's lamp examination of hairs to look for the typical apple green fluorescence seen is useful to rule out isolates of *Microsporum canis.* Direct microscopic examination of hairs mounted in either potassium hydroxide or lactophenol cotton blue is useful to look for fungal hyphae and spores. Fungal culture of hairs and scale should be performed in all cats. Cytology from exudative lesion, crust or ulcers can identify infection or the presence of abnormal cells such as acanthocytes in pemphigus foliaceus or neoplastic cells where tumours are present. Skin biopsy is necessary where nodular and ulcerative lesions are present. Where lesions are large care should be taken to ensure the biopsy is taken from the periphery of lesions to include both normal and affected skin. Where infection is suspected tissue culture is useful and the laboratory should be primed to look for specific causes of deep infectious disease which can require special culture conditions. Other diagnostic tests will depend on the clinical presentation but may include FeLV, FIV testing, viral culture, abdominal ultrasound or pulmonary radiographs.

Therapy

Specific therapy will of course depend on the diagnosis in each case and is beyond the scope of this paper.

Emergency

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Treating shock: part one

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The Emergency Phase

When a patient has clinical signs of shock, attempts to restore circulating volume starts with crystalloid fluids. Circulatory shock can be due to inadequate volume, poor cardiac function, maldistribution of blood flow or a combination. The initial "shock volume" of fluids is often given and serves to answer the question: "Is this patient volume responsive?" Aggressive fluid loading has the potential to cause harm. One of the worst things we can do is cause pulmonary edema when pushing fluids too quickly. It is essential for all members of the team to be cognizant of the possibility and aware of signs of early fluid overload.

The "shock" volume of fluid necessary to reverse the signs of shock is different for every patient. Rather than giving a full blood volume each hour of crystalloid solutions it is safer to carefully titrate fluids while observing the patient for evidence of resolution and fluid overload. Rather than giving textbook shock volumes (In the dog this has been 90 ml/Kg/hr and for the cat about 44 ml/Kg/hr) we recommend giving 25% of this volume to find out if the patient is volume responsive. Once we see clinical resolution of shock (normal heart rate, improved pulse quality, normal capillary refill) we can move to the second phase of fluid resuscitation (dehydration phase).

One must be cautious about overhydration, hemodilution and hypothermia. Overhydration during the emergency phase is most likely to occur when large volumes are administered to animals with pulmonary contusions, preexisting pulmonary edema, aspiration pneumonitis, hypoproteinemia, brain injuries, and congestive heart failure.

Hypovolemic Shock

The problem in hypovolemic shock is an inadequate circulating volume. This can be from sudden massive blood loss as in surgery or trauma or fluid loss from vomiting, diarrhea or renal disease. Because cardiac output relies on stroke volume and heart rate, the patient with inadequate volume will be tachycardic to compensate. Neurohormonal pathways detecting a drop in blood pressure will lead to increased vascular tone in an attempt to shunt circulation from the periphery to vital tissue beds. This results in cool extremities, tachycardia, prolonged capillary refill, oliguria and weakness.

Treatment should be directed at the primary source of fluid loss while correcting the fluid deficit. Crystalloid fluids can be used initially to restore circulating volume. Crystalloids will improve cardiac output and should not be withheld for fear of diluting the red blood cell mass. Oxygen delivery is a function not only of oxygen content but of cardiac output as well. With a treatment goal of improving oxygen delivery to the tissues we can increase cardiac output by increasing stroke volume (fluids). Oxygen content can be increased by increasing the hemoglobin concentration (Red cell transfusion) and increasing oxygen saturation (Oxygen supplementation).

Volumes of fluid for resuscitation should be tailored to the individual patient. An initial goal with crystalloid fluids is to give a blood volume (40 ml/lb) in an hour. This is often more than enough fluid and in extremely debilitated patients may lead to fluid overload (pulmonary and cerebral edema). It may be more practical to titrate this dose in 1/4 increments. (makes the math easier too!). Give 10 ml/lb of crystalloids rapidly and reassess the patient's clinical signs. Are the pulses stronger? Slower? Is the patient more alert? If not and we determine the shock state still exists give another 10 ml/lb.

Following the second dose of fluids the packed cell volume and total solids should be compared to prefluid values. If a patient receiving large quantities of crystalloids becomes anemic or hypoproteinemic, the fluid should be switched to an appropriate colloid such as whole blood, packed red blood cells, plasma or a synthetic product like hetastarch or dextrans. If the total solids have dropped to less than 50% of pretreatment a colloid should be considered for further resuscitation. If the PCV has dropped precipitously, whole blood and a search for the source of blood loss is indicated. Often, in the case of traumatic hemorrhage, correction of blood loss and pressure can open torn vessels leading to more hemorrhage. Therefore close attention is important. Once the shock is controlled, fluid deficits can be replaced along with maintenance volumes and ongoing losses over the course of one to two days.

Crystalloid Fluids for Resuscitation

Crystalloid fluids are mixtures of sodium chloride and other physiologically active solutes. They are generally isotonic with plasma and have sodium as their major osmotically active particle. The distribution of sodium determines the distribution of infused crystalloid fluids. Sodium is the major solute in the extracellular space and 75% of the extracellular space is extravascular. Therefore, infused sodium will reside primarily outside the vascular compartment.

Colloids

Coloids are fluid solutions containing large molecules help to pull and water within the vascular space. Natural colloids include plasma, whole blood, and albumin transfusions. The albumin in natural colloids displaces water, stays in the vasculature and maintains an oncotic gradient keeping water in the vessels. VetStarch, hetastarch and similar fluids are synthetic colloids relying on large sugar molecules to protect oncotic pressure when albumin levels are low. They have been used when hypoproteinemic patients continue to need fluids and those fluids are leaking out of the vessels into the surrounding tissue. Previously recommended in patients with total protein values below 4.5 g/dl, studies in people linking colloid use to acute renal injury and coagulation defects have caused clinicians to rethink their use. Instead, criticalists now tend to favor of a more conservative approach with crystalloid fluids and blood products.

Hypertonic Saline

The use of concentrated crystalloid solutions is appealing because of the reduced volumes of fluid required. This decreases the risks of pulmonary edema and the need for specialized equipment for delivery of very large volumes of fluids. Combining hypertonic saline with something like 6% dextran-70, Hetastarch or VetStarch will prolong the response. Hypertonic saline was popular for rapid volume replacement with 4 ml/kg quickly giving the volume expanding effects of a 90 ml/kg isotonic crystalloid. More recently hypertonic saline has been used to improve microvascular blood flow. It is dehydrating and can shrink swollen endothelial cells improving blood flow at the tissue level. This effect is seen at a lower dose and it is currently used at a dose of 1-2 ml/kg for such conditions as head trauma and organ dysfunction.

The Replacement Phase

The volume of fluid administered during the dehydration phase is based on an assessment of fluid needs for (1) returning the patient's status to normal (deficit volume), (2) replacing normal ongoing losses (maintenance volume), and (3) replacing continuing abnormal losses (continuing losses volume). Maintenance volumes are normal ongoing losses. Ongoing losses are divided into sensible and insensible losses. Sensible losses can be measured and are water losses in the urine and feces. Insensible losses are normal but are not easily quantitated. These water losses occur during panting or sweating. One-third of the maintenance volume is made up of the insensible volumes and two-thirds, sensible volumes. Traditionally, maintenance volumes have been estimated at about 66 ml/kg/day, or 30 ml/lb/day.

The Maintenance Phase

The last phase of fluid therapy is the maintenance phase. At this point the patient has received enough fluid to compensate for shock (if necessary) and has had a partial replacement of any deficit volume. Chronologically, this phase begins no sooner than 24 hours after fluids were begun. Objective signals that the patient is ready to be placed in the maintenance phase are an absence of clinical signs of shock or dehydration, and the body weight will have increased by at least the percentage of dehydration already corrected. During the maintenance phase, you will be providing both maintenance volumes and continuing losses volumes.

Treating shock: part two

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Introduction

Circulatory shock is divided into 3 major classifications; hypovolemic shock, cardiogenic shock or pump failure, and distributive shock. Though the mechanisms for each are distinctly different, each results in **reduced oxygen delivery** (DO_2) to tissues through low blood flow or uneven distribution of flow. In actual practice, each primary event can lead to a cascade of complex physiologic problems, neurohormonal compensations and cascades that activate various biochemical mediators and inflammatory responses integral to the shock syndromes. A single patient may have several pathologic processes simultaneously resulting in reduced perfusion of tissues. We have already discussed hypovolemic shock in a previous lecture.

Cardiogenic Shock

Cardiogenic shock occurs when the pumping function of the heart is severely impaired leading to circulatory failure. As with hypovolemic shock, the patient will be tachycardic, weak, oliguric, have cool extremities and weak pulses. The patient with cardiac failure may also have evidence of cardiac disease with a murmur, ascites, jugular venous distention, pulmonary edema or cardiac arrhythmias. The primary defect in oxygen delivery is a reduced cardiac output.

Cardiac Output (CO) = Heart rate x Stroke volume*

*Stroke Volume is determined by preload, afterload and contractility

Within limits, cardiac output increases as heart rate increases. Very high heart rates actually decrease cardiac output by impairing cardiac filling and subsequently stroke volume. Excessively fast heart rates may be the result of cardiac arrhythmias or physiologic responses to low volume. Specific antiarrhythmic therapy and correction of underlying causes of tachycardia should be used to normalize heart rate. Clinically significant bradyarrhythmias are less common but include sick sinus syndrome and third degree atrioventricular block. It is uncommon for these slow heart rates to require emergency treatment. Often these patients have compensated with increased stroke volume and can be referred for pacemaker treatment.

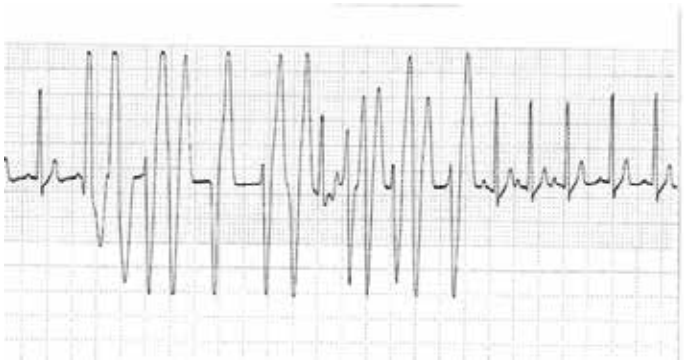
Stroke volume is dependent upon three determinants of cardiac function: Preload, afterload and contractility. With congestive heart failure, the pump is failing due to decreased contractility. The body attempts to compensate by increasing pre-load (sodium and fluid retention). Normally, the heart is able to pump all fluid presented to it through the Frank-Starling mechanism (increase stretch leading to increased contractility) so that by increasing pre-load, the heart will increase stroke volume. With failure however, the excess fluid cannot be moved and accumulates downstream of the failing ventricle. This results in pulmonary edema in the case of left-ventricular failure and ascities, pleural effusion and hepatic congestion in the case of right-ventricular failure.

Stroke volume (and cardiac output) can be maximized by recognizing and treating the primary defect. In the case of congestive failure, pre-load can be optimized by monitoring central venous pressure, administering diuretics like furosemide and venodilators such as nitroglycerine. With obstructive failure as is seen with pericardial effusion, removal of even a small amount of pericardial fluid will relieve the pressure on the right ventricle and allow more normal filling. Cardiac output can also be enhanced by decreasing afterload with calcium channel blockers or ACE inhibitors. These are especially useful in treating failure due to mitral insufficiency where contractility may be normal to increased but the cardiac output is going backwards into the left atrium instead of to systemic circulation. In documented myocardial failure, contractility can be enhanced with positive inotropic drugs such as digoxin or dobutamine.

Ventricular ectopy and ventricular tachycardia can often be diagnosed without an electrocardiogram. One should get in the habit of palpating pulses while listening to the heart. Early heart sounds without a corresponding pulse are typical of the dropped beats of premature ventricular contractions. A run of ventricular beats and fast than normal heart rates is termed ventricular tachycardia. Because the beats are initiated in the ventricular muscle and not within the conduction system the complexes are much wider than normal.

The ventricular arrhythmia is likely a sign that the heart muscle isn't happy. Much the way azotemia indicates renal malaise or a high bilirubin suggest hepatic disease, ventricular arrhythmias indicate cardiac dissatisfaction. Treatment of ventricular tachycardia is directed at several fronts. Specific antiarrhythmics and all possible underlying causes. In cases of clinically significant v-tach (weak pulses, evidence of poor perfusion, R on T pacing) a ventricular antiarrhythmic such as lidocaine or procainamide are usually started. Note: Lidocaine is initially dosed as a bolus, but then followed up by continuous infusion. Patients with v-tach and ventricular ectopic activity should also be evaluated for ineffective oxygen delivery (shock! Check volume status, and

oxygenation). Because hypokalemia and hypomagnesemia are associated with ventricular arrhythmias, these electrolytes should be evaluated and supplemented as necessary. Stress and anxiety can increase circulating catecholamines. Since catecholamines lower the cardiac threshold for ventricular arrhythmias the careful use of appropriate analgesic and sedative drugs can go a long way toward resolving the arrhythmias.



Malignant V-tach with R-on-T pacing

Distributive Shock

Distributive shock is probably the most challenging of the shock syndromes and one of the most difficult to reverse. The defect with distributive shock is an abnormal or systemic vasomotor response leading to peripheral vasodilation and a maldistribution of blood flow. There may also be increased vascular permeability. Both of which result in decreased perfusion of vital tissues. There can be components of the other forms of shock. Fluid loss into body cavities and interstitial spaces results in a relative hypovolemia. The release of inflammatory mediators as in septic shock can depress the myocardium resulting in a cardiogenic component. Therapy must be directed at the underlying systemic defect. In the case of sepsis, drainage and control of the infected focus. Because systemic inflammation resulting from sepsis and other inflammatory disease can affect oxygen delivery in so many different places, serial monitoring of many variables becomes necessary to treat the variety of problems an individual may face. The following table lists many of these important variables and optimal values for each. Interventions are also listed:

Physiologic Variable	Optimal Values	Intervention
Systemic arterial pressure	>90 mmHg	Fluids, inotropes
Central venous pressure	<3 cmH20 (5-10 cm H2O if loading)	Diuretics, venodilators
Urine output	>1 ml/lb/hr	Diuretics, dopamine
Blood Glucose	>3.9 mmol/L	Nutrition, dextrose
Packed Cell Volume	25-35%	Transfusion
Total Serum Solids	>35 g/L, < 50 g/L	Plasma, colloids
Albumin	>10 g/L	Plasma
Arterial blood gasses	PaO2 > 70 mmHg	Supplemental oxygen
	PaCO2 < 35 mmHg	Ventilatory support
	HCO3 > 14, < 24	Fluids, perfusion
	pH > 7.3, < 7. 5	Fluids, perfusion
Heart rate	>70, <150 BPM	Fluids, analgesics, anti-arrhythmics
Activated clotting time	90-120 seconds	Blood, plasma, heparin

Vascular access techniques

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Introduction

Intravenous catheterization is one of the most important skills to master in veterinary emergency and critical care. Veterinarians and veterinary technicians that can reliably gain vascular access in the sickest patients are extremely valuable members of the patient care team. Often, the patients most in need of vascular access are the hardest to catheterize. In these situations, intraosseous catheterization is an option for quick access to the vascular compartment. The landmarks are easy to identify regardless of blood pressure or degree of hydration. The purpose of this seminar is to provide a review of available catheters, indications for their use and the techniques and tricks to placing each.

Catheterization Basics

Intravenous catheters are invasive devices and their use must be managed with potential complications in mind. These foreign materials provide direct access to the bloodstream for fluids, intravenous medications and repeated blood sampling. Direct access can also allow infectious agents a means of bypassing defenses to colonize the host. These "foreign" materials can also cause a variety of inflammatory complications from mild vasculitis to serious thrombosis and vascular occlusion.

A surgical preparation of the skin over the catheter site, and sterile handling of catheter and connection tubing will minimize infectious complications. Hair should be clipped within 2 to 4 cm of the site. The area should then be cleaned and disinfected with surgical scrub for 3 minutes. While it is not necessary to wear sterile gloves when placing intravenous catheters, the person handling the catheter should have clean hands. We recommend wearing disposable vinyl or latex gloves when scrubbing the skin, then drying or changing the gloves when handling catheters and tubing.

After the catheter is in place, the site should be covered with an antibiotic ointment and clean dressing. The catheter needs to be held securely, however think about how you will remove or change connections when you are applying the bandage. Incorporating "T-port" connectors in the final bandage will take the strain of movement off the catheter. These connections are also easily replaced without having to replace the entire dressing.

Catheters should be checked every 24 to 48 hours. The bandage should be removed, the catheter site examined and the vessel palpated. The catheter should be removed and a new one placed in another site if there is any evidence of inflammation or thrombosis.

Peripheral Catheters

Short, polyvinyl chloride catheters are designed for use in small, peripheral arteries and veins. These catheters typically vary in size from 18 to 22 gauge and are 1-2 inches in length. The easiest to place, the catheters should be used for short-term procedures, in patients requiring several "lines" at one time and in those where central catheters may be contraindicated (see Central Catheters). The main advantage to peripheral catheters is the ease of placement. Peripheral vessels are easy to visualize, prep and catheterize. Peripheral vessels are easier to bandage and a better choice when it is desirable to stay away from the head as with anesthetized or seizing patients. The biggest disadvantage to peripheral catheters is the changes in fluid flow with position of the limb. Tightly flexed limbs can occlude venous drainage and impede the continuous flow of intravenous fluid. Although easy to place, these catheters may be relied up too much in the critical care setting. When vascular access is going to be needed for several days, a larger, central catheter should be considered.

An alternative to a large jugular catheter is the use of a long, Teflon catheter in the lateral saphenous vein in dogs and the medial saphenous vein in cats. These catheters provide many of the advantages of a central catheter for blood sampling, and uninterrupted flow. They also may be easier to place in patients in which you want to avoid the head or neck. Rear leg catheters are easily soiled and should be avoided in patients with urinary incontinence or diarrhea.

Central intravenous catheterization

Central venous catheterization is essential in many critically ill patients. A large-bore, teflon, jugular venous catheter can be maintained for days. Incorporated into a bandage around the animal's neck, these catheters tend to stay dryer, cleaner and can remain in longer than peripheral catheters. Central venous catheters are useful in obtaining a central venous pressure (CVP) which provides important information about fluid loading and the hearts ability to pump the fluid presented to it. Large central catheters can also be useful for repeated blood sampling. By drawing 3 cc of blood back into a syringe with 0.5 cc-heparinized saline, any volume of blood can then be sampled. The heparinized blood can then be returned to the patient and the line flushed.

This "Three-syringe" technique will minimize patient discomfort and iatrogenic blood loss. The most common central venous catheters are through-the-needle catheters. The largest of these is 16 gauge. Larger central catheters include triple lumen catheters and Swan-Ganz catheters with multiple injection ports and balloon tips to allow placement in the pulmonary artery. These larger catheters are inserted into the jugular vein over guide wires threaded through a 14-18-gauge needle. Once in place these advanced catheters facilitate monitoring of such variables as mixed venous oxygen content, central venous pressure, right atrial, right ventricular and pulmonary artery pressure as well as cardiac output and pulmonary capillary wedge pressure.

Central venous catheters should be avoided in patients with bleeding disorders, seizures or hypercoagulable states (autoimmune hemolytic anemia, hyperadrenocorticism, DIC, protein losing nephropathy).

Intraosseous catheters

Intraosseous catheters are for short-term use. They are an excellent means of delivering fluids, blood products and intravenous medications. Unlike other intravascular catheters, this procedure is easily mastered using fresh cadavers. The downside is that there are afferent pain fibers in the periosteum and endosteum making these catheters less comfortable than their intravenous counterparts. It is also difficult to impossible to sample blood from these catheters. These two issues make the placement of an intravenous catheter desirable in patients requiring ongoing intravenous therapies. The good news is that the improved vascular volume and blood pressure provides by intraosseous resuscitation make intravenous catheterization easier.

Intraosseous catheters are invasive devices and their use must be managed with potential complications in mind. These foreign materials provide direct access to the central bone cavity bloodstream for fluids, intravenous medications. Direct access can also allow infectious agents a means of bypassing defenses to colonize the host. These catheters in the proximal central spaces of long bones can dislodge bone and fat into the bloodstream. This is especially true if fluids are delivered through these catheters at high pressure.

Hypodermic needles (20-22 gauge) can be used in the smallest patients. In a bird, small mammal, puppy or kitten the needle can be worked through the soft, thin cortex. The disadvantage to this inexpensive choice is that bone material may block the lumen.

To avoid the potential for obstruction, 18-20 gauge disposable **spinal needles** are an excellent choice. With an internal stylet, these needles can enter the bone without plugging. These are stocked in our emergency room for this purpose. The needles can be inserted in all the above cases, but because they are fairly weak, not designed to cut through bone, they are not practical choices for dogs and cats with mature, hard bone and thick cortices.

If a bone marrow biopsy needle or intraosseous system is not available, a **Steinman pin** can be used to create a hole in the bone that can provide catheter access. Pin size should be checked with the outer diameter of the over-the-needle IV catheter to be used. With close attention to landmarks placement through the pre-drilled hole is easier than it sounds.

The best way to get through the cortex of mature bone is with a styleted **bone biopsy needle**. The classic choice is a metal Rosenthal needle. We tend to use 18 or 20 gauge sizes as intraosseous catheters. These needles are robust enough to cut through bone. They are reusable options built to sample bone marrow. They can be sharpened, sterilized and reused. A number of purpose made intraosseous catheters have taken this design and incorporated larger handles to make handling and insertion easier.

Vidacare's EZ-IO, Pyng's First Responder, and WaisMed's BIG (Bone Injection Gun) are elegant mechanical systems that have been developed and have quickly become commonplace with human first responders and trauma centers. Though more expensive than bone marrow needles, these devices can provide vascular access faster than any catheter system. The EZ-IO is a purpose made electric drill and low profile needles. This system is easily used in veterinary patients. The First Responder is a hand-powered system specifically designed to access the manubrium in people. The Bone Injection Gun is a spring-loaded system that has been used at other access points.

Intraosseous access points

The intraosseous catheter can be placed in medullary space of the femur (dogs and cats), the humerus (dogs and cats), the tibia (ferrets, pocket pets) or the ulna (birds). Birds use the spaces within the humerus and femur as part of the respiratory system so these bones must not be used for fluid replacement.

Small animal trauma

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Trauma is a common small animal emergency. Panicking owners may go to the first veterinary hospital they find. With a basic understanding of the systemic complications of trauma, and rationale treatment, patients that can be saved can be managed in most veterinary hospitals.

Each trauma patient should be evaluated in an orderly and systematic manner. Injuries that interfere with vital physiological functions should receive the highest priority. These are injuries that involve the respiratory system, cardiovascular system, or neurological system. Serious injuries that are not immediately life threatening include: fractures, luxations, and intra-abdominal injuries (ruptured spleen, liver or damage to the urological system). Minor injuries may merely require observation, monitoring, and serial evaluations to assure they do not slip to a more serious status.

Initial Assessment

The purpose for the initial assessment of the trauma patient is to identify life-threatening physiological injuries. Whenever a problem is identified immediate therapy is begun. This "primary survey" follows the ABC (and D's) of triage and resuscitation:

- **A**irway – Is the patient having difficulty breathing? Are there mandibular injuries that are interfering with the airway? Has a penetrating wound disrupted the larynx or trachea? Obstruction of the upper airway typically results in a slow, deep (obstructive) breathing pattern.
- **B**reathing – Is the patient dyspneic? What is the color of the mucous membranes? Is there evidence of thoracic penetration or is there a flail chest? Pulmonary contusions, pneumothorax, diaphragmatic hernia, and broken ribs can all result in a rapid, shallow (restrictive) breathing pattern.
- **C**irculation – Is there evidence of hemorrhage? Is the hemorrhage arterial or venous? How large is the swelling associated with the extremity fracture? Are the mucous membranes pale and tacky? Are the femoral pulses weak and rapid? Are the extremities cold? Is the abdomen distended?
- **D**isability – Is there evidence of neurological injury? What is the posture of the animal? Is the animal bright, alert and responsive? Does the animal respond to painful stimuli? Are the pupils dilated, constricted, of equal size, and responsive to light? Is there an extremity fracture that might threaten a peripheral nerve?

After arterial hemorrhage, respiratory function represents the highest priority in trauma. These injuries require immediate recognition and treatment. As aggressive intravenous fluid therapy can make some of these injuries worse, it is important to assume some degree of thoracic injury in all trauma patients. In one study, thoracic injuries were present in 57.7% of the dogs presented for treatment of orthopedic injures. Pulmonary contusions, pneumothorax, and fractured ribs were most commonly observed.

Pulmonary Contusions

Lung contusion is the most common acute pulmonary complication of blunt chest trauma. Hypoxemia will result from pulmonary shunt as blood flows through these non-ventilated portions of lung. It is important to note that contusions may not be evident on radiographs for several hours after the injury. The use of large volumes of rapidly administered crystalloid solutions can exacerbate the hypoxemia associated with the contusions. Conservative fluid replacement in trauma patients is recommended.

Pneumothorax

Small amounts of gas cause pleural pressure to increase slightly, but it remains sub atmospheric so that slight separation of the pleural spaces does not compromise ventilation. Tension pneumothorax is characterized by a progressive increase in pleural pressure sufficient to impair circulation. With a 20-gauge needle attached to an intravenous extension set, 3-way stopcock, and 60 ml syringe, one will aspirate air, fluid, or both. It is advisable to aspirate from both right and left sides of the thorax.

Intracranial Injuries

Normal pupillary function implies that the midbrain and third cranial nerve are intact. Midbrain damage can produce midposition and unreactive pupils. Dilated unreactive pupils that develop from miotic pupils imply brain stem lesions and a grave prognosis. Treatment of the brain trauma patient is supportive. In order to preserve brain function, and prevent ongoing neuronal damage, patients are given supplemental oxygen and maintenance intravenous fluids. Often, they receive hypertonic (7%) saline at 1-2 ml/kg to maintain microvascular blood flow. Theoretically, mannitol could exacerbate bleeding in patients with intracranial hemorrhage, and should not be used if there is evidence of focal disease. Anisocoria, and strabismus are suggestive of focal

bleeding. Mannitol is reserved for comatose patients with bilaterally symmetrical pupils or patients with deteriorating neurologic signs. Corticosteroids may actually increase cytologic damage and are no longer part of recommended treatment protocol.

Spinal Cord Injury.

Severe lesions of the T_2 - L_3 cord segments will produce the Shiff-Scherington motor response with forelimb extensor rigidity and flaccid paralysis of the rear limbs. For prognosis, the ability of the patient to perceive superficial and/or deep pain is critical.

Abdominal Trauma

Abdominal injuries are occult. Injuries caused by blunt trauma include lacerations of the liver and/or spleen, urological trauma, infarcted bowel, or reproductive organ damage during pregnancy. Penetrating injuries from gunshot, impalement injuries, and bite wounds are more obvious. Physical examination findings and diagnostic studies are required in deciding which abdomen should be surgically explored following penetrating injury. This decision is generally based upon signs of peritoneal penetration, unexplained shock, ileus, organ evisceration, free gas on radiographic examination or evidence of bacteria or plant debris following abdominocentesis or peritoneal lavage.

A focused abdominal ultrasound or A four quadrant abdominocentesis can identify fluid. Packed cell volume, total solids, cytology, bilirubin, and creatinine can be compared to blood drawn at the same time. if the packed cell volume of centesis fluid exceeds the peripheral packed cell volume, very likely there is either a splenic, hepatic or renal parenchymal laceration. In the dog or cat our approach is to treat these patients as conservatively as possible.

Emergency management of intraperitoneal rupture of the bladder, urethra, and/or ureters involves drainage of the abdominal fluid via an indwelling catheter until the patient is sufficiently stable to undergo anesthesia and surgical repair. Prior to surgery, contrast studies of the kidneys, ureter, and bladder should be performed to assess the severity of injury using an excretory urogram. If there is evidence of lower urinary tract injury, positive contrast urethrography and cystography may be necessary.

Hypothermia, acidosis and coagulopathy

The relationship of hypothermia to the development of coagulopathy is seen both in vitro and in the clinical patient. Hypothermia impairs platelet aggregation and decreases function of coagulation factors in pre-resuscitation (undiluted) blood. Clinically, human patients with a temperature lower than 34°C had elevated PT and PTT. Studies documenting this effect showed a linear relationship between the elevation in the coagulation profile times and the drop in the patient's core temperature. Acidosis, which occurs in the setting of trauma as a result of bleeding and hypotension, also contributes to the failure to clot. Experimentally, animals with a pH less than 7.20 have impaired hemostasis. It has been shown that the presence of acidosis is one of the strongest risk factors for the development of life-threatening hemorrhage in patients receiving massive transfusions. Even therapeutic options, such as factor VIIa, may be less effective in a low pH environment.

Tranexamic acid ε-aminocaproic acid

Tranexamic acid is a synthetic derivative of lysine. It is used to reduce bleeding in human elective surgery patients. It is an antifibrinolytic that competitively inhibits the activation of plasminogen to plasmin, preventing the degradation of fibrin, the major framework of blood clots. It is similar to but more potent than ε-aminocaproic acid.

One study, the CRASH-2 trial, showed that administration of tranexamic acid to adult trauma patients with, or at risk of, significant hemorrhage, within 8 h of injury, significantly reduced all-cause mortality with no apparent increase in pathologic thrombosis.

Respiratory emergencies

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Introduction

Respiratory distress in small animals can present a therapeutic dilemma. Patients can be so severely compromised that diagnostics and treatment can stress them to the point of respiratory and cardiac arrest. A diagnosis should not come at the expense of the patient. An induction chamber for cats and small dogs or a large oxygen cage can be valuable to give the patient added oxygen while the clinician can observe the patient in an attempt to localize the problem.

Dyspnea perpetuates dyspnea. Animals will become more and more distressed. Mild sedation with very low doses of sedative agents can be life saving by allowing the anxious dyspneic animal to breathe more efficiently.

Restraint for catheterization, radiographs and physical examination may have to wait until the patient is relaxed and breathing easier. In the case of pleural effusion or pneumothorax, a thoracocentesis can provide a diagnosis at the same time it is providing the life-saving treatment.

Try to determine the nature of the problem first with observation. A rapid shallow respiratory pattern suggests restrictive disease while a slow deep inspiratory pattern is seen with airway obstruction. With the restrictive pattern, auscultation can help differentiate pleural space

disease (pneumothorax, hydrothorax) from parenchymal diseases (pneumonia, pulmonary edema). Signalment and history can help determine a cause of upper airway obstruction (playing with small toys, brachycephalic airway diseases, and laryngeal paralysis).

Once the patient has been stabilized and calmed, treated for shock and hyperthermia definitive diagnostics can be performed. Some of the important emergency diagnostics will be reviewed.

Imaging

Animals presenting with upper and lower respiratory signs should have a thoracic radiograph. Bronchial patterns develop as the peribronchiolar tissues become inflamed. Interstitial patterns develop with thickening of the fibrous structures of the lung. Alveolar patterns characterized by "Air bronchograms" are caused by fluid accumulation in the alveoli. Thoracic and cervical radiographs can be used to diagnose collapsing trachea, tracheal or laryngeal foreign bodies, and tracheal or laryngeal masses. Airway dynamics can be assessed by taking inspiratory and expiratory views of the trachea or through the use of fluoroscopy.

Laryngeal Paralysis

Laryngeal paralysis can be either congenital or acquired and is a common cause of emergency visits in large breed dogs. The paralysis may be either unilateral or bilateral.

Animals with laryngeal paralysis will present with varying degrees of exercise intolerance, stridor, voice change, inspiratory effort, cyanosis and hyperthermia. They will have a pronounced inspiratory stridor with a slow, deep (obstructive) breathing pattern. Often an obvious inspiratory wheeze will be heard loudest over the larynx. Diagnosis is by direct examination under a light plane of anesthesia. Large breed dogs with laryngeal paralysis may present in extreme distress. Dyspnea from upper airway obstruction can cause the animals to become anxious and more dyspneic. Patients with prolonged hyperthermia should be hospitalized and observed for complications. The kidneys, GI tract, liver and nervous tissue can all be damaged by excessive heat. Disseminated intravascular coagulation is another common complication. Once the patient is stable and signs of heat stroke have resolved definitive treatment for laryngeal paralysis can proceed.

Pulmonary Edema

Noncardiogenic pulmonary edema occurs occasionally in dogs and cats secondary to electric cord bites, sepsis, following near drowning or choking, snake bites, uremia, smoke inhalation, upper airway obstruction, and the acute or adult respiratory distress syndrome (ARDS).

The pathogenesis of edema is thought to be increased pulmonary capillary hydrostatic pressure and increased alveolar-capillary permeability. Increased pulmonary capillary hydrostatic pressure is likely due to a centrally mediated burst of sympathetic activity, which causes constriction of resistance and capacitance vessels leading to a shift of blood from the splanchnic viscera into the circulation.

Pulmonary edema occasionally develops secondary to upper airway obstruction in dogs. Laryngeal and pharyngeal diseases are most common. Inspiratory and expiratory stridor, dyspnea, crackles, and cyanosis are common physical examination abnormalities. Mixed interstitial and alveolar lung infiltrates are detected in the

perihilar and dorsocaudal lung fields. Treatment can include administration of oxygen, diuretics and glucocorticoids, as well as tracheostomy if needed. Edema is primarily related to decreased intrathoracic pressure resulting in decreased interstitial hydrostatic pressure and hypoxia resulting in increased alveolar capillary permeability.

Pneumonia

Pneumonia is inflammation of the lung parenchyma; bronchopneumonia is pneumonia that has begun in the terminal bronchioles. Bacterial pneumonia in dogs is rarely a primary disease. Occasionally, *Bordetella bronchiseptica* or *Mycoplasma* spp. can induce pneumonia in dogs and cats due to their adverse effects on mucociliary function. Most cases of bacterial bronchopneumonia are secondary to immunosuppressive diseases or previous inflammatory insults including viral infection, aspiration, and irritant inhalation. Owners should be carefully questioned concerning potential exposure to other animals and clinical signs associated with immunosuppressive diseases or aspiration.

One of the most important treatments of bacterial pneumonia is hydration. The mucociliary apparatus function best in a well-hydrated animal and is essential for the clearance of infection. Affected animals should receive parenteral fluid therapy until able to maintain hydration orally. Airway hydration can be accentuated by nebulization or by placing the animal in a closed bathroom while running hot water through the shower.

Common bacterial isolates include *Bordetella bronchiseptica*, *Pasteurella multocida*, *Klebsiella* spp., *Streptococcus* spp., and *Escherichia coli*.

Feline Bronchial disease

There is no clear terminology for the bronchial obstructive diseases in the cat. Bronchitis is inflammation of the airways. Asthma generally implies a reversible bronchoconstriction related to hypertrophy of smooth muscle in airways, hypertrophy of mucous glands, and infiltrates of eosinophils. Asthma in cats is primarily due to Type I hypersensitivity reactions; the etiology is generally undetermined. Cats with bronchitis not due to asthma generally have infiltrates of neutrophils or macrophages as well as hypertrophy of mucous glands, hyperplasia of goblet cells, excessive mucous, and ultimately fibrosis secondary to chronic inflammation. Etiologies include bacterial infection, mycoplasmosis, viral infection and parasitic infections.

Cats presenting dyspneic as an emergency are often to distressed to handle. Physical examination, intravenous catheterization and diagnostics could prove fatal. The best approach is to place them in an oxygen cage while obtaining a history and observing their respiratory efforts.

Pleural Space Disease

Diseases of the pleural space cause a decreased tidal volume and a restrictive breathing pattern. Characterized by the rapid, shallow respirations and dull lung sounds, fluid and air in the pleural space can be diagnosed and treated by rapid thoracocentesis. Once removed, fluid can be examined to determine the likely cause.

Thoracocentesis is performed using a 20 or 21g needle attached to a short piece of extension tubing. Alternatively, a larger (19g) butterfly catheter may be used. The needle and tubing are attached to a 20-60 cc syringe through a 3-way stopcock. A 2x2 cm area over the lateral chest wall between the 6th and 10th intercostal spaces is surgically prepared. With the animal comfortable in either sternal or lateral recumbency, the needle is advanced into the thoracic cavity. It is helpful to mark the location of the needle's bevel before advancing into the chest. Once either air or fluid start flowing, the needle can be laid flat against the chest wall with the bevel facing in toward the lungs. This will allow complete re-inflation without lacerating the lung on the needle. A negative thoracocentesis may be due to fibrous adhesions and small pockets of fluid. Diaphragmatic hernias may also cause significant pleural restriction but yield a negative tap. These patients should be given supplemental oxygen while radiographs are taken.

Large volumes of air or fluid, continuous production of air, or suppurative inflammation are reasons to place a chest drain. A variety of drains are available which come over a trocar for ease of placement. Tubes should have a radiopaque line to evaluate placement. Thoracostomy tubes are placed into the chest with the tip lying in the cranioventral thorax. The tubes should pass into the chest through the 7th or 8th intercostal space after traveling several rib spaces under the skin to provide so protection against iatrogenic pneumothorax. Once in place, the chest tube should be secured to the skin with a self-tightening knot and all connections secured with circlage wire.

Necrotizing soft tissue infections and newer epidemics: MRSA, MRSP

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Pet owners hearing the term "MRSA" or "antibiotic-resistant infection" in the context of their own pet's illness will need our help understanding the implications. Veterinarians need to be able to manage these cases responsibly and help educate clients with these animals to alleviate fears and minimize actual risk. In our hospital one of the biggest challenges is getting information to all the staff that may come in contact with these cases. Often a lack of knowledge leads to fear and misinformation when these cases come through our hospital. It is the goal of this talk to focus on our approach to the multi-drug resistant *Staphylococcus* spp. and discuss concerns about *Clostridium difficile*.

Drug-resistant *Staphylococcus* spp.

Dogs primarily carry *Staphylococcus pseudintermedius* while humans carry *Staphylococcus aureus*. Infections from both these species have been identified in humans and animals. Antibiotic overuse across species has lead to *Staphylococcus* spp. acquiring genes that confer resistance to beta-lactam antibiotics including penicillins, amoxicillin, and methicillin. Methicillin resistant *Staphylococcus* spp. is indicated by the presence of the *mecA* gene, which encodes a mutation in PBP2a, that reduces the affinity for the beta-lactam ring, rendering the organism resistant to all beta-lactams. Methicillin resistance is assumed in many veterinary reference laboratories (including our own) by bacterial resistance to oxacillin. Cefoxitin may be another indicator of multidrug resistance in *S. aureus* but not *S. pseudintermedius*. Only 10% of *Staphylococcus* infections are now sensitive to penicillin. Overuse of extended spectrum B-lactams and cephalosporins have led to more resistant bacteria. Many of these may acquire resistance to alternative antibiotics such as clindamycin, chloramphenicol, trimethoprim sulfa, doxycycline, and rifampin.

Clinically we usually see recurrent, or non-healing skin wounds turn up with drug resistant infections. These skin infections may start as a small area that looks like a bug bite or skin irritation. In fact, it is common for this classic, progressive *Staphylococcus* lesion to be misdiagnosed as the bite of the brown recluse spider (*Loxosceles reclusa)*. Redness, heat, swelling, and pain may be the first symptoms. Pustules, crusting and deep pyoderma may be the first signs with draining tracts developing later. Non-healing surgical sites or drainage from a surgical site is also common. Resistant *Staphylococcus* infections in dogs result in chronic skin infections or non-healing surgical wounds. These may lead to systemic complications like bacteremia, pneumonia, septic arthritis, osteomyelitis, and valvular endocarditis.

When the *Staphylococcus* bacterium enters the skin it can cause illness through direct infection and from bacterial toxins. *Staphylococcus* spp. can live within cells replicating out of the reach of the immune system and antibiotics that can't penetrate. *Staphylococcus* spp. are frequently implicating at colonizing indwelling devices (intravenous, urethral, and arterial catheters, chest tubes, drains, orthopedic devices). The infectious plaques they form can make it difficult for antibiotics to penetrate. In canine patients, *Staphylococcus* infections are commonly seen with atopic dermatitis though we often see it in trauma cases, in oncological and post-surgical patients.

Methicillin-resistant *Staphylococcus aureus*

Methicillin-resistant *Staphylococcus aureus* **(**MRSA) is an important human pathogen causing many clinical infections every year. The incidence of MRSA doubled in human medicine in the 7-year period between 1999 and 2006. The impact of MRSA in veterinary medicine is increasingly problematic, not only because of its impact on the patient, but the public health considerations. Historically, MRSA has been a leading cause of hospital-associated infection, but since the 1990s, community-associated MRSA has emerged, and in some regions has become a significant pathogen. MRSA was considered a human pathogen, with early animal infections thought to be acquired from humans. However, recent evidence shows that animals may play an important role in human infection. MRSA can easily be spread from one person to another. Healthy human patients sharing clothing, razors, towel, and sports equipment or in individuals with skin irritation from eczema, shaving, bite wounds, or other conditions making the skin more fragile are at greater risk for contracting an MRSA infection.

Risk factors for the presence of MRSA in working and companion dogs include contact with human hospitals and children. Similar organisms have been isolated in family members and pets in the same household, but this is likely to reflect transmission from humans to the pet. It is likely that colonization is transient in animals. However, healthy pets have been demonstrated to be potential reservoirs for transmission of MRSA to both healthy handlers and immunocompromised individuals. One study examined MRSA colonization in people and companion animals in the same household with an infected person or infected pet, all isolates in dogs and cats in the same household were indistinguishable. Colonization of humans by MRSA was found to be 17.9% and that of dogs in the household was 8.3%. The conclusion: Canine companions can serve as a reservoir of antibiotic resistant bacteria.

Methicillin-resistant *Staphylococcus pseudintermedius*

With studies suggesting that up to 50% of dogs are colonized with *S. pseudintermedius, 0.58*-9 % are reported to be colonized with Methicillin-resistant *Staphylococcus pseudintermedius* (MRSP). Primarily a commensal bacteria of dogs, infections are most likely to occur in previously colonized dogs and infected patients probably pose a higher transmission risk than colonized patients. Clinicians should suspect MRSP in dogs if there is a wound or infection that does not heal as expected, especially with prior antimicrobial treatment. Case should also be suspect if there is a history of MRSP colonization or infection in other animals with which the patient has contact.

These patients should be treated as highly contagious. High-risk patients should not be allowed to remain in common use areas within a veterinary facility. They should be moved as soon as possible to isolated care areas where contact and potential contamination can be more easily controlled. Hospitalized MRSP patients should be segregated to manage contact transmission. We routinely use disposable barrier nursing gowns, and gloves with these case. As with any suspected infectious agent, hand washing or use of hand sanitizers should be practiced by anyone coming in contact with affected patients. The waste (e.g. bandages) from these patients is double bagged and disposed of normally. Because there is negligible zoonotic hazard for personnel related to MRSP exposures we don't employ the same alerts to cleaning staff and hospital personal are educated on the differences between MRSP and MRSA.

Methicillin resistance in *S. pseudintermedius* identifies a strain carrying genes that make it resistant to all beta-lactam antimicrobial drugs, including potentiated penicillins, cephalosporins, and imipenim. Many strains of MRSP are also resistant to other drugs commonly used to treat infections, such as tetracyclines, gentamicin, and enrofloxacin. If systemic antibiotics are indicated we will usually consult with our diagnostic laboratory regarding the specific antibiotic profile for each patient. MRSP should be considered resistant to ALL beta-lactam drugs regardless of in vitro susceptibility results indicating otherwise. Topical therapies should be employed whenever possible, including topical antiseptic therapies (e.g. chlorhexidine).

Culture of suspected cases and appropriate diagnostic testing is required for inpatients suspected of being infected with MRSP. This usually means culturing non-healing wounds and surgical site infections. Not only do we take appropriate specimens from infection sites but also we may screen the patient colonization by swabbing the nares and anus. It is important to communicate with your laboratory and request enriched MRSP cultures on laboratory submissions. We consider any *Staphylococcus* spp. isolate that is resistant to oxacillin a methicillin resistant organism. Note: Unlike MRSA, resistance to cefoxitin and ceftiofur is not a sensitive marker for methicillin resistance in *S. pseudintermedius*. In other words, isolates may be MRSP and show in vitro susceptibility to cefoxitin and ceftiofur. Methicillin-resistance does not necessarily indicate that the strain is more infectious or pathogenic than other strains of *S. pseudintermedius*.

Avoiding Transmission of Resistant *Staphylococcus*

Wound care becomes the most problematic aspect of these cases. There may be a desire to refer a case when antibiotic resistance is identified. Most of these cases require aggressive wound care with regular bandage changes, wound debridement and care of surrounding, healthy tissues. We practice barrier precautions and isolate these patients until they have two negative cultures a least a week apart. Animals that don't require hospitalization (the majority) are seen as regular outpatients. They are admitted into our isolation unit for their short stay. All procedures are performed away from the general hospital population. Soiled bandages are bagged and disposed of immediately. Wounds are debrided and packed with easily removed absorbent, antimicrobial dressings as indicated by the wound itself.

Proper hygiene within the clinic is essential. Hand washing should between handling all animals with soap and warm water or using an alcohol-based hand sanitizer. We wear disposable gloves when doing wound care, changing bandages, touching bedding, or handling indwelling devices. Personal barrier precautions should also be used when cleaning kennels of any animal known to carry MRSA, or MRSP. Perform hand hygiene after glove removal. Soiled bedding should be washed in hot water and laundry detergent and dried in a hot dryer. Disinfectants or a 1:100 dilution of bleach (2 Tablespoons of bleach to 1 quart of water) are used to clean surfaces that might be contaminated. People in contact with these patients should keep any cuts or scrapes covered with clean dry bandages. Avoid contact between infected animals and staff or owners that are immune suppressed or have had a recent surgery.

Endocrinology

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Federico Fracassi graduated in Veterinary Medicine (DVM) *"cum laude"* State University of Bologna. At the same Faculty he followed an internship. In 2005 he defended his PhD thesis. After the PhD he become a permanent staff member at the Department of Veterinary Medical Sciences - University of Bologna where at present is Associate Professor of Internal Medicine and head of the unit of Internal Medicine. He completed an Alternative Residency in Internal Medicine at the Clinic for Small Animal Internal Medicine, University of Zurich, Switzerland and gained his status of European Specialist in Internal Medicine in 2012 (Diplomate ECVIM-CA-internal medicine). Duties include patient care, education (to students, post graduate training and training of interns and residents) and research in internal medicine of companion animals, with special emphasis on endocrinology. He is the vice president of the European Society of Veterinary Endocrinology (ESVE), president of the Italian Society of Veterinary Internal Medicine (SIMIV), board member of the executive committee of the ECVIM-CA congress and editor of "Veterinaria", a peer review journal. His publication list consists of journal articles, research abstracts, case reports and book chapters. His main research focus is the field of small animal endocrinology.

Treatment and therapeutic monitoring of diabetes mellitus

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The treatment objective of diabetes mellitus is to resolve the clinical signs and to prevent the onset of diabetic ketoacidosis (DKA) and the development of hypoglycemia, thus ensuring good quality of life. This usually applies when blood glucose velues values are maintained between 90-250 mg/dL or 5-14 mmol/L. If given the correct insulin therapy, in combination with a suitable diet, the diabetic patient has a life prognosis comparable to a healthy subject. Effective treatment is only achieved when there is good communication and mutual trust between the owner and the veterinarian; good client education is essential, and the pet owner should be kept informed of the objectives and therapeutic progress on an ongoing basis. In particular, clinical examination and monitoring should be at set time points (see Table 1).

Table 1. Therapeutic and monitoring protocol for diabetic dogs and cats.

Initial diagnosis and treatment

- Diagnosis: history, physical examination, hyperglycemia, glycosuria and increased fructosamine levels
- Diagnostic protocol - Complete blood count, blood chemistry, urinalysis with culture, abdominal ultrasound and cPLI or fPLI (canine/feline pancreatic lipase immunoreactivity), if indicated. TT4 in cats if indicated. Discontinuation of any diabetogenic drugs
- Therapy: administration of intermediate (dogs)- or long-acting insulin (cats) q12h
- Treatment of concomitant diseases
- Diet
  - Quantity: standardize the amount of food fed every 12 hours at the same time as, or just before, insulin administration. In cats frequency of feeding is likewise not critical, continuing the normal frequency for the individual cat is generally advised (assuming a minimum of two meals daily)
  - Type: high-fibber, low-carboidrates commercial diets for diabetic dogs, and low-carboidrates, high proteins commercial diets for diabetic cats
  - Any other concomitant disease (chronic kidney disease, liver disease, allergies/intolerances, pancreatitis) from a dietary point of view should be considered priority with respect to DM
  - Emaciated dogs or those with BCS < 4/9 should be fed a balanced maintenance diet until an optimal BCS is achieved
  - Obese or overweight dogs and cats: a program should be drawn up to reduce body weight by 1-2% per week
- Owner education: verbal instructions supported by demonstrating insulin administration and information leaflets
- All entire females dogs should undergo ovariohysterectomy as soon as possible

Clinical re-evaluation 1 week after diagnosis

- Physical exam
- Perform a blood glucose curve (BGC), with food and insulin administered at the clinic after the first glucose measurement. Alternatively (almost always in cats), patients can be taken to the clinic immediately after the owner has administered the meal and insulin at home
- Determination of serum fructosamine concentration
- Therapy: insulin dosage adjustment by 10-15% if necessary

Clinical re-evaluation 2-3 weeks after diagnosis

• Physical exam • Perform a BGC • Determination of serum fructosamine concentration • Therapy: insulin dosage adjustment by 10-15% as necessary • Introduction of home monitoring for glycemic control, with written instructions for the owner • The owner should produce a BGC every 2 weeks and measure fasting glycemia approximately twice a week

Clinical re-evaluation 6-8, 10-12 weeks and every 4 months after diagnosis

• Physical exam • BGC and serum fructosamine, with dosage adjustment as necessary • Evaluate the owner's insulin administration technique

Insulin

Various types of insulin can be used to treat canine and feline DM, and insulin analogues (*e.g.,* glargine, detemir) may also be useful in managing the condition, as indicated in *Table 2*. It is important to ensure that the correct dose of insulin is given. Owners should be aware that the units/mL can vary with different types of insulin (see *Table 2)* and must use the correct syringes. Some owners may find that an insulin "pen" helps/ improve administration of the insulin.

Table 2. Types of insulin commonly used for the long-term therapeutic management of canine and feline DM.

Insulin type	Origin	Concentration (IU/mL)	Frequency of administration	DOG:	CAT Starting dose (IU/cat/injection)
Lente	Swine	40	BID	0.25	1-1.5
NPH	RH	100	BID	0.25	Not recommended for cats (short duration of action)
PZI	RH	100	BID	0.25-0.5	1-1.5
Glargine	RH	100	BID (SID)	0.3	1-1.5
Detemir	RH	100	BID (SID)	0.1	1-1.5

NPH: neutral protamine Hagedorn. PZI: protamine zinc. RH: recombinant human

Diet

Diet is an extremely important aspect in the therapeutic protocol. Dogs with DM may be underweight or overweight; in both cases, the objective is to reach and maintain an ideal body weight. In an obese or overweight dog, a reduced-calorie diet rich in insoluble fiber and low in fat should be provided. Reduction of body weight is essential; animals that present with a BCS > 5/9 at the time of diagnosis can easily gain weight after treatment starts due to the anabolic effect of exogenous insulin. Underweight or emaciated patients should be fed a balanced maintenance diet. As a rule, dogs with DM should be fed every 12 hours with a standardized amount of food, with insulin administered at the same time. In the cat: different studies have shown that a protein-rich diet extremely low in carbohydrates improves glycaemic control and allows the dose of insulin to be reduced significantly.

Physical exercise

Controlled physical exercise is recommended for all dogs with DM; this enables the glycemia concentrations to be reduced and better absorption and use of insulin by the muscles. Physical exercise should be consistent; this is particularly relevant for working dogs, as sudden exertion can predispose to hypoglycemia and should be avoided. If a dog has to perform substantial, non-routine physical exercise, the insulin dose on that day should be reduced by about 50%.

Glycemic monitoring

Blood glucose curves

Serial blood glucose curves (BGC) are essential for both short- and long-term monitoring, and enable rational adjustment of the insulin dose as necessary. To measure blood glucose, portable glucometers (PBGM) are commonly employed, requiring a small blood sample usually obtained from the dog's pinna. There are many PBGMs on the market, but most are designed for human use and can be inaccurate when used in dogs. PBGMs designed specifically for veterinary patients (e.g. AlphaTRAK) are preferred, as they require an extremely small sample volume (0.3 µL) and deliver more precise results.

Blood glucose curves can be produced in the clinic or at home by the owner. The test requires glucose levels to be monitored every 2 hours, starting immediately before a meal and insulin administration and continuing for about 8-10 hours throughout the day. The trend of the BGC is used to verify insulin effectiveness, identify the glucose nadir (ideally between 90-150 mg/dL or 5-8 mmol/L), and determine the time necessary to reach peak glycemia, as well as the degree of fluctuations in glucose levels. The BGC is considered optimal when most of the glycemic values fall between 90-250 mg/dL (5-14 mmol/L). If poor glycemic control is noted during a clinical exam, it is essential that the cause is identified and corrected as soon as possible. Various possible scenarios can lead to poor glycemic control; the more frequent reasons include owner errors, such as using a 100U/mL syringe with 40U/mL insulin, or vice versa.

Home monitoring

Blood glucose curves are usually produced in the clinic by the veterinarian; however, the cost of this procedure for the owner, in terms of both time and money, should not be undervalued. Furthermore, blood glucose levels can be affected by the stress animals (in particular cats) feel in an unfamiliar environment. In order to overcome these problems, some authors have suggested that the monitoring could be carried out at home by the animal's owner. This is possible by pricking the capillaries of the internal surface of the pinna or labial mucosa and testing the collected blood in a portable device for measuring glycaemia. There is a good correlation between the levels of glucose measured in venous blood and capillary blood. The procedure is quick and easy for the veterinarian, but also for the animal's owner.

Fructosamine

This parameter has the advantage of not being affected by stress-induced hyperglycaemia or lack of food intake, conditions which often alter the outcome of the blood glucose curves measured in the clinic. However, other factors can influence its blood concentration and sometimes there are discrepancies between serum fructosamine levels on the one hand and the clinical signs and blood glucose curve on the other.

Continuous glucose monitoring

More recently, continuous glucose-monitoring systems (CGMS) have been introduced which enable glycemic trends to be evaluated without the need for serial blood samples. These systems actually monitor interstitial glucose concentrations, which reflect serum glucose concentrations. Some CGMS are capable of recording a patient's glycemic trends by means of wireless technology, whereby a flexible sensor inserted subcutaneously transmits data to a monitor up to 3 meters distant. However, this type of instrument needs to be calibrated 2-3 times daily. Newer technology enables real time monitoring of glucose values; the name of the instrument is FreeStyle Libre; a small sensor applied to the neck area delivers instant read-outs to a monitor when scanned, with the added advantage that a sensor lasts for 14 days and does not require calibration.

Main causes of poor therapeutic control in dogs and cats with DM.

- Owner error when administering insulin - Inappropriate administration method - Inappropriate syringe (*e.g.,* using 100 U/mL syringes with 40 U/mL insulin)
- Incorrect insulin storage or preparation - frozen insulin - inadequate mixing of insulin (*e.g.,* with zinc suspension insulin and NPH)
- Insulin underdose
- Somogyi Effect (hypoglycemia and resulting hyperglycemia secondary to compensatory mechanisms due to the action of hormones that increase blood sugar levels)
- Short-acting insulin
- Inadequate insulin absorption (*e.g.,* chronic skin inflammation, allergic reactions)
- Concomitant diseases leading to insulin resistance (*e.g.,* inflammatory, infective, endocrine, neoplastic or immune-mediated processes, and the use of diabetogenic drugs).

Feline hyperthyroidism

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Spontaneous hyperthyroidism (thyrotoxicosis) is a clinical condition resulting from an excessive production and secretion of thyroxine (T4) and triiodothyronine (T3) by the thyroid gland. In 98% of cases of feline hyperthyroidism, the hormone excess is caused by a functional thyroid adenomatous hyperplasia (or, less frequently, by an adenoma) involving one, or more often (70-75%) both lobes of the gland. At histology, the thyroid lobes involved have one or more foci of hyperplastic tissue which, by merging, may form palpable nodules ranging between 1 mm and 3 cm in size. In the approximately 2% of remaining cases, the disease is caused by a carcinoma of the thyroid gland.Spontaneous hyperthyroidism (thyrotoxicosis) is a clinical condition resulting from an excessive production and secretion of thyroxine (T_4) and triiodothyronine (T_3) by the thyroid gland. The aetiopathogenetic mechanisms capable of causing the disease in cats are still unknown, and thus treatment is aimed at directly controlling excessive hormone production by the thyroid neoplasia. Hyperthyroidism is the most common endocrine disorder found in elderly or middle-aged cats. The average age of subjects at the time of diagnosis is 12-13 years (range 4-22 years), of which fewer than 5% are under age 8. Typical owner complaints include weight loss, polyphagia, polyuria, polydipsia, intermittent vomiting and/or diarrhoea and altered behaviour, including increased irritability/ aggression. Apathetic hyperthyroid cats may be lethargic with a decreased appetite. Physical examination will usually identify poor body condition and muscle condition score, tachycardia with or without a gallop rhythm and/or cardiac murmur and tachypnoea. A palpable goitre is present in 80–90% of cases. A plantigrade stance or cervical ventroflexion may be present due to muscle weakness. The coat may be matted and unkempt from under-grooming or have areas of alopecia from over-grooming Systolic blood pressure may be elevated although this is not as common as initially suggested. A retinal examination should be performed, especially if blood pressure cannot be measured, as evidence of retinal haemorrhage supports the presence of the systemic hypertension rather than that caused by the 'white-coat' effect. History and physical examination may be strongly suggestive of hyperthyroidism; however, as concurrent disease is common in geriatric cats a full evaluation will lead to informed management decisions. Routine haematology may be normal, but erythrocytosis and/or lymphopenia may be present. Routine biochemistry often shows mild / moderate elevations in liver enzymes. Azotaemia, elevated inorganic phosphate and hypokalaemia may be variably seen. Urinalysis should be performed to evaluate for concurrent disease such as chronic kidney disease, urinary tract infection or diabetes mellitus. Definitive diagnosis is usually obtained by demonstration of elevated total T4 (thyroxine). Concurrent illness may lower thyroxine into the (upper half of the) normal range in the hyperthyroid cat. In this circumstance free T4 may be measured by equilibrium dialysis as it is less susceptible to influence by concurrent illness; it is best assessed together with thyroxine. A high free T4 and low or low normal thyroxine is more suggestive of non-thyroidal illness than hyperthyroidism. Other diagnostic tests that may be performed include measurement of thyroid-stimulating hormone (TSH) or a T3 suppression test, although these are rarely required. Scintigraphy involves subcutaneous injection of radioactive sodium pertechnetate which is taken up by the thyroid glands. Comparison of uptake between the salivary glands and the thyroid glands enables demonstration of enlarged and ectopic thyroid tissue. This is only available at certain referral centres and does not allow differentiation between carcinomas and adenomatous hyperplasia.

Three therapeutic options are available, each of which presenting advantages and disadvantages that should be carefully evaluated for each individual patient.

- Destruction of the neoplastic tissue with radioactive iodine
- Surgical thyroidectomy
- Inhibition of hormone secretion with antithyroid drugs
- Limited-iodine food

Treatment with radioactive iodine: In view of its high therapeutic efficacy and the relative absence of complications, radioactive iodine therapy is considered the treatment of choice for feline hyperthyroidism. However, due to the radioactive potential of the agent used, this treatment can only be carried out in authorised facilities, which explains why its use is still limited. The β radiations of I^{131} selectively destroy hyperfunctioning thyroid cells, sparing normal thyroid tissue. The radioactive agent may be administered orally, intravenously or subcutaneously and a single administration is usually sufficient to attain the therapeutic effect (a second treatment is needed in only 5% of cases). The treatment requires hospitalisation and isolation of the cat for at least one week after the administration of I^{131} because of the high degree of elimination of radiation through faeces and urine. Radioactive therapy is especially advantageous in cats with bilateral thyroid lobe involvement, in the presence of pathological ectopic thyroid tissue, or in the rare cases of carcinoma of the thyroid gland. The possible complications associated with the use of this treatment are related to the possible onset of a nephropathy, not caused by the treatment itself but by the return to a euthyroid state in subjects in whom the hyperthyroidism present before treatment was masking the presence of the disease. Before implementing this treatment it is therefore advisable to first check, with the aid of medical antithyroid treatment, whether the concentration of T_4 can be reduced safely without causing renal failure.

Surgical treatment: Uni- or bilateral thyroidectomy is a quick, effective and relatively easy procedure, and is often the preferred treatment approach for the disease in clinical practice. In 70% of cases adenomatous hyperplasia involves both thyroid lobes, even though bilateral involvement is not always recognized in view of the small size of the neoformations. In these cases, if a unilateral thyroidectomy is performed, a recurrence of the disease can be observed months later. Once the need for surgery has been established and the correct thyroid lobes have been identified (ideally a nuclear medicine scan should be performed before surgery), the next step is to stabilize the cat for surgery. Most hyperthyroid cats are elderly and have heart disease resulting from their thyroid condition. To set the patient on the road to recovery, the thyroid level is brought into the normal range with two to four weeks of oral medication (usually methimazole). Alternatively, certain heart medications (propranolol or other beta-blockers) are often used to compensate for the heart disease associated with hyperthyroidism, especially in cats with resting heart rates greater than 220 beats per minute. After thyroid levels have normalized, it is important to watch for an exacerbation of renal disease that may be unmasked by the treatment of hyperthyroidism. Concurrent kidney problems complicates anesthesia and may even preclude the surgery. Thyroidectomy can be performed using extra- or intracapsular techniques; however, the latter technique is preferable, as it reduces the risk of impairment (damage or removal) of the adjacent parathyroid gland. If a bilateral thyroidectomy is performed the main postoperative complication is the onset of iatrogenic hypoparathyroidism, which is generally temporary; for this reason, after this type of operation serum calcium concentrations should be monitored for at least one week. Hypocalcaemia should only be treated if clinical signs are present or if serum calcium concentrations are below 6.5 mg/dl, even in the absence of clinical symptoms. Sings of hypocalcaemia include anorexia, lethardy, anxiety, irritability, cramps or muscle pain, muscle tremor, especially in the face and ears, tetany and seizures. In all cases of bilateral thyroid surgery the plasma Calcium levels should be monitored by measuring preoperatively and at about 20 h after surgery. In cases with severe hypocalcemia and clinical symptoms (tremors, tetany, convulsions) IV administration of 0.5 mmol Ca^{2+}/kg as calciumgluconate is given under close ECG monitoring. The same dose that was needed to control tetany and tremors is diluted with at least an equal volume of saline 0.9% and administered subcutaneously, 2–4 times a day. In cases without clinical symptoms of hypocalcemia the following protocol is used: if plasma calcium is < 1.0 mmol/l or more than 10% below the preoperative value, calcium(boro) gluconate (1–2 ml/kg) is administered subcutaneously, 2–4 times a day, diluted with at least the same volume of 0.9% saline. As soon as the cat is eating oral supplementation is started. Calcium carbonate powder (15–20 mg/kg) is added to each meal and dihydrotachysterol is provided at a starting dose of 0.05 mg per cat once a day during the first 3 days, and then decreased to 0.025 mg once a day.Supplementation may be needed for only a few days or for life depending on the damage to the parathyroids. Some surgeons prefer to perform thyroidectomy in stages, removing one thyroid lobe and transplanting the associated parathyroid gland into a local muscle belly to preserve its blood supply. The same procedure is performed on the other thyroid lobe two to three weeks later. The staged procedure reduces the risk of hypocalcemia but does involve two anesthetic procedures on the senior feline patient who is already somewhat debilitated by the hyperthyroid situation. Hypocalcemia is not a concern for cats requiring removal of only one thyroid lobe. Usually levothyroxine supplementation is not needed if only one lobe is removed. After bilateral thyroidectomy oral substitution with L-thyroxine is provided (50 µg per cat twice daily, starting on the fourth day after surgery). Plasma T4 levels are measured after one month and then every six months. The dosage of L-thyroxine is adjusted if necessary to maintain plasma T_4 within the normal range.

Medical treatment: Medical treatment is a practical option that does not require the use of special equipment and, at least initially, is not expensive. With the exception of the rare cases of thyroid gland carcinomas, medical treatment has few contraindications, mostly connected above all with the onset of side effects. The more commonly used drugs in both human and veterinary medicine for the long-term control of hyperthyroidism are the *thiouracil derivatives:* **carbimazole and methimazole (thiamazole).** These drugs inhibit the action of the thyroid peroxidase enzyme and consequently block the synthesis of thyroid hormones. When hormone production is inhibited a rapid return to a condition of euthyroidism is generally observed, in around 2-4 weeks after the start of treatment. Compared to the treatment with radioactive iodine and to thyroidectomy, these drugs allow a reversible control of the disease: 24-72 hours after discontinuation of the drug, the cat returns to a state of hyperthyroidism. Although this is considered a negative aspect, in some situations this result may be considered advantageous: it can be used to test whether with the return of euthyroidism the cat develops an overt renal failure or before thyroidectomy to stabilise the patient. Carbimazole after oral administration is metabolised and converted into methimazole, the molecule with antithyroid properties. A 5 mg dose of carbimazole is equivalent to approx. 3 mg of methimazole. The initial dosage of carbimazole is 2.5 mg/cat twice daily and the initial starting dose of carbimazole is 10-15 mg/cat once a day. Methimazole, like carbimazole, is a drug that, if well tolerated by the subject, has an efficacy greater than 90% in the treatment of hyperthyroidism. The most common side effects of methimazole are anorexia, vomiting and lethargy. These symptoms generally arise within the first 4 weeks from the beginning of treatment and they can be resolved by reducing the dosage or by using the transdermal formulation of methimazole. In some cases more severe adverse reactions to the drug have been reported, including: blood dyscrasia (thrombocytopenia, leukopenia), facial excoriations from self-trauma and liver disease. In these cases the administration of the drug should be discontinued. **Transdermal methimazole:** in the cat, methimazole may be more easily administered transdermally, using a pharmaceutical preparation expressly formulated with PLO (pluronic lecithin organogel). The owner, wearing gloves, should apply the ointment 2 times a day, to the inner pinna. The starting dose is 2-2.5 mg/cat every 12 hours. The bioavailability of the drug is inferior compared to that of the formulation for oral administration, and hence the efficacy is lower. Nevertheless, this formulation is especially advantageous for those cats in whom tablets are

difficult to administer, and especially if tablets are associated to the appearance of gastrointestinal effects. **Therapeutic monitoring**: The first control after the beginning of treatment is at 2-4 weeks, to test the concentration of T_4, to exclude the onset of possible drug adverse reactions and to monitor renal function. It is important to evaluate renal function and T_4 concentration simultaneously during treatment, to make sure that the latter is maintained even with GFR values associated with a state of euthyroidism. Hormonal monitoring may be carried out independently from the moment of drug administration but is inportant that in the day ofr monitoring the medication has been administrated; concentrations of T_4 within the lower half of the reference range are generally associated with a good control of the disease. Is it common to see an increase of creatinine (also above the reference range) during treatment, this is for the normalization of GFR (that before treatment is abnormally high). In such cases a reduction of the a methimazole/carmimazole is not usually indicated. However, if with the recovery of euthyroidism the cat becomes azotaemic and symptomatic, the drug dosage may be reduced to ensure that the concentration of T_4 remains within the upper half of the reference range.

Limited-iodine food: Production of thyroid hormone requires uptake by the thyroid gland of sufficient amounts of iodine, which is provided by dietary intake. The only function for ingested iodine is for thyroid hormone synthesis. This observation led to the hypothesis that limiting dietary iodine intake could be used to control thyroid hormone production and potentially manage hyperthyroidism in cats. After more than a decade of research and development, a limited-iodine therapeutic food (Hill's® Prescription Diet® y/dTM Feline) containing < 0.3 ppm (mg/kg) iodine on a dry matter basis (DMB), is now available as an option for managing cats with hyperthyroidism. Results from few recent studies suggest that an iodine-restricted food may be effective in reducing serum total thyroxine concentration in hyperthyroid cats. The main problem is the palatability and many cats after a while refuse the dete. Compared to oral or transdermal methimazole, the dietetic treatment did not cause any increase in serum creatinine, but was less effective in improving bodyweight, liver parameters and general control of the disease.

Treatment of canine Cushing's syndrome

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Cushing's Syndrome/Disease (spontaneous form) is represented by the clinical and laboratory alterations resulting from a chronic and pathological hypercortisolaemic state. It is very important to underline that there is no syndrome without clinical signs and asymptomatic dogs should not be tested. Before performing an endocrine test is very important to do complete hematology, urinalysis and serum biochemistry. The evaluation of the clinicopathologic abnormalities are very important to support the diagnosis and exclude relevant concurrent disorders (e.g. kidney disease, infections) that could influence the diagnostic tests. In order to obtain diagnostic confirmation in a subject with suspected hypercortisolism, **it is appropriate to have confirmation from at least two specific** endocrine **tests.** The tests and the other differential type diagnostic methods include LDDST, HDDST on urine, HDDST on blood, endogenous ACTH measurement, abdominal ultrasound and advanced diagnostic imaging techniques like computed tomography and nuclear magnetic resonance. The main treatment options for hyperadrenocorticism are medications (trilostane and mitotane) and surgery (adrenalectomy, hypophysectomy). Trilostane and mitotane are both effective for the pituitary form of the disease and for the adrenal form of the disease. The first treatment opinion for the adrenal for is surgery (adrenalectomy).

Treatment and monitoring TRILOSTANE:

1. The drug should be administered at a set time, SID or BID (maybe BID is better), with food, at a starting dose about 0.5 and 1 mg/kg. The first ACTH stimulation test should be performed 7-14 days after the beginning of treatment, 2-3 hours after the morning tablet (N.B. treatment should be made also on the day of the sampling). On this occasion, besides the clinical history and physical examination, it is recommended to perform some haemato-chemical-urine tests (control of kalaemia and of renal and haepatic parameters are especially important).
    - If the clinical signs have regressed and the post-ACTH blood cortisol levels are between 1.5 and 5.4 mcg/dl (40-150 nmol/l), the treatment is defined optimal and maintained until the next controls.
    - If the clinical signs have regressed, but the blood cortisol levels are below 1.5 mcg/dl in both samples, the recommendation is to discontinue treatment for 5-7 days and to then resume it at a lower dosage. In most cases these patients do not show signs of hypoadrenocorticism since blood cortisol levels tend to increase rapidly.
    - If post-ACTH blood cortisol levels are above 5.4 mcg/dl and the dog does not show clinical improvement, the dosage is increased. No precise indications are available to date with regard to an optimal increase in the dosage for dogs that fall within this category; however, the following scheme proposed by Galac *et al*. (2008) may be used: from 10 to 20 mg, from 20 to 30 mg, from 30 to 40 mg, from 40 to 60 mg, from 60 to 90 mg, from 90 to 120 mg, etc.
    - If the patient presents blood cortisol levels as in point a) but not clinical improvement is present, based on the severity of the symptoms decide on whether to increase the dose, change to administration every 12 hours or re-evaluate after 4 weeks continuing with the same dose.
    - If the clinical signs have regressed but post-ACTH blood cortisol levels are between 5.4 and 9.0 mcg/dl (150-250 nmol/l), continue with the same dose and check again after 4 weeks.
    - If the dog is in an obvious pathological state: discontinue treatment, identify the cause of the disease (perform ACTH stimulation test and haemato-chemical-urine tests) and treat symptomatically.
2. Physical, haematological and biochemical signs are to be monitored and the ACTH stimulation test is to be repeated 4 and 12 weeks after the beginning of treatment, then every 3 months and 10 days after every dosage variation. The procedures listed in point 1 are also applicable for medium-long term monitoring.

NEW POSSIBLE MONITORING OF DOGS TREATED WITH TRILOSTANE: A proposed method for monitoring dogs being treated with trilostane without the use of ACTH stimulation test results includes the use of the serum cortisol concentration measured just before giving the morning dose of trilostane. The "pre-pill cortisol" has been found to be in better agreement with an owner-based scoring scheme than the post-ACTH cortisol in one study based on 70 measurements made on 53 dogs, most treated once daily but some being treated twice daily, when monitored after 1 month on a consistent dose of trilostane. Good clinical control tends to be associated with pre-pill cortisol concentrations in the range of 40-120 nmol/L (1.4-4.3 mcg/dL) and can be regarded as the approximate target range. Since the cortisol concentration fluctuates, ideally the pre-pill pill value should be obtained from the mean of 2 measurements of cortisol obtained 1 h distant from each other, before trilostane administration.

Treatment and monitoring MITOTANE:

Mitotane must be administered together with food as this increases its absorption. To obtain a selective action

on the fascicular and reticular zones, treatment with o,p'-DDD involves a loading phase with a dose of 25-50 mg/kg subdivided into two administrations for 5-9 days. It is not necessary to administer glucocorticoids, but it is advisable to keep them on hand in case of emergency. It is appropriate to discontinue treatment whenever the patient shows a drop in appetite, when water consumption decreases below 60 ml/kg/day, in case of vomiting, diarrhoea or if the animal appears tired and lethargic. An ACTH stimulation test is performed after the first phase; if the response is normal or excessive, the treatment is continued with 3-7 day cycles. This is followed by the maintenance phase, in which the initial daily dose is administered within the space of one week (in 2-3 administrations). Treatment monitoring continues with the ACTH stimulation test. It is recommended to perform the ACTH stimulation test after the induction phase, one month after the beginning of the maintenance period, then every 3-6 months, unless problems occur, such as the reappearance of the signs and symptoms ascribable to Cushing's Syndrome or symptoms of hypocortisolism or other side effects. For complete lysis of the adrenal cortex the drug has to be administered for 25 continuous days at a dose of 50-75 mg/kg (100 mg/kg in small dogs) subdivided into three or four doses, always given with food, to reduce the gastrointestinal tract problems that may occur. The drug should be administered daily for the first 5 days and then every other day. The replacement therapy with cortisone acetate (2 mg/kg/day) (in alternative prednisolone) and fludrocortisone acetate (0.0125 mg/kg/day), both divided preferably into two daily administrations, begins on the third day. A follow-up visit is performed after the 25th day and the cortisone acetate/prednisolone dose may be reduced to 0.5-1.0 mg/kg per day. The subject must then be treated as an Addisonian for the rest of its life.

Surgery for adrenal tumor (ADH) (medical considerations):

ADH can be treated surgically, performing an adrenalectomy of the gland affected by the neoplasm. However, before the operation it is essential to evaluate whether or not the subject is a good candidate for the surgery. It is important to evaluate blood pressure; indeed, hypercortisolism causes hypertension in over 50% of subjects and the urinary protein/creatinine ratio is often increased. A marked increase in these parameters could indicate a high risk of thromboembolism. For the same reason it is advisable to make sure that the antithrombin III concentration in the serum is not below the norm. Some authors believe that a pre-treatment with trilostane to control the clinical signs is recommended. During the operation and in the first six hours after surgery, it is important to administer 0.05-0.1 mg/kg i.v. of dexamethasone to the subject. The same dosage should be repeated 2 or more times during the day of the operation. On the following days the same dosage should be administered subcutaneously BID or TID until the animal is able to take it orally without risk of vomiting. Hydrocortisone, with both glucocorticoid and mineralocorticoid activity, can replace dexamethasone During surgery, hydrocortisone can be given IV (4 to 5 mg/kg) and thereafter 1 mg/kg, IV, every 6 hours until oral medication is tolerated. An ACTH stimulation test, completed the morning after surgery and about 8 hours after the last dose of dexamethasone, helps to determine whether the tumor was completely excised (low serum cortisol concentrations before and after ACTH) and to determine need for continuing glucocorticoid supplementation. Intensive postoperative monitoring is essential for preventing or responding to complications. Hyperkalemia and/or hyponatremia should be treated with mineralocorticoid: oral fludrocortisone (0.01 to 0.02 mg/kg q 12 h) or IM desoxycorticosterone pivalate (DOCP; 2.2 mg/kg SC every 21-25 days). Electrolyte abnormalities may reflect mineralocorticoid deficiencies and are usually, but not always, transient, only lasting a few days. Antibiotics, analgesia, and heparin (75 units/kg SC q 8 h) may be recommended. Oral prednisone should be considered for all dogs whose post-surgical ACTH stimulation test results were below normal. An initial dosage of 0.5 mg/kg PO q 12 h for 3 days is then tapered to 0.2 mg/kg/day over 2-4 weeks. Results of an ACTH stimulation test performed then, and months later, should be used to determine need for continuing glucocorticoid supplementation.

Surgery for pituitary tumors (PDH) (medical considerations):

Transsphenoidal surgical removal of the pituitary tumor causing PDH is the treatment of choice for humans, while complete surgical hypophysectomy has been used for dogs with PDH. After hypophysectomy in PDH dogs, the 1-year estimated relapse-free fraction was 90%. The 1, 2, 3, and 4-year estimated survival rates (86, 83, 80, 79%, respectively) compare favorably to results seen in dogs treated with either mitotane or trilostane. This kind of surgery is performed only in few centers and only by few surgeons. A good intensive care unit is very important to manage the post-operative period. Postoperative intensive care includes close monitoring of vital functions, plasma electrolytes (sodium and potassium), plasma osmolality, and central venous pressure. Oral water intake is encouraged as soon as possible. Postoperative medication includes antibiotics and analgesics. Hormone replacement consists of hydrocortisone (1 mg/kg IV every 6 hours) and desmopressin, a vasopressin analogue (4 µg administered as a drop into the conjunctival sac every 8 hours for 2 weeks). When the dog has resumed eating and drinking, oral replacement therapy is started: cortisone acetate (1 mg/kg every 12 hours) and thyroxine (15 µg/kg every 12 hours). Over a period of 4 weeks the dose of cortisone acetate is gradually tapered to 0.25 mg/kg every 12 hours. Desmopressin (0.01%) is administered for 2 weeks, 1 drop into the conjunctival sac every 8 hours.

Addison's disease: how to recognize, diagnose and treat the disease

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Hypoadrenocorticism, or Addison's disease, is uncommon in the dog (2.3 cases every 10000 patients) and even rarer in the cat (around 40cases described) and is characterised by an insufficient production of mineralcorticoids and/or of glucocorticoids by the cortical substance of the adrenal glands. Hypoadrenocorticism has also been called "the great imitator" since, from the symptomatological viewpoint, it mimics other diseases and is therefore a challenge for clinician. Symptoms usually appear when at least 85-90% of the cortical tissue of the adrenal glands is impaired and they can appear subtly and progressively or quickly and suddenly. Although the symptomatological picture is extensive and non-specific, the diagnostic protocol is easily carried out and the treatment, if correctly performed, makes it possible to ensure the patient a completely favourable prognosis.

Hypoadrenocorticism derives from a spontaneous or iatrogenic alteration of the hypothalamic-pituitary–adrenal axis. Based on the location of the problem the forms present may be primary (adrenal), secondary (pituitary) or tertiary (hypothalamic).

The hereditary, autosomal recessive nature of the disease has been described in some canine breeds, such as the Portuguese Water Dog, Poodle and Nova Scotia Duck Tolling Retriever.

Occasionally, hypoadrenocorticism may be associated with one or more endocrine gland disorders, such as hypothyroidism, diabetes mellitus, hypoparathyroidism, vitiligo or azoospermia. In these cases reference is made about "autoimmune polyglandular syndrome".

Iatrogenic hypoadrenocorticism is caused by drugs (e.dg trilostane or mitotane), used during hypercortisolism, which can damage the adrenal cortex. There is also a iatrogenic secondary form that usually occurs following the sudden discontinuation of a glucocorticoid treatment.

Hypoadrenocorticism may affect subjects of all ages, but it is more common in young animals (average 4-5 years). In dogs it occurs more often in females than in males (69%). Hypoadrenocorticism can appear in any breed, but some have a higher incidence (e.g. Great Dane, Poodle, West Highland White Terrier, Portuguese Water Dog, Bearded Collie, Rottweiler, Soft Coated Wheaten Terrier, Springer Spaniel, Basset Hound, Nova Scotia Duck Tolling Retriever, Saint Bernard).

In 25-43% of the cases clinical signs are episodic and irregular, while in other patients the symptoms show a progressive trend. The number and severity of the clinical signs, as well as the speed of progression of the endocrine disorder, vary from subject to subject. In fact, at the physical examination, the patient may present from moderately dehydrated but alert, to severely dehydrated, in shock, with increased capillary refill time and a weak pulse. It should be underlined that the various forms of hypoadrenocorticism are not easily distinguishable through the clinical signs. The most common symptoms are disorexia/anorexia, letargy, vomiting/regurgitation, weakness, weight loss, diarrhoea, bradycardia, collapse, polyuria/polydipsia, tremors, weak pulse, abdominal pain and hypothermia.

Complete blood count: a mild normochromic, normocytic, nonregenerativeanaemiais found in 21-25%of subjects. In a debilitated patient, the absence of a stress leukogram (lymphopenia, eosinopaenia) allows to suspect the presence of hypoadrenocorticism, especially in the atypical form in which the absence of electrolytic alterations makes it more difficult to suspect the disease. Eosinophilia is found in 10-20% of cases and lymphocytosis in 10-13% of patients.

Biochemical profile: hyperkalaemia (around 95% of patients) with the classic form. This finding is not observed during secondary or atypical hypoadrenocorticism. Hyponatraemia: (86%) of patients with primary hypoadrenocorticism. The same alteration appears in 34% of subjects with the secondary form, following gastrointestinal losses and lack of appetite. Other abnormalities are: hypochloraemia, low (<27) Na:K ratio, pre-renal azotaemia, hyperphosphataemia, low urine specific gravity (sg),metabolic acidosis hypercalcaemia, hypoglycaemia, hypoalbuminaemia, increase in alanine aminotransferase (ALT) and aspartate aminotransferase (AST) and hypocholesterolaemia.

Abdominal ultrasound: Through abdominal ultrasound it is possible to highlight a decrease in the length and thickness of the adrenal glands, however, their dimensions in subjects with hypoadrenocorticism may sometimes be similar to those of a healthy animal.

Hypotension is frequently found in subjects with hypoadrenocorticism.

The detection of bradycardia should lead to the suspicion of the presence of a cardiac conduction disturbance

triggered by hyperkalaemia. However, the heart rate can be normal when the potassium concentrations are not high enough to cause it to decrease and/or when it is increased by the sympathetic action triggered by hypovolaemic shock; thus the ECG of a patient with hypoadrenocorticism may be normal or show serious alterations, including:

bradycardia, tachyarrhythmia, conduction disturbances, sinoventricular rhythm ventricular fibrillation, asystole.

ACTH stimulation test: This is a simple and easy-to-perform test. It should be carried out before starting treatment with corticosteroids; however, the administration of dexamethasone before the test does not change the results. It is the **gold standard** for the diagnosis of hypoadrenocorticism since the test verifies the ability of the adrenal glands to produce cortisol following maximum stimulation. The test is performed by measuring the basal blood cortisol level before the intravenous or intramuscular administration of synthetic ACTH (5 µg/kg in dogs and 125 µg in cats) and measuring the cortisol level a second time one hour after administration. Serum cortisolo post ACTH < 2.0 µg/dl is suggestive of hypoadrenocorticism.

Basal cortisolaemia: The measurement of basal cortisol alone is not sufficient to make a diagnosis of hypoadrenocorticism, however recent studies show that a basal cortisolaemia below 1.0 µg/dl (27.59nmol/L) has 100%sensitivity and 98.2%specificity for detecting subjects with the disease. Basal cortisolaemias < 2 µg/dl (55.2 nmol/L) have 100% sensitivity but a lower specificity (78.2%). It is unlikely that a subject with hypoadrenocorticism will have a basal cortisolaemia >2 µg/dl (55.2 nmol/L). Consequently, the evaluation of basal cortisolaemia is useful for excluding hypoadrenocorticism; nevertheless the ACTH stimulation test is necessary to confirm the disease.

Endogenous ACTH

By measuring endogenous ACTH it is possible to distinguish a primary from a secondary hypoadrenocorticism: subjects with the primary form have high endogenous ACTH (>100 pg/ml) while those with secondary hypoadrenocorticism, on the other hand, have endogenous ACTH below the reference values (<20 pg/ml).

TREATMENT DURING AN ADDISON CRISIS: An Addison crisis is a true medical emergency since the patient usually presents in a state of severe hypovolaemia, dehydration, hypotension and with electrolyte and acid-base imbalances. Treatment must therefore be started immediately with the following interventions:

- restore blood volume and the state of hydration;
- resolve the electrolyte imbalances (especially hyperkalaemia and hyponatraemia);
- restore the acid-base balance;
- supplement corticosteroids.

Blood volume and perfusion must be restored quickly through the intravenous infusion of fluids. In the case of shock the speed of infusion in dogs should be up to 60-90 ml/Kg for the first 1-2 hours and then the dosage should be adjusted based on the needs of the individual patient. It is appropriate to infuse saline solution (NaCl 0.9%) since it makes it possible to restore the natremia, to reduce the kalaemia and to improve the acid-base state. When NaCl 0.9% is not available, other types of crystalloids may be used (lactated or acetated Ringer's solution); the small amount of potassium contained does not seem to harm the patient undergoing an Addison crisis. Fluid therapy should be continued for at least 48 hours, monitoring the patient frequently. Hypoglycaemia, when symptomatic, should be treated with an initial bolus of 0.5-1 ml/Kg of 50% glucose solution, administered intravenously in 15-20 minutes. For hyperkalaemia the first intervention consists in carrying out a suitable fluid therapy that allows a decrease in the kalaemia owing to the dilution effect, to the improvement in renal perfusion, to the potassium "shift" from the extracellular to the intracellular space and thanks to the improvement in the metabolic acidosis**.** In the most severe cases, often associated with bradyarrhythmias, an additional therapeutic intervention becomes appropriate:

The simplest treatment for lowering kalaemia consists in the intravenous administration of 0.2 IU/Kg of regular insulin, followed by a bolus of glucose (4 ml of 50% glucose solution for each unit of insulin administered) and by the addition of glucose to the fluids (5%). The insulin transports the potassium inside the cells and thus lowers the kalaemia. After the administration of insulin, glycaemia should be monitored every 30-60 minutes.

Another method for lowering kalaemia, to be used only if the patient can be closely monitored with blood-gas analysis, consists in the slow administration of 1-2 mEq/Kg of sodium bicarbonate intravenously. The effect appears after about one hour and lasts several hours. This procedure can be dangerous due to the risk of development of a difficult to correct metabolic alkalosis.

The administration of 10% calcium gluconate (0.5-1 mL/Kg up to 10 mL/dog through intravenous infusion in 10-15 minutes) may be useful in critical subjects. This intervention does not lower the kalaemia but merely limits the cardiac conduction disturbances that a severe hyperkalaemia can trigger. By virtue of the well-known side effects during infusion, it is essential to monitor the ECG.

During the recovery of the electrolyte balance, the natremia should not increase by more than 10-12 mmol/L per day. Indeed, a rapid increase may cause damage to the central nervous system (pontine myelinolysis). The clinical signs connected with this complication arise several days after correction of the natraemia and consist

in: lethargy, weakness, lockjaw, decreased response to threat, ataxia that progresses to hypermetria and spastic tetraparesis. Thus, in cases of severe and protracted hyponatraemia, it might be appropriate to administer fluid therapy based on NaCl 0.45%.

Many patients undergoing an Addison crisis have a mild-moderate metabolic acidosis that is usually resolved with fluid therapy. When a severe acid-base imbalance persists (pH<7.1 or HCO3<12 mmol/L) in spite of the fluid therapy, sodium bicarbonate can be administered, calculating the patient's HCO_3 deficit with the following formula:

bicarbonate deficit (mmol/L) = 0.3 x body weight in Kg x (24 – patient's HCO_3)

It is necessary to administer ¼ or ½ of the calculated deficit intravenously in 2-4 hours and then repeat the blood-gas analysis. The goal of the administration of bicarbonates should not be to resolve the metabolic acidosis completely but to bring the patient's bicarbonate level above 12 mmol/L and the pH above 7.2.

Glucocorticoid supplementation

Glucocorticoid supplementation is of essential importance when treating an Addison crisis and it should be carried out in conjunction with fluid therapy. Ideally the ACTH stimulation test should be carried out before administering corticosteroids since, except for dexamethasone, they can alter the test results.

	DOSAGE	CHARACTERISTICS
Dexamethasone sodium phosphate	0.2 mg/Kg i.v. every 2-6 hours	Fast action. May be administered before the ACTH stimulation test. Does not have any mineralcorticoid activity.
Prednisolone sodium succinate	1 mg/Kg i.v. every 2-6 hours	Has slight mineralcorticoid activity
Methylprednisolone sodium succinate	1 mg/Kg every 2-6 hours	
Hydrocortisone sodium succinate	0.625 mg/Kg/h i.v. or initial bolus of 5 mg/Kg followed by 1 mg/Kg i.v. every 6 hours	Also has mineralcorticoid activity

Mineralcorticoid supplementation

Some authors maintain that intensive fluid therapy and glucocorticoid supplementation are sufficient to stabilise the patient, while others recommend starting mineralcorticoid supplementation during the crisis.

	DOSAGE	NOTES
Desoxycorticosterone acetate (DOCA)	0.1 mg/Kg s.c once a day	Short-acting, parenteral formulation
Desoxycorticosterone pivalate (DOCP)	2.2 mg/Kg i.m. every 25 days	Not available in Italy. Do not administer i.v.!
Fludrocortisone	0.01 mg/Kg *per os* twice a day	Only in tablets

Other adjunctive therapies

Many patients undergoing an Addison crisis experience vomiting and/or nausea, thus it may be useful to administer antiemetics and gastroprotectors when these symptoms do not disappear with the beginning of the treatment described above. A blood transfusion may be indicated when the anaemia is very severe. A broad-spectrum antibiotic therapy can also be started since a bacterial translocation from the damaged intestine is possible, with consequent sepsis. The Addison crisis is usually resolved in a few hours with suitable fluid therapy and glucocorticoid supplementation.

MAINTENANCE THERAPY

Patients with classic primary hypoadrenocorticism require lifelong glucocorticoid and mineralcorticoid supplementation. Subjects with atypical primary hypoadrenocorticism will require lifelong glucocorticoid supplementation only; the mineralcorticoids may instead be discontinued after a certain period of time. Animals with secondary hypoadrenocorticism require lifelong glucocorticoid supplementation but not for mineralcorticoids.

Glucocorticoid supplementation

	DOSAGE	NOTES
Prednisone	0.22 mg/Kg *per os* twice a day	Owner should be advised that during times of stress (trips, changing house, etc.) the dose should be increased by 2-10 times the dose normally used.
Prednisolone		0.1-0.2 mg/Kg *per os* twice a day

Mineralcorticoid supplementation

MINERALCORTICOIDS	DOSAGE	NOTES
Desoxycorticosterone pivalate (DOCP)	2.2 mg/Kg i.m. every 25 days	Long acting. Formulated for i.m. injections. Pure mineralcorticoid. Initially controls at 12 and 25 days after each injection for 2-3 injections. Control at 12 days to correct the dosage: should electrolytic alterations persist, the next time increase the dosage by 5-10%. If hypokalaemia and/or hypernatraemia appear, decrease the dose by 5-10%. Control at 25 days: this allows to decide on the frequency of administration. If hyperkalaemia and/or hyponatraemia appear on the 25th day, decrease the frequency of administration by 1 day.
Fludrocortisone acetate	0.01 mg/Kg *per os* twice a day	Increase the dosage by 0.05-0.1 mg/Kg/day when the electrolytes are abnormal. It also has a glucocorticoid activity, therefore only 50% of patients require an additional glucocorticoid supplementation. It is often necessary to increase the dosage during the first 6-10 months of treatment.

MONITORING AND PROGNOSIS

The goal of treatment should be to eliminate the clinical signs and to have a patient in a good state of health and with haematobiochemical parameters within the normal range. Once a suitable dosage of corticosteroids has been attained, the controls can be carried out every 3-4 months. The control consists of gathering the medical history, a careful clinical examination and execution of haematobiochemical tests (at least electrolytes).

Every time the patient shows the onset of clinical signs he/she must be re-evaluated quickly. The presence of electrolytic alterations suggests an adjustment of the mineralcorticoid level, while the onset of vomiting, diarrhoea, lack of appetite or lethargy indicates that the glucocorticoid component should be increased.

Subjects with hypoadrenocorticism, if treated correctly, have an absolutely favourable prognosis and can lead a totally normal life.

How to approach the patient with polyuria and polydipsia

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The symptom polyuria / polydipsia (PU / PD) is quite common to dogs and cats and requires an accurate, but above all, systematic medical approach. From most of the authors, polyuria is defined in the dog as an intake of water> 100 ml/kg /day while in the cat> 45 ml/kg/day. This data is easily obtainable by asking the owner to measure his own animal's water within 24 hours. In the dog, polyuria is defined as a urine output greater than 50 ml / kg / day. For obvious reasons this is difficult to objectify. Urine production and water production are controlled by the interaction between the kidneys, the pituitary gland and the hypothalamus. Urine production is also influenced by atrial baroceptors that are present at the level of the aortic arch. The antidiuretic hormone (ADH) is produced in thehypotalamus and released by the neurohypophysis following appropriate stimuli. Its action is performed at renal tubular level, allowing the opening of the water channels, allowing for the re-absorption of H2O by osmotic gradient and influencing urine concentration. To get properly concentrated urine, there is a need for proper ADH secretion and action, at least 1/3 of the nephrons being functioning and that there is adequate osmolality in the renal medulla. From a classification point of view there is primary polydipsia (with secondary polyuria) or primary polyuria (with secondary polydipsia). In both situations the causes can be central (encephalus / pituitary) or peripheral (kidney).

The causes of **primary polydipsia** are rather infrequent and are essentially represented by:

- Psychogenic polydipsia
- Abnormalities in the center of thirst
- Metabolic disorders (eg hyperthyroidism, liver disease)

Primary polyuria is much more common and given by:

DIABETES INSIPIDO PRIMARY CENTRAL
DIABETES INSIPIDO SECONDARY CENTRAL

- Trauma induced
- Neoplastic
- Cushing's syndrome
- Drugs
- Polycythaemia
- Blood hyperviscosity
- Pheochromocytoma
- Gastrointestinal disorders

PRIMARY NEFROGENIC DIABETES INSIPIDUS

SECONDARY NEFROGENIC DIABETES INSIPIDUS:

- Acromegaly
- Aldosteronoma
- Liver disease
- Hypercortisolism
- Hypercalcemia
- Hyperthyroidism
- Hypoadrenocorticism
- Hypokalemia
- Chronic renal disease
- Pyelonephritis
- Pyometra
- Septicaemia (E. coli)
- Drug administration

Osmotic diuresis (excess of diuresis is linked to the presence of osmotically active molecules at the tubule collector level)

- Diabetes mellitus
- Resolving urethral obstruction
- Osmotic diuretics
- Renal glycosuria

RENAL MIDDLE HYPOTONICITY

- Hepatic impairment
- Kidney failure
- Addison's disease
- Hypoproteic regimen

In evaluating the patient with PU / PD it is essential to evaluate some aspects:

- The animal species and breed: some breeds are particularly predisposed to particular diseases, for example in a Boxer dog we usually first think of a Cushing's syndrome or an hypercalcemic neoplastic disease rather than diabetes mellitus since diabetes mellitus is extremely rare in this breed
- Distinguish PU / PD from pollachisuria or urinary incontinence
- The onset of symptoms
- The duration of the symptoms
- The age at which the symptoms have arisen: congenital forms such as diabetes insipidus or hepatic shunt (congenital vascular abnormality) are usually present in young sanimals, and diseases such as feline hyperthyroidism are extremely rare in animals under 8 years of age.
- Possible medication (especially corticosteroids or diuretics)

An adequate and complete physical examination should be performed in all animals with PU / PD and in particular it is important to focus on:

- Nutrition status (thinness in neoplastic diseases, hyperthyroidism, etc., obesity in animals with diabetes mellitus)
- Exportable lymph nodes (neoplastic diseases, hypercalcemia)
- Dermatological changes (Cushing's syndrome, hyperthyroidism)
- Vaginal discharge (pyometra)
- Abdominal palpation (masses, kidney problems, pyometra, hepatomegaly, etc.)
- Rectal exploration (anal sac carcinoma)
- Thyroid palpitation (hyperthyroidism)

Following the physical examination, the first laboratory evaluations to be performed are the evaluation of glycosuria and urinary specific gravity. A classic diagnostic protocol that can be performed in animals with PU/ PD, intended to confirm or exclude possible differential diagnoses is as follows:

- Urinalysis + colture evaluation (urine taken for cystocentesis)
- CBC
- Biochemical profile and ideally ionized calcium evaluation.
- Adrenal function tests (dog) if indicated
- Measurement of serum T4 (cat)
- Chest radiography
- Abdominal ultrasound
- H2O deprivation test or similar test (eg clinical response of the animal to the administration of desmopressin)

ENT – medicine and surgery

Gert ter Haar (NL)
DVM, PhD, MRCVS, DECVS

Gert ter Haar is from the Netherlands and studied veterinary medicine at the Faculty of Veterinary Medicine in Utrecht, where he graduated in February 1997. After an internship and surgical residency in Utrecht, he became a Diplomate of the European College of Veterinary Surgeons in 2002 and Head of the Department of ENT at the University of Utrecht in February 2003. His research on hearing in dogs led to his PhD thesis, a 220-page book entitled "Age-related hearing loss in dogs" in 2009. On the same day he became President of the International Veterinary Ear Nose and Throat association (IVENTA). He joined the surgical team of the Royal Veterinary College in London, United Kingdom, in 2011 as a senior lecturer in soft tissue surgery and got promoted to associate professor in soft tissue surgery and head of the ENT department and ENT, Audiology and Brachycephaly clinics at the RVC in 2015. In March 2017 he joined the team of the SDU (specialistische dierenkliniek Utrecht), the largest European specialist referral centre in the Netherlands. He is author and co-editor of three surgical books and has published over 20 peer-reviewed articles in his field of interest. He gave over 300 national and international lectures on ENT medicine & surgery, hearing research in dogs and soft tissue/reconstructive surgery in dogs and cats. His current research involves hearing loss in dogs and cats and brachycephalic obstructive airway syndrome.

Pharyngolaryngoscopic diagnosis in dogs and cats

Gert ter Haar DVM, PhD, MRCVS, DECVS

SPECIALISTISCHE DIERENKLINIEK UTRECHT

Otorhinolaryngology

ENT

Pharyngolaryngoscopic Diagnosis in Dogs and Cats

Gert ter Haar, DVM, PhD, DECVS
European Specialist in Small Animal Surgery
Ear, Nose & Throat, Brachycephaly related and Audiologic Diseases
Oncologic and Reconstructive Surgery

AniCura

Introduction to Lecture

ENT

- Anatomy pharynx & Larynx
- Physiology pharynx & larynx
- Pharyngolaryngoscopically diagnosed diseases

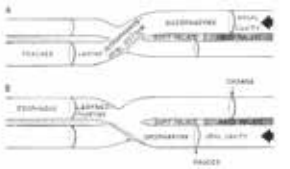
SDU

Anatomy & Physiology 1

ENT

- Pharynx

  Fibromuscular tube that serves as a conduit for both the respiratory and the digestive tract



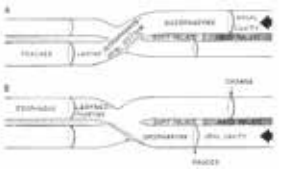
Evans H.E. and Christensen G.C. Anatomy of the DOG, 2nd edition, 1993; The Respiratory Apparatus, fig 8-9, page 517

SDU

Anatomy & Physiology 2

ENT

- Pharynx
  - Nasopharynx
  - Oropharynx
  - Laryngopharynx



Evans H.E. and Christensen G.C. Anatomy of the DOG, 2nd edition, 1993; The Respiratory Apparatus, fig 8-9, page 517

SDU

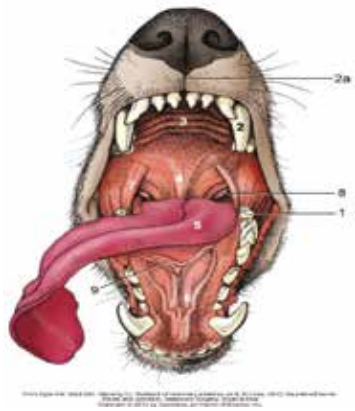
Physiology

ENT

- Pharynx
  - Swallowing (deglutition)
    - Physiological phenomenon that occurs many times daily
    - May be initiated voluntary, but most swallows occur subconsciously between meals, about once a minute in awake individuals
    - Driven by salivation, which stimulates the sensory receptors in the mouth and pharynx
    - Salivation & swallowing cease during sleep

SDU

ENT



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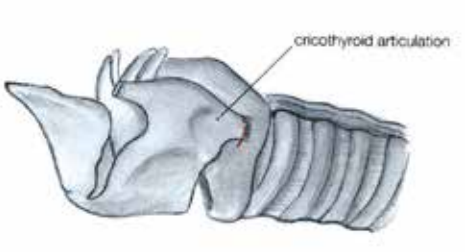
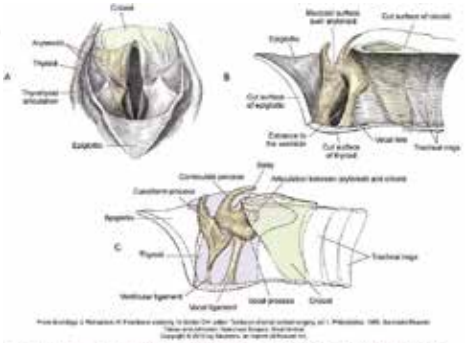
Laryngeal Anatomy



Hedlund C.S. Atlas of Small Animal Ear-, Nose-, and Throat-Diseases, 2002



Physiology

- Larynx
  - Protection of lower airways
    - Coughing reflex
    - Laryngeal closure reflex
  - Regulation of air flow
  - Vocalization

Diagnosis

- History
  - Patient
  - Clinical signs
    - Dysphagia, dyspnea, dysphonia, salivation, stridor!
- Physical Examination
  - Stridor!

Diagnosis

- Pharyngo-Laryngoscopy!
  - Throat inspection with laryngoscope
  - Nasopharyngoscopy
- Diagnostic Imaging
- Electromyography

Diagnosis

- (Naso)Pharyngo-Laryngoscopy!
- Electromyography

Diagnosis

- Diagnostic Imaging
  - Videofluoroscopy
  - Radiography
  - Ultrasonography
  - CT-scan / MRI

Diagnosis

- History
  - Patient
  - Clinical signs
    - Dyspnea, stridor, dysphonia, dysphagia and salivation!

Physical examination

- General examination
- Examination of respiratory system
  - Upper respiratory tract
  - Lower respiratory tract
- Examination of cardiovascular system

Diagnosis

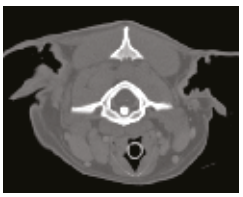
- Laryngoscopy!

Diagnosis

- Laryngoscopy!
- Diagnostic Imaging
  - Radiography

Diagnosis

- Laryngoscopy!
- Diagnostic Imaging
  - Radiography
  - Ultrasonography
  - CT-scan / MRI
- Electromyography

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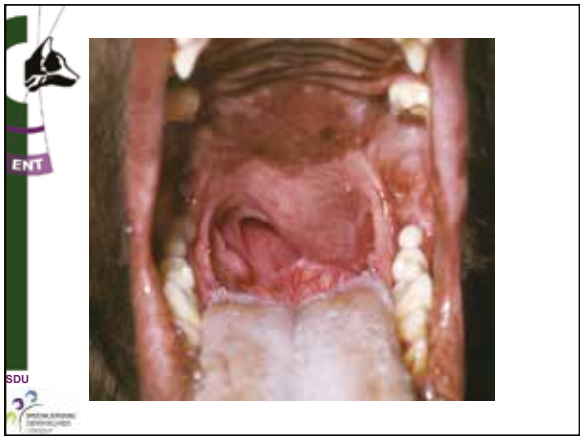
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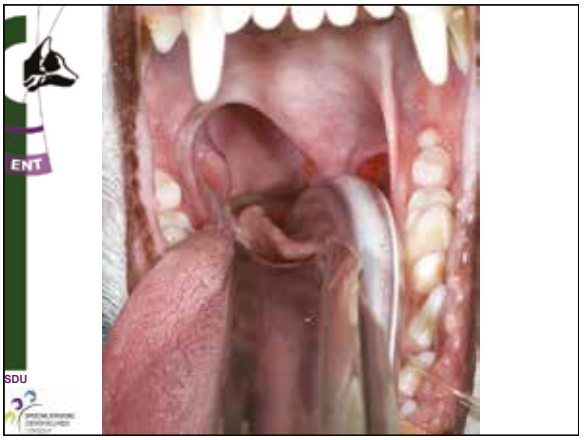
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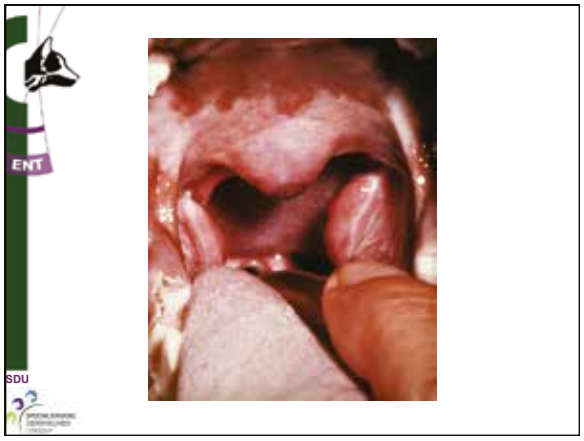
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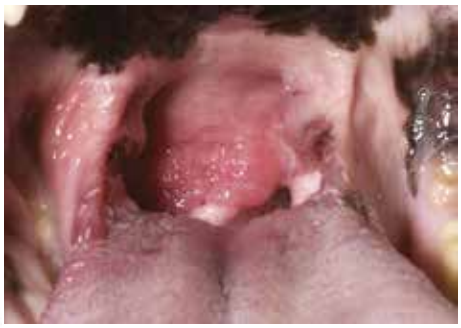
Stick Injury 1
ENT
SDU

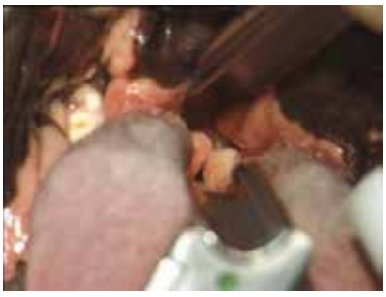
Pharyngeal mucocele

- Rather common abnormality in dogs and rare in cats
- Cyst arising from distended mucosal mucous glands in pharyngeal wall or soft palate
- Dyspnea and snoring
- Diagnosis on throat inspection
- Treatment: marsupialisation or removal

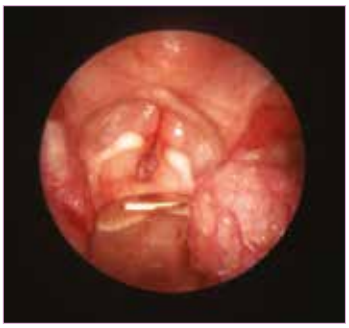
BOS: overlong soft palate



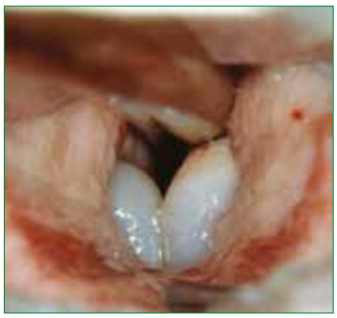
BOS: Patient 1

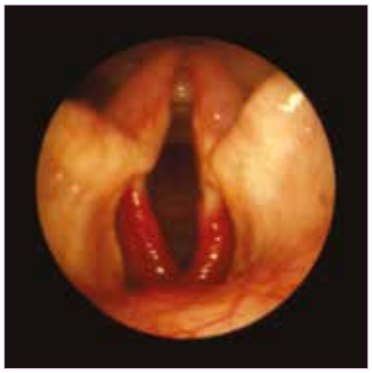


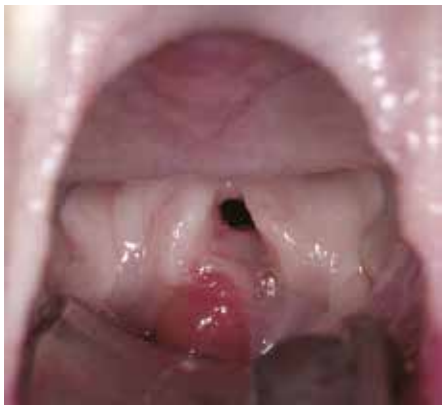
BOS: laryngeal hypoplasia

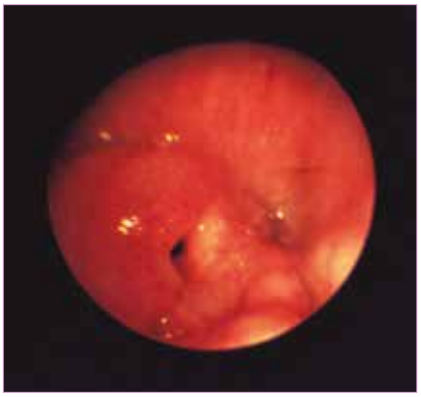


BOS: laryngeal collapse

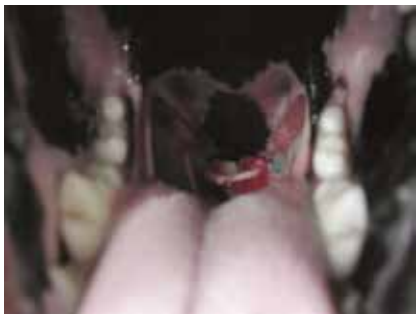


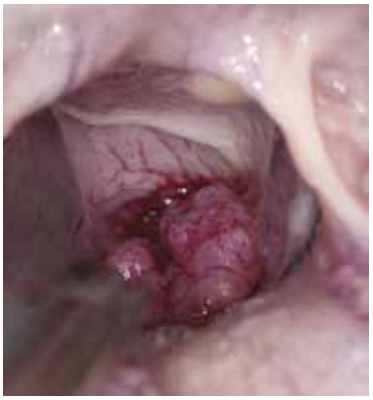


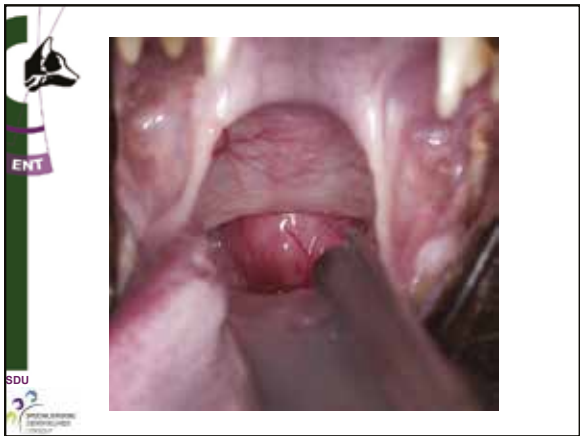


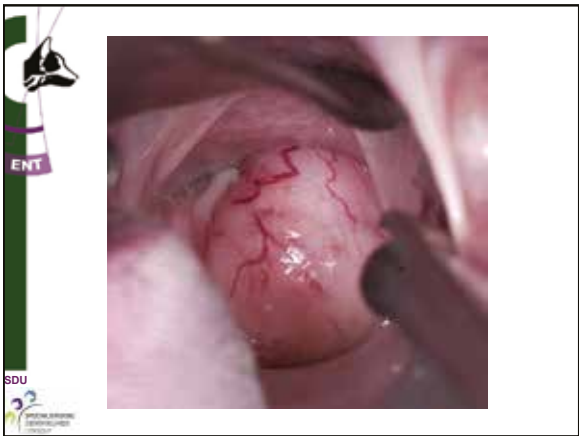


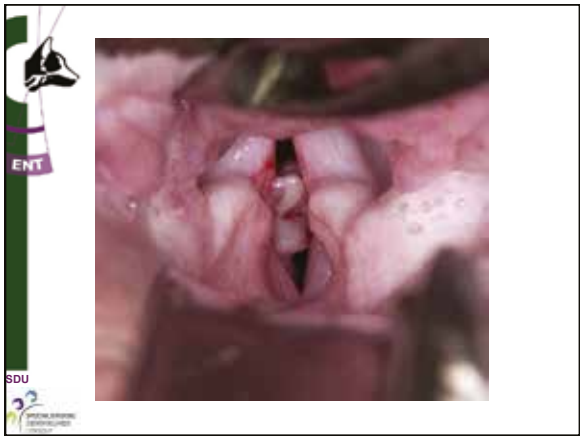
Laryngeal FB











Patient

- Maremmer Berghond, ♂, 9 years old
- Dyspnea with stridor

Patient

- Anatomical diagnosis ?
  - Larynx
- Differential diagnosis ?
  - Laryngeal paralysis
  - Laryngeal tumor
  - Laryngeal FB, abscess, stenosis, collapse

Patient

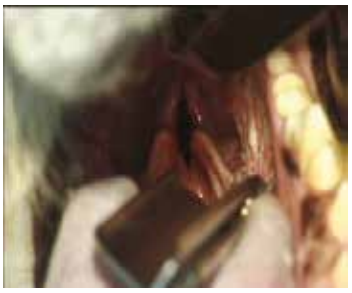
- Work up?
  - Radiography (neck and thoracic cavity)
  - Blood examination
  - Laryngoscopic examination under a light plane of anesthesia

Laryngeal function

Normal laryngeal function

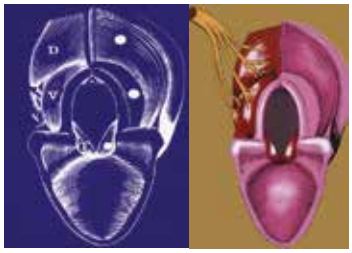
Patient

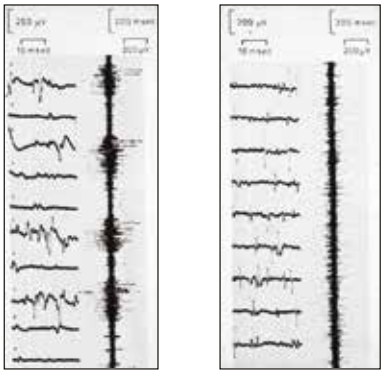
Laryngoscopy:



Laryngeal paralysis

- Diagnosis
  - EMG
  - Normal action potentials with denervation potentials





Discussion

- Pharyngoscopy
- Laryngoscopy

- Questions?

Endoscopic and surgical management of tracheal diseases

Gert ter Haar DVM, PhD, MRCVS, DECVS

SPECIALISTISCHE DIERENKLINIEK UTRECHT

Otorhinolaryngology

ENT

Endoscopic and Surgical Management of Tracheal Diseases

Gert ter Haar, DVM, PhD, DECVS
European Specialist in Small Animal Surgery
Ear, Nose & Throat, Brachycephaly related and Audiologic Diseases
Oncologic and Reconstructive Surgery
President of the IVENTA

AniCura

Introduction

ENT

- Cough
- Anatomy & Physiology
- History & Physical examination
- Diagnostic Tests
  - Radiography
  - Tracheoscopy / bronchoscopy
  - Tracheobronchial culture and cytology
- Patients with specific tracheal diseases

SDU

COUGHING

ENT

- A clearing mechanism to protect the lower airway
- A reflex evoked by stimulation of "cough receptors" or evoked voluntarily

SDU

The cough reflex

ENT

- First, a deep inspiration, glottis wide open
- The glottis closes and the expiratory muscles strongly contract
- Then sudden opening of the glottis forcefully expels air and any material

SDU

"Cough receptors"

ENT

- Sensory receptors responding to:
  - Airway pressure
  - Vocal fold motion
  - Tactile stimuli
  - Chemical stimuli
- Variable threshold

SDU

"Cough receptors"

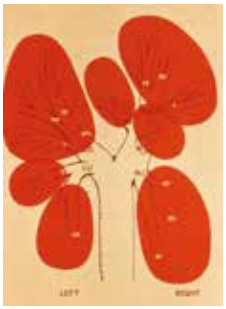
ENT

- Mucosa of the
  - Larynx
  - Trachea
  - Carina
  - Bronchi

SDU

Anatomy & Physiology

- Semirigid, flexible
- 35-45 C-shaped cartilages
- Elastic annular ligament
- Dorsal tracheal membrane

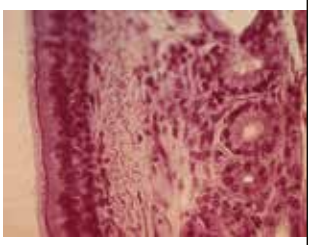


Anatomy & Physiology

- Pseudostratified ciliated columnar epithelium with goblet cells
- Submucosa with tubular glands

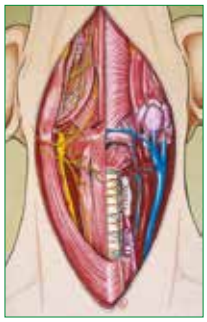
Anatomy & Physiology

- Mucociliary transport system propels mucus and inspired debris toward the pharynx



Anatomy & Physiology

- Thyroid arteries
- Recurrent laryngeal and vagus nerves
- Middle cervical ganglion and sympathetic trunk



History

- Clinical signs
  - Cough (gagging)
  - Stridor - Wheeze
  - Dyspnea
  - Exercise intolerance
  - Cyanosis/syncope
  - Hemoptysis

Physical examination

- General examination
- Examination of respiratory system
  - Upper respiratory tract
  - Lower respiratory tract
- Examination of cardiovascular system

Additional examination

- Diagnostic Imaging
- Endoscopy

Diagnostic tests

- Radiography
  - Two views
    - Lateral
    - Ventrodorsal or dorsoventral view
  - Cervical / thoracic trachea!
  - Videofluoroscopy (motion studies)
  - Different phases of respiration
  - Tracheobronchography

Diagnostic tests

- Tracheoscopy and bronchoscopy
  - Rigid scope (preferred) or flexible scope
  - Biopsy specimens
  - Cytology
  - Culture
  - Removal of foreign bodies
  - Evaluation disease progress or response to treatment

Diagnostic tests

- Transtracheal wash
  - Trough-the-needle style catheter
  - Large gauge hypodermic needle with small gauge urinary catheter
  - Cricothyroid ligament or between tracheal rings
  - Catheter to near the tracheal bifurcation
  - 5-20 cc saline infusion and suction

Transtracheal wash

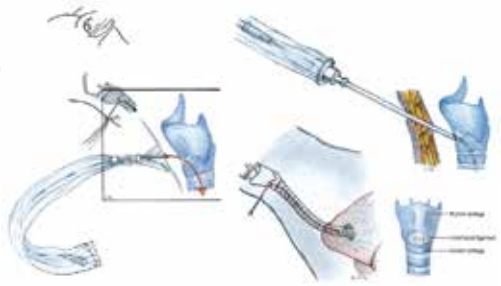


Fig. 4.4. Hedlund & Taboada; Clinical Atlas of ENT diseases in small animals, 2002

Tracheobronchoscopy

- Pre-oxygenation
- Rapid intravenous induction
  - Propofol
  - Isoflurane
  - NO ATROPINE in dogs
  - Sometimes atropine in cats
- Very superficial for laryngeal function!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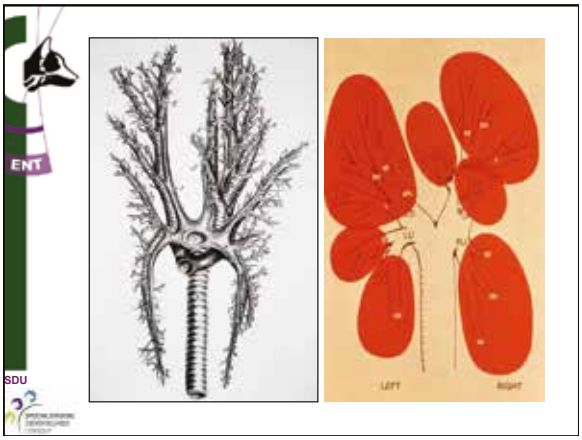
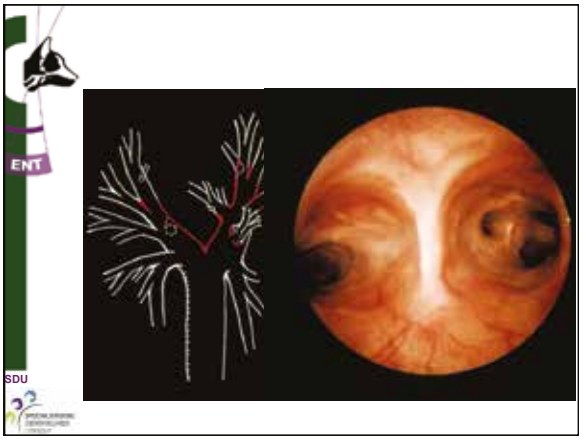
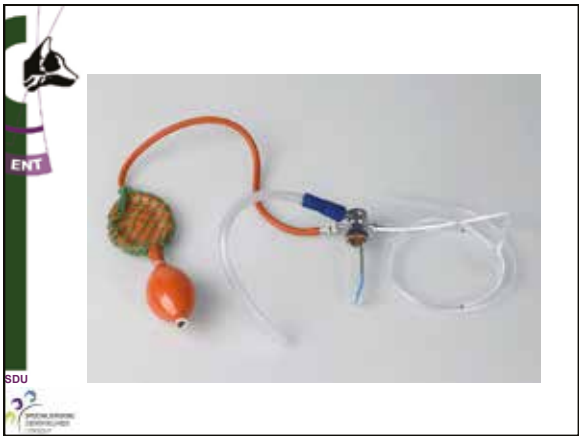




Tracheobronchoscopy

- Evaluate laryngeal function
- Evaluate trachea and tracheal dynamics
- Evaluate carina, mainstem bronchi and more peripheral bronchi
  - Local disease: BIOPSY
  - Diffuse disease: BAL (cytology & culture)
  - Pulmonary disease: BAL + more diagnostics



Patient 1

- Bouvier, male castrated, 3 years old
- Coughing, dry
- No systemic illness
- PE:
  - Bright and alert
  - Coughing on tracheal palpation
  - Normal lung sounds

Patient 1

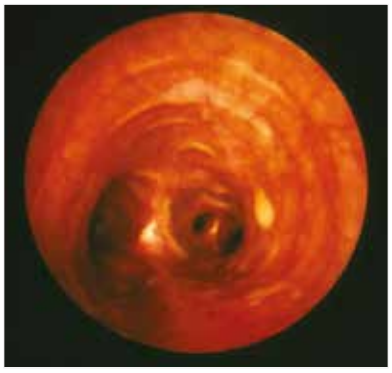
- Differential Diagnosis?

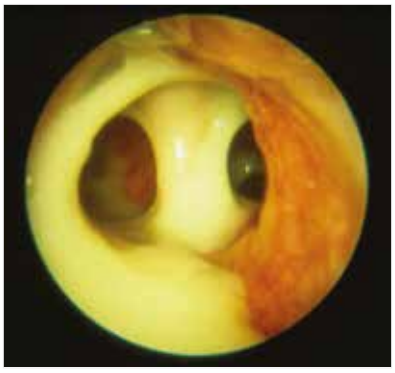
Patient 1

- DDx:
  - Infectious tracheitis or tracheobronchitis
  - Traumatic tracheitis
  - Irritant tracheitis
  - Parasitic tracheitis
  - Tracheal collaps
  - Tracheal obstruction (FB, tumor)
  - Acute bronchitis
  - Bacterial / viral pneumonia

Patient 1

- Diagnosis
  - History / PE
  - CBC
  - Thoracic radiographs
  - Transtracheal wash
    - Cytology
    - Bacterial culture and sensitivity testing
  - Tracheobronchoscopy





Patient 1

- Tracheoscopy
  - Red mucosal lining of trachea, edema and exsudate
- Cytology
  - Many neutrophils with intracytoplasmatic bacteria
- Culture
  - Bordetella and mycoplasma

Infectious tracheobronchitis

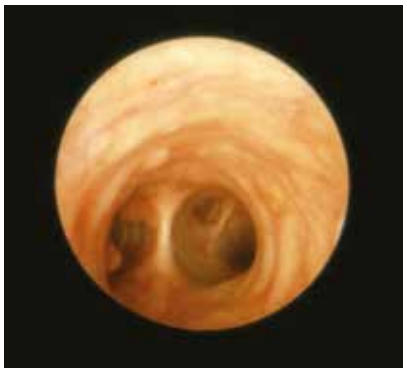
- Organisms
  - Viral
    - Canine parainfluenza virus
    - Canine adenovirus 2
    - Canine herpes virus
    - Canine reovirus
  - Bacterial
    - Bordetella bronchiseptica
    - Mycoplasma

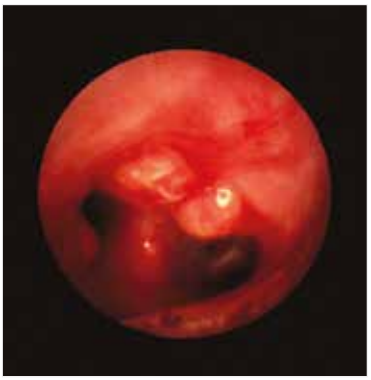
Infectious tracheobronchitis

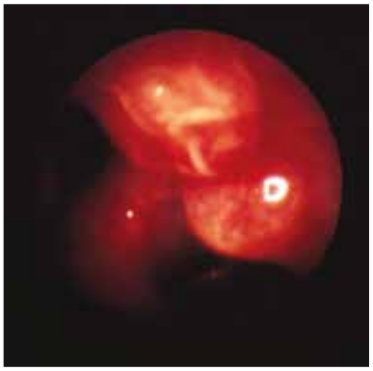
- Therapy
  - Usually selflimiting: no therapy!
  - AB only with sec. Bacterial infection!
    - Doxycycline 5 mg/kg PO 2 dd
    - Tetracycline 15-22 mg/kg PO 3 dd
    - Enrofloxacine 2.5-10 mg/kg PO 2dd
    - Amoxicilline/clav 12.5 mg/kg PO 2dd
  - Prednisolone (NOT in dogs with pneumonia)
  - Antitussiva/Bronchodilaters

Patient 2

- Yorkshire Terrier, female, 9 months
- Coughing for several months
- Dyspnea in the last few months
- No response on AB
- PE: soft cough on tracheal palpation
- Diagnosis?







Parasitic tracheitis

- Oslerus osleri (Filaroides osleri; nematode)
  - Usually in young dogs housed in kennels
  - Transmission from bitch to puppies through grooming or regurgitant feeding
  - Ingested L1 larvae migrate from intestine to right side of the heart and then to the tracheal wall to become adults

Parasitic tracheitis

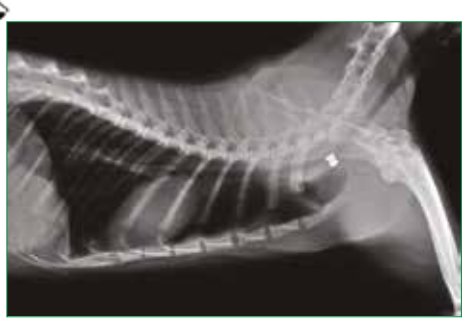
- Oslerus osleri
  - Irritant tracheitis with sign of coughing, wheezing and dyspnea
  - Typical small nodules (<8 – 10 mm) occur near the carina and main stem bronchi
  - Occasionally large parasitic granulomas

Parasitic tracheitis

- Oslerus osleri
  - Therapy
    - Fenbendazole 50 mg/kg PO, 1dd, 10-30 days
    - Ivermectin 0.2-0.3 mg/kg PO, once
    - Surgical or endoscopic removal may be necessary for persistent nodules

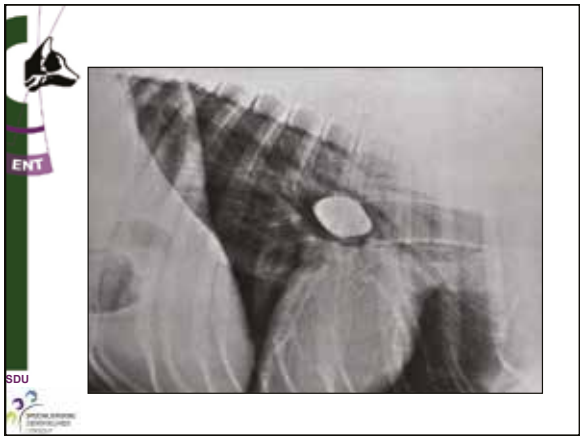
Patient 3

- Cat, male intact, 4 years old
- Progressive dyspnea
- Coughing
- PE: no abnormalities, slight cough on tracheal palpation
- Work up
  - Radiography



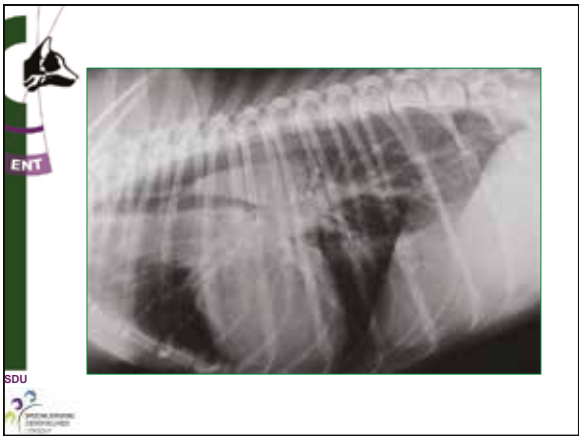
Patient 4

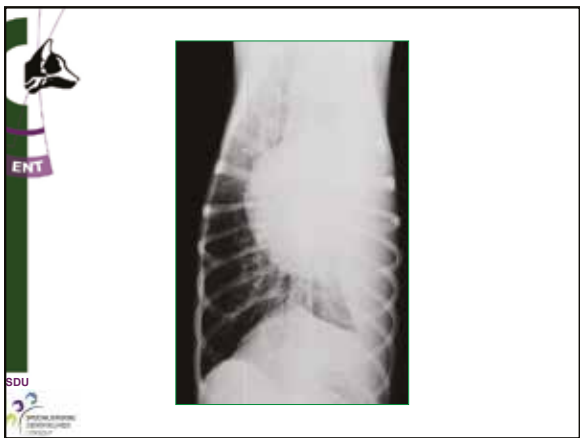
- German Sheperd dog, male, 5 years
- Acute severe dyspnea
- Acute coughing
- After playing outside with sticks and stones
- Work-up
  - Radiography

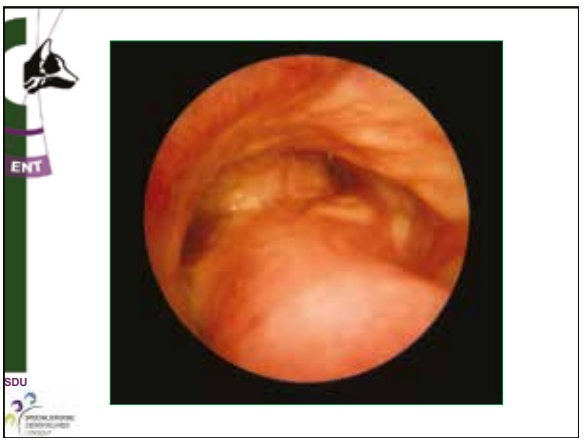


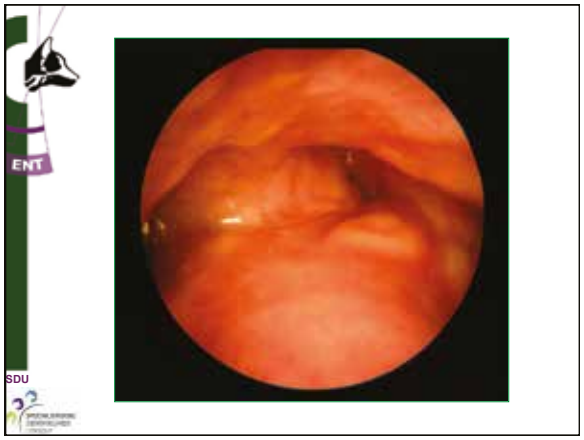
Patient 5

- Mixed-breed dog
- Male, intact, 5 years old
- Dyspnea
- Cough
- Emaciation
- Radiography: process in left lung







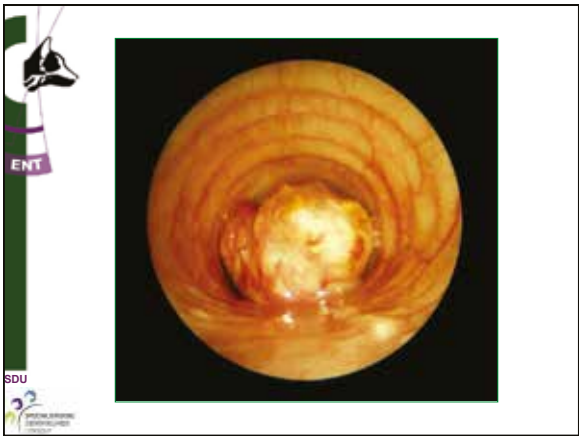


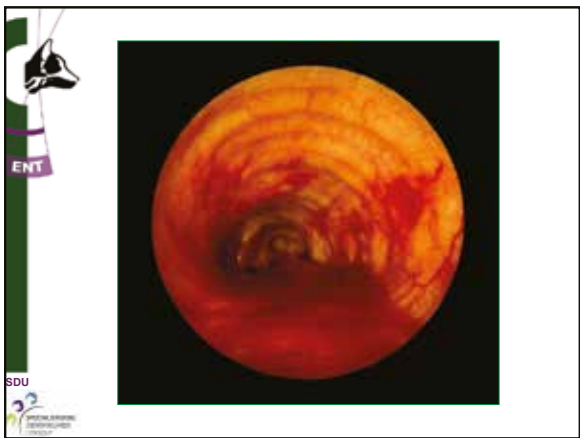
Patient 5

- Tracheobronchoscopy
  - Tumor obstructing entire left main stem bronchus en partially obstructing right bronchus
  - Cytology: malignant lymfoma
  - Therapy: chemotherapy

Tracheal tumors

- Rare
- Signs of luminal obstruction
- Acute or slowly progressive
- Osteochondroma, leiomyoma, osteosarcoma, chondrosarcoma, mastocytoma, malignant lymphoma, adenocarcinoma, squamous cell carcinoma, osteotoma, chondroma and oncocytoma
- Therapy: surgical excision

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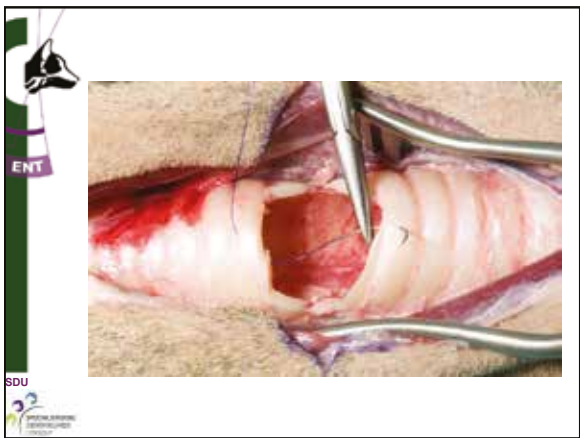
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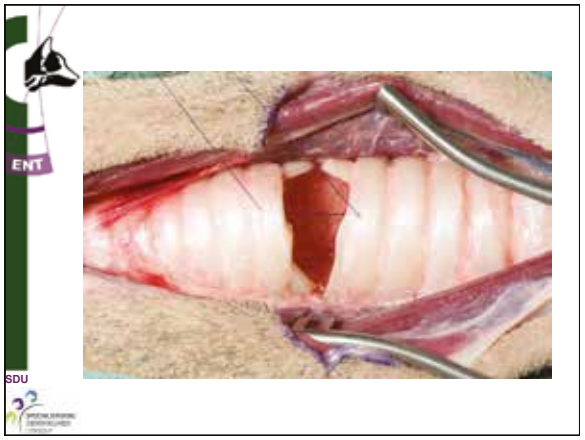
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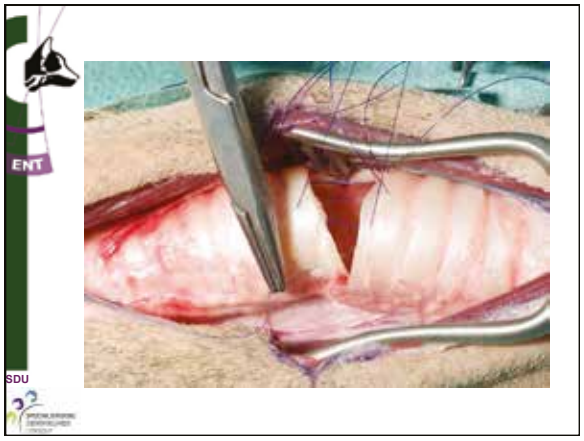
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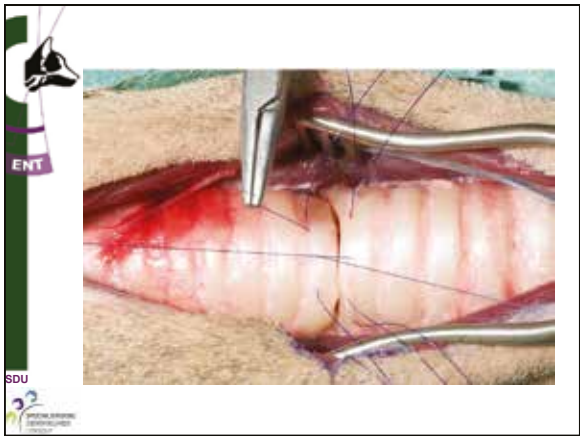
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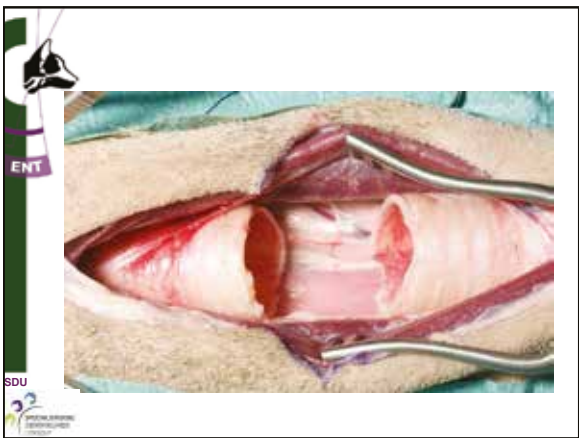
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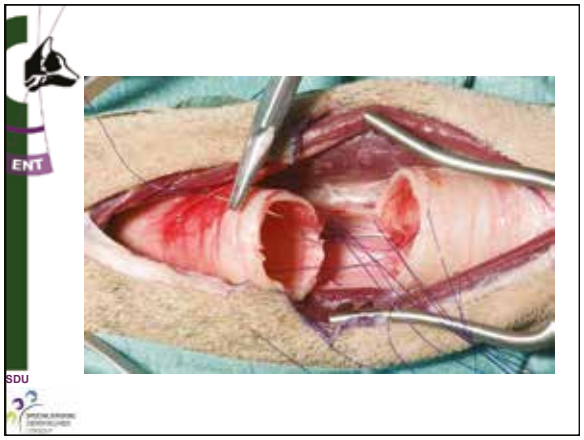
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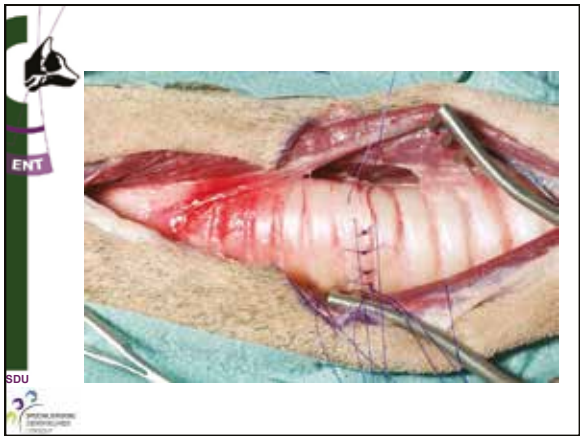
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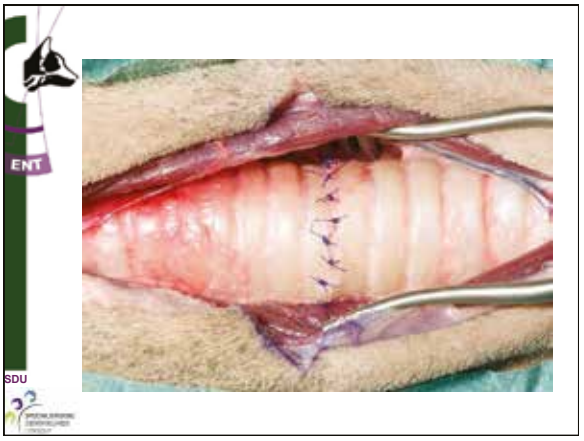
ENT
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Tracheal Collapse

6 year old Yorkshire terrier, female
Coughing
Dyspnea

Tracheal collapse

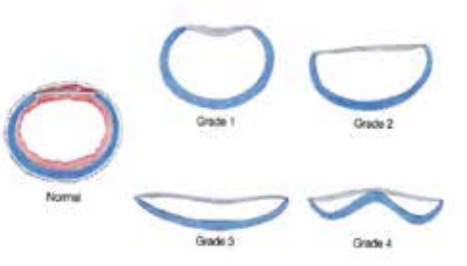
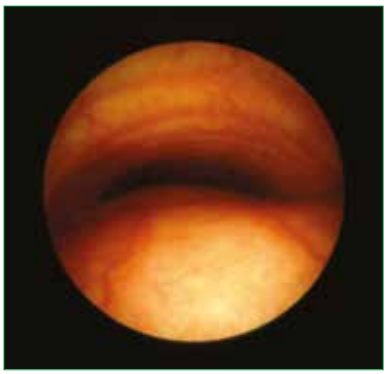


Fig. 4.20. Hedlund & Taboada; Clinical Atlas of ENT diseases in small animals, 2002



Tracheal collapse

- Surgery
  - Recommended for animals with grade II to IV collapse that have failed initial medical management.
  - Total ring tracheoplasty (external ring)
  - Intraluminal stents
  - Plication of trachealis muscle and chrondrotomy are no longer recommended

Tracheal Collapse

Extraluminal ring prosthesis

- Total ring prosthesis first described by Hobson in 1976
- Polypropylene rings can be purchased commercially or premade from syringe cases and sterilized before procedure
- Ventral midline approach to cervical trachea
- Place rings around the trachea, medial to recurrent laryngeal nerves and tracheal vessels

Tracheal Collapse

Extraluminal ring prosthesis

- Secure rings circumferentially with 4-0 monofilament nonabsorbable suture material with 1 or 2 sutures dorsally
- Use tag sutures to mobilize and retract trachea to allow ring placement as distal as thoracic inlet

Surgery tracheal collapse

Surgery tracheal collapse

Tracheal Collapse

Extraluminal ring prosthesis

- Immediate improvement of respiratory function is evident in majority of dogs that escape severe complications
- Laryngeal paralysis
- Tracheal necrosis
- Pneumothorax

Tracheal Collapse

Extraluminal ring prosthesis

- Laryngeal paralysis
  - Occurs in 11 – 30% of operated dogs
  - Identify and preserve the recurrent laryngeal nerves during ring placement
  - Frequent contact of the nerve with prosthetics after surgery?
  - Permanent tracheostomy in 19% of patients
  - Unilateral arytenoid lateralization at time of ring placement recommended by some authors

Tracheal Collapse

Extraluminal ring prosthesis

- Tracheal necrosis
  - Damage to segmental blood supply
  - Modifief technique with skeletonizing only a single pedicle (right side)
  - Or: make a tunnel only where the ring is passed around the trachea, take care to preserve the recurrent nerves
- Pneumothorax
  - Dissection of distal cervical trachea

Questions?

Diagnosis and treatment of diseases of the external ear canal – the surgeon's view

Gert ter Haar DVM, PhD, MRCVS, DECVS

SPECIALISTISCHE DIERENKLINIEK UTRECHT

Otorhinolaryngology

ENT

Diagnosis & treatment of diseases of the external ear canal - surgeons point of view

Gert ter Haar, DVM, PhD, DECVS

European Specialist in Small Animal Surgery

Ear, Nose & Throat, Brachycephaly related and Audiologic Diseases

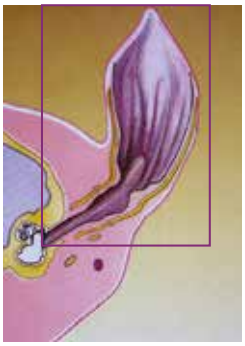
Oncologic and Reconstructive Surgery

AniCura

Introduction

ENT

- Anatomy
  - Outer Ear
  - Middle Ear
  - Inner Ear
- Physiology



SDU

Introduction

ENT

- Anatomy
  - Outer Ear
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SDU

Introduction

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- Anatomy
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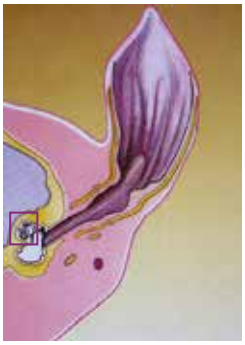


SDU

Introduction

ENT

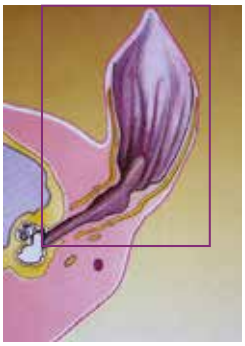
- Auricle
  - Ear laceration
  - Othaematoma + bandaging technique
  - Pinna amputation
- Ear canal
  - Lateral wall resection (Zepp, LaCroix)
  - Vertical Ear canal resection
  - Total Ear canal resection
- Middle Ear Cavity
  - Lateral bulla osteotomy

SDU

Anatomy

ENT

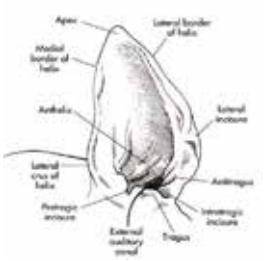
- Pinna (Auricle)
- External Ear Canal
  - Auricular cartilage
    - Pinna
    - Vertical canal
  - Annular cartilage
    - Horizontal canal
- Tympanum
- SKIN!!!!

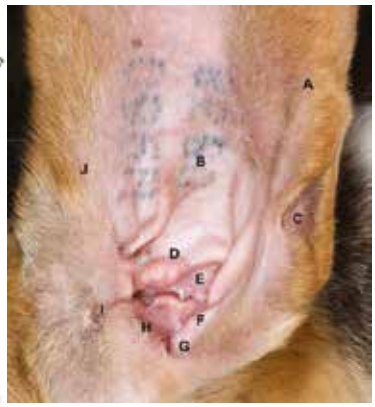


SDU

Anatomy

- Pinna
  - Concave/convex
- Tragus
- Anthelix
- Antitragus
- Scapha

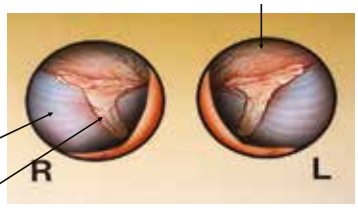




Anatomical landmarks.
A = Lateral border of helix.
B = Scapha.
C = Marginal pouch.
D = Antihelix.
E = Concha.
F = Antitragus.
G = Intertragic incisure.
H = Tragus.
I = Tragohelicine incisure.
J = Medial border of helix.

Anatomy

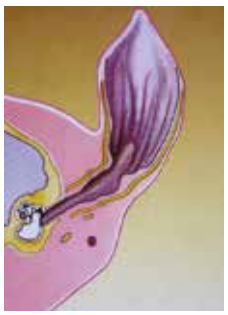
Pars flaccida

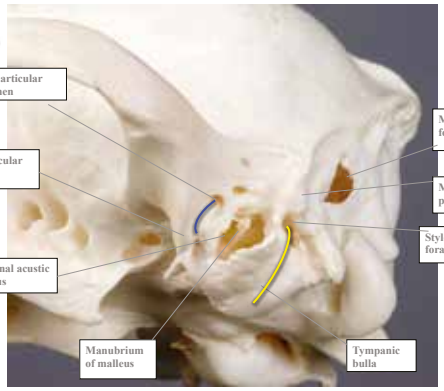


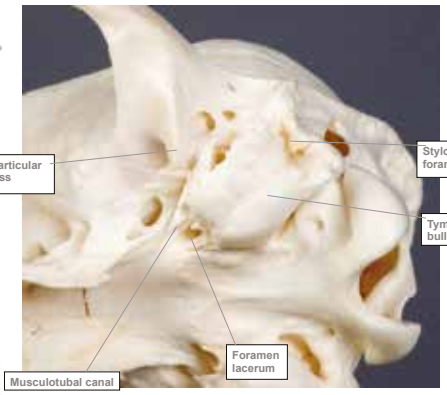
Pars tensa

Malleus

Anatomy







Anatomy

- Middle Ear
  - Tympanum
  - Malleus, incus and stapes
  - Eustachian tube
  - Air filled, respiratory epithelium
  - Sympathic nerves/Facial nerve

Anatomy Middle Ear

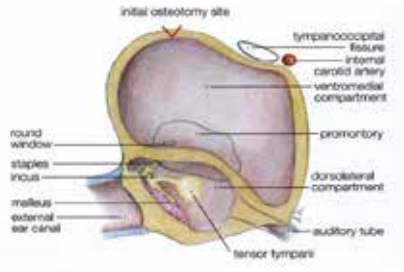
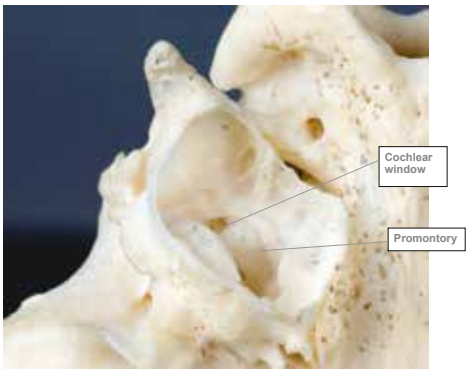
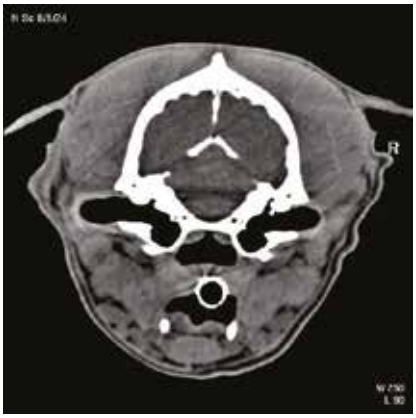


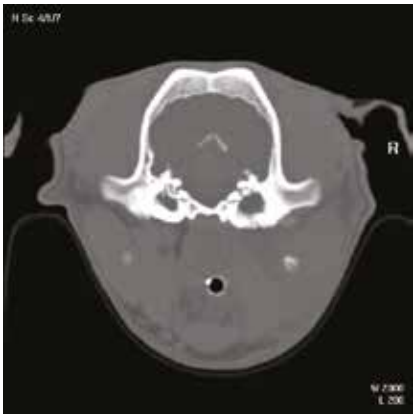
Fig. 1.61A: Hedlund & Taboada, Clinical Atlas of ENT diseases in Small Animals, 2002



Anatomy Inner Ear

- Inner Ear
  - Cochlea
    - Scala tympani
    - Scala media
    - Scala vestibuli
  - Vestibulum
    - Sacculus
    - Utriculus
    - Semicircular canals





Traumatic wounds 1

- Etiopathogenesis: Fighting
- Symptoms: tear with bloody edges
- Diagnosis: on symptoms
- Therapy: because of blood loss; immediate suturing!

Traumatic wounds 2

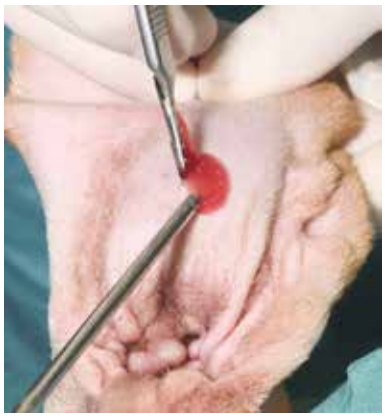
- Therapy: Suture skin on both sides with Monocryl, start at pinnal edge, don't perforate the cartilage.
- Postoperative: Collar, analgesics, AB

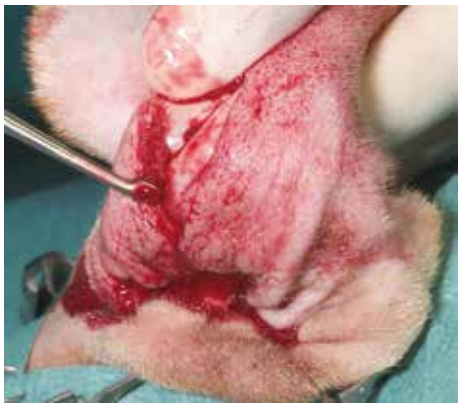
Othaematoma 1

- Etiopathogenesis:
  - Scratching and head shaking
  - Fracture auricular cartilage
  - Fibrin deposition; fibrosis and contraction
- Symptoms:
  - Fluctuating, non painfull thickness on concave side; Otitis externa
- Therapy: Surgery!

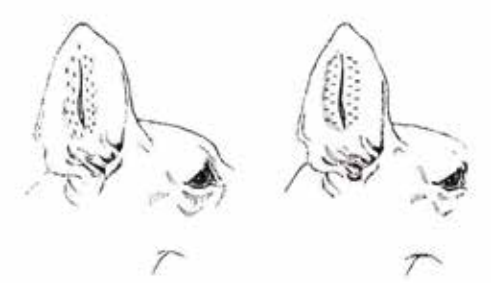
Othaematoma 2

- Surgery
  - S-shaped incision concave side pinna
  - Remove clots and fibrin
  - Flush
  - Close cavity with full-thickness sutures (Monocryl 2-0)
  - Protective bandage
  - Collar, AB and analgesics



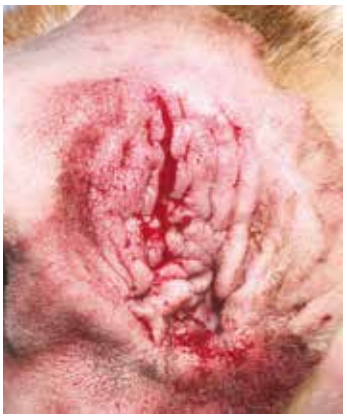


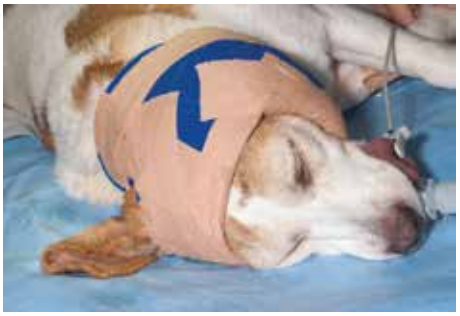
Othaematoma 6



GOOD *FALSE*

Slatter; Textbook of Small Animal Surgery, 1993





Othaematoma 3

- **Complications**
  - **Recurrence**
  - **Cauliflower ear (cartilage degeneration and ossification)**
  - **Abscessation**
    - **Pinna amputation**

Auricular Diseases

- Tumor
  - Dog
    - Sebaceous gland carcinoma
    - Histiocytoma
    - Mast cell tumor
  - Cat
    - Squamous cell carcinoma (mimics granulomatous inflamm.)
    - Basal cell carcinoma
    - Hemangiosarcoma
    - Malignant melanoma

Total Ear Canal Ablation

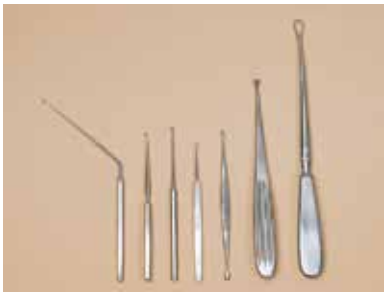
- Indications
  - Chronic proliferative otitis externa
  - Non-responsive otitis externa and media
  - Otitis media and interna in brachy dog
  - Para-aural abscessation
  - Cholesteatoma
  - Tumor

Ear Surgery

- Pre-operative antibiotics & analgesia
  - Amoxicillin/clavulonic acid, enrofloxacine
  - NSAID´s AND opioid analgesics
  - Intravenous corticosteroids (?!)
  - Eye lube!!!!
- Surgical preparation
  - Beware of ototoxicity (desinfectants)
  - Positioning!

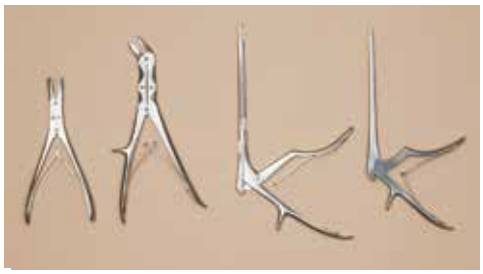
Ear Surgery; Instruments

Ear Surgery; Instruments



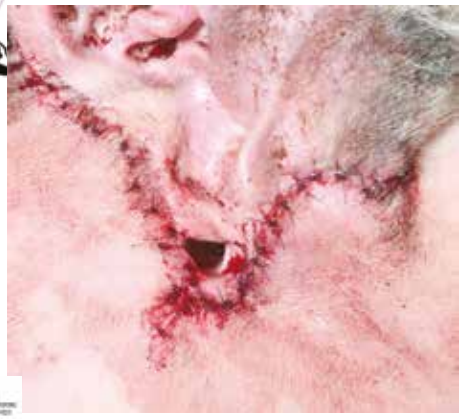
Ear Surgery; Instruments

Ear Surgery; Instruments



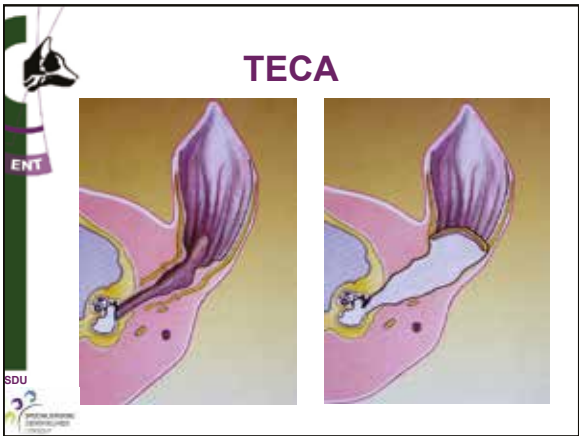
Lateral wall resection (Zepp)

- **Indications:**
  - **Local benign tumor of lateral wall vertical ear canal**
  - **Malignant tumor in skin/subcutis overlying the vertical ear canal**
  - **NOT FOR OTITIS EXTERNA!!!**



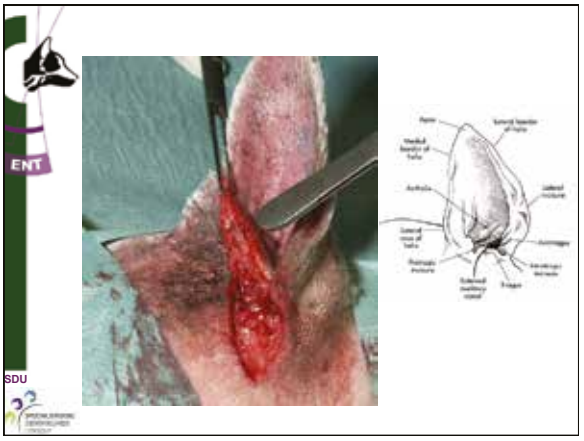
Vertical Ear Canal Ablation

- **Indications:**
  - **Tumor limited to vertical ear canal**
  - **After avulsion of ear canal (separation of horizontal part from vertical part of ear canal)**
  - **NOT FOR OTITIS EXTERNA!!!**



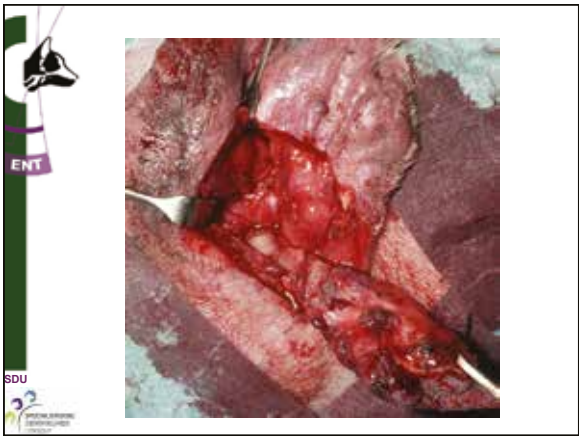
Total Ear Canal Ablation

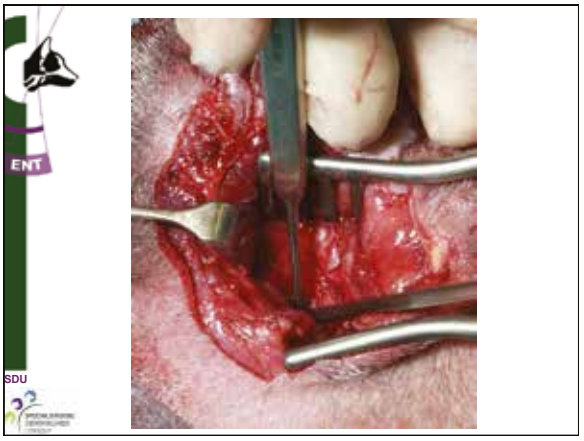
- Technique
  - V-shaped incision in skin from tragus
  - Dissect skin flap towards basis
  - Dissect sharply and bluntly to ear canal and free ear canal cranial and caudal
  - Use scissors to free ear canal from pinna

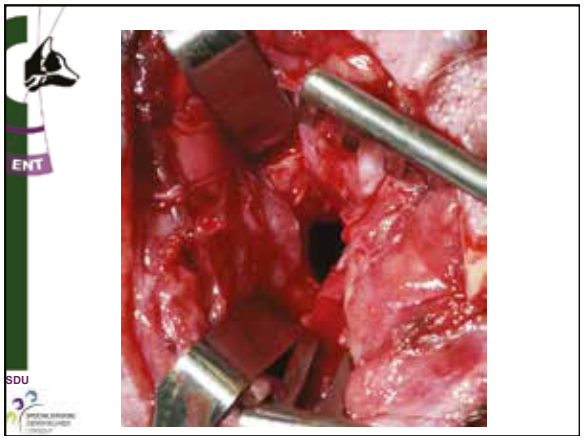


Total Ear Canal Ablation

- Technique
  - Dissect sharply ear canal towards bony meatus acusticus, be careful not to damage facial nerve
  - Remove ear canal from meatus
  - Remove all epithelial remnants with rongeurs, sharp scissors, curette & flushing
  - Close with Penrose-drain

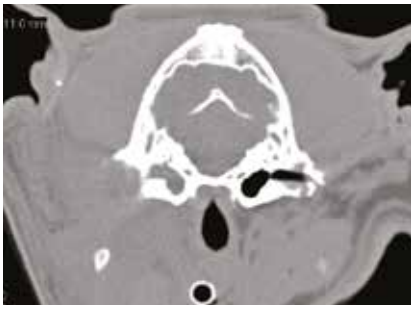






Total Ear Canal Ablation

- Complications
  - Wound infection
  - Facial nerve paralysis
  - Fistulation
    - Otitis media
    - Epithelial remnants



Para-aural abscessation

- Etiopathogenesis: avulsion ear canal, bite- and scratchwounds, complication of otitis media/ externa, after ear surgery
- Symptoms: sick animal with painful and fluctuating process/swelling around base ear
- Diagnosis: symptoms, FNAB, CT-scan, otoscopy
- Therapy: Drainage, TECA+LBO

Questions?

Otitis media and middle ear effusion

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SPECIALISTISCHE DIERENKLINIEK UTRECHT

Otorhinolaryngology

ENT

Otitis Media & Middle Ear Effusion (OME) in Dogs

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AniCura

Otitis Media Introduction

ENT

- **Etiopathogenesis:**
  - Chronic otitis externa (secondary otitis media)
  - Previous surgery (TECA/LBO)
  - Cholesteatoma
  - Brachycephaly (primary otitis media)
  - Primary secretory otitis media in CKCS

SDU

Gotthelf, L. N. (2004). Diagnosis and treatment of otitis media in dogs and cats. Veterinary Clinics of North America, Small Animal Practice, 34(2), 469–487.

Secondary Otitis Media Dog

ENT

- 16% acute OE, >50% chronic OE
- Complex multifactorial etiopathogenesis
  - L-shaped form ear canal, accumulation of exsudate with proteolytic enzymes against thinnest part eardrum
  - Ulceration of skin leads to leakage of serum with liberation of bacterial proteases, collagenases, elastases, and lysozymes from phagocytic cells as well as epidermal maceration

SDU

Gotthelf, L. N. (2004). Diagnosis and treatment of otitis media in dogs and cats. Veterinary Clinics of North America, Small Animal Practice, 34(2), 469–487.

Otitis Media Dog

ENT

- **Clinical Signs**
  - Otalgia (otic pain)
  - Facial paralysis
  - Horner's syndrome
  - Painful mastication
  - General illness
  - Deafness/vestibular disease
  - Otitis externa!!!!

SDU

Diagnosis

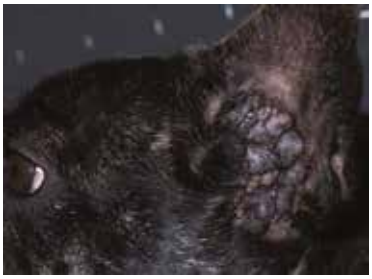
ENT

- History and clinical signs
- General, neurologic & ear examination
- Diagnostic imaging
  - Radiography
  - CT-scan, MRI
- Culture, cytology, histopathology (myringotomy)
- Hearing assessment (BERA)

SDU

Proliferative OE

ENT



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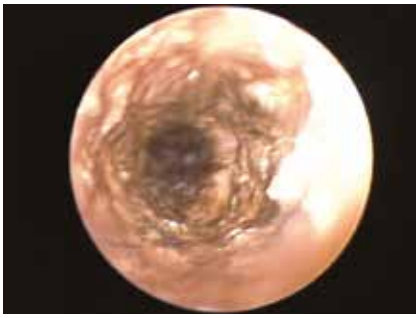
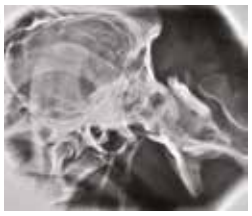
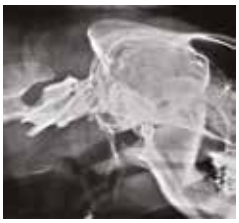
Otoscopy

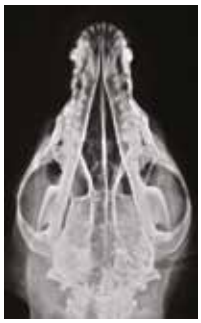


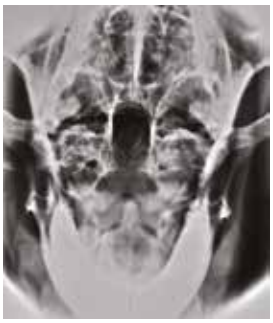
Radiography

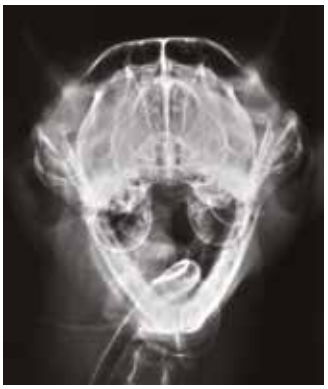
- Positioning! Anaesthesia!
- Dorsoventral, lateral oblique en rostrocaudal view (open mouth)
- Breed and individual variation
- Increased soft tissue opacity and sclerosis or lysis of the bulla wall
- Not very sensitive!







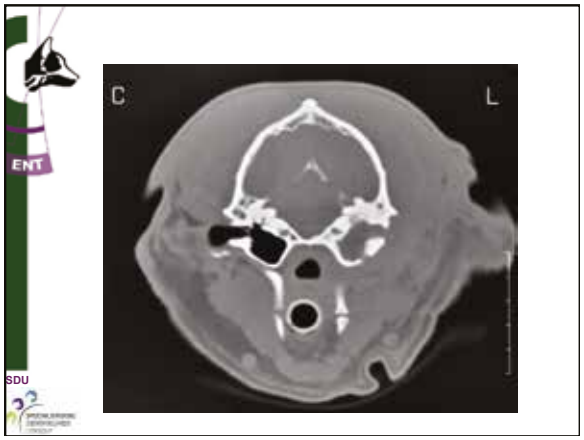




CT-scan/MRI

- Expensive! Anaesthesia!
- CT gives better resolution of bony lesions
- MRI gives better resolution of soft tissue lesions
- More sensitive than radiology, yet still not specific for otitis media or neoplasia

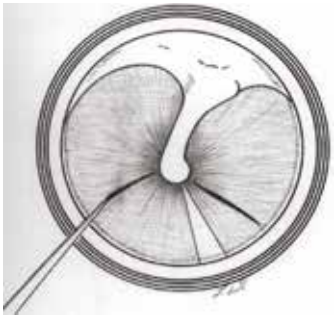
Pre-operatively!



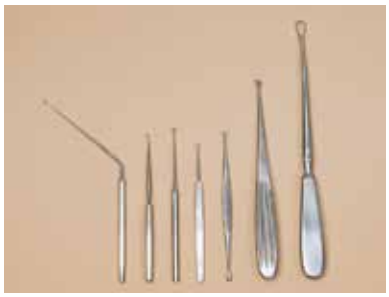
Myringotomy

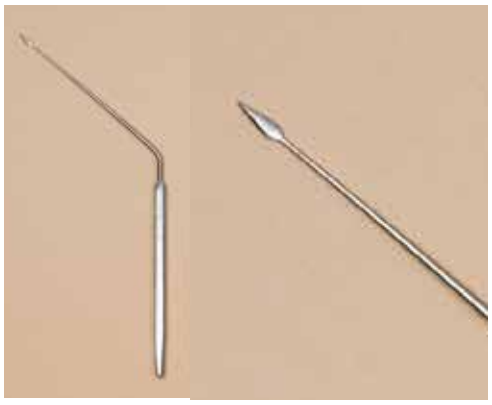
- For draining fluid
  - Obtain material for culture and cytology
  - Relieve pressure
  - Instill medication
- Method
  - Curvilinear
  - Radial
  - paracentesis

Myringotomy location



Ear Surgery; Instruments



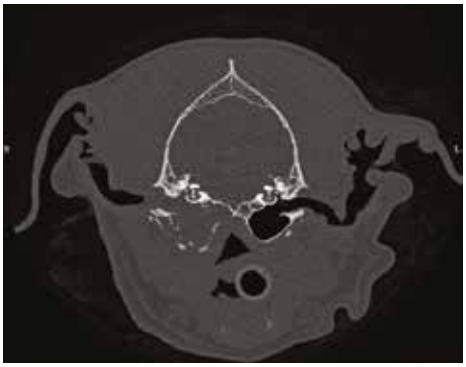


Ear Surgery; Instruments

Otitis media

- Therapy:
  - Broadspectrum antibiotics 4-6 weeks
  - Flushing ear canals and middle ear
  - No ototoxic topical medications
  - Tympanotomy
  - Treat otitis externa
  - Surgery (TECALBO)

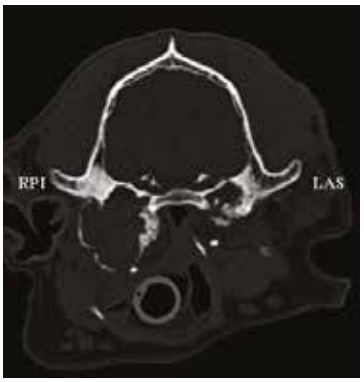
Palmeiro, B. S., Morris, D. O., Wiemelt, S. P., & Shofer, F. S. (2004). Evaluation of outcome of otitis media after lavage of the tympanic bulla and long-term antimicrobial drug treatment in dogs: 44 cases (1998-2002). J Am Vet Med Assoc, 225(4), 548–553.



Aural Cholesteatoma

- Epidermoid cyst forming within middle ear
- Keratin debris surrounded by keratinizing stratified squamous epithelium
- Congenital or acquired
  - Retraction of tympanic membrane
  - Migration of squamous epithelium through a tympanic membrane perforation
  - Slow expansion, severe inflammatory response
  - Bone lysis and bone formation, bulla expansion!

Hardie A.M, Linder K.E., Pease A.P. Aural Cholesteatoma in Twenty Dogs. Vet Surg 2008,37:763-770

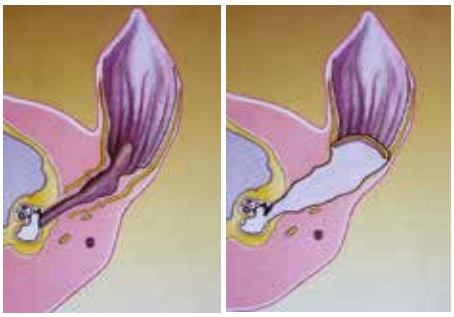


Aural Cholesteatoma

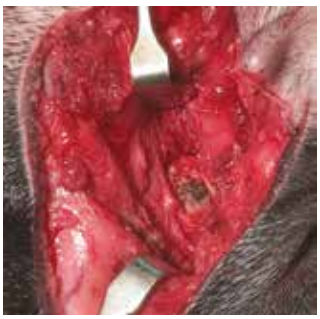
- Treatment
  - TECALBO
  - VBO
- About 50% need medical management after surgery
- Risk factors for recurrence after surgery
  - Inability to open mouth
  - Neurologic signs on admission
  - Lysis of temporal bone on CT scan

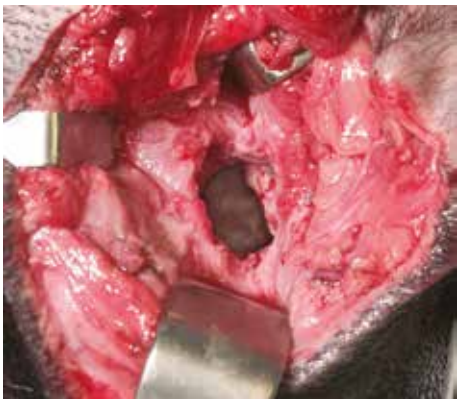
Hardie A.M, Linder K.E., Pease A.P. Aural Cholesteatoma in Twenty Dogs. Vet Surg 2008,37:763-770

TECA



Lateral Bulla Osteotomy



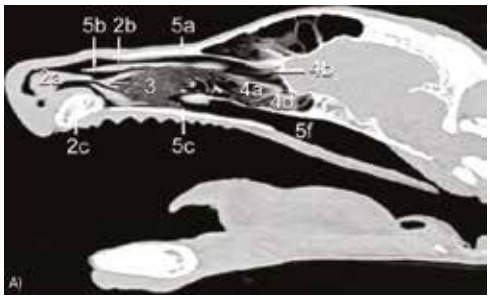


Primary Otitis Media Dog

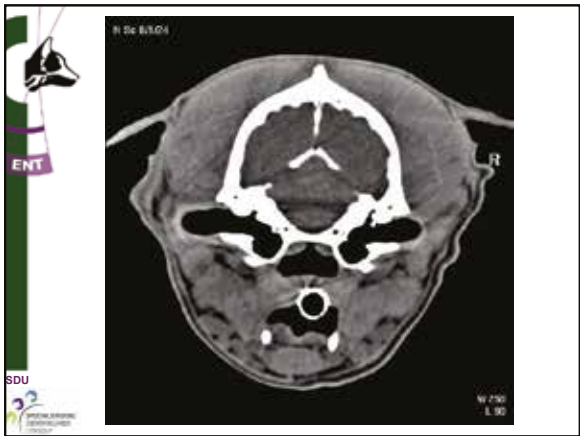
- **Brachycephalic Dogs**
  - **French Bulldog, Pug**
  - **Reduced bony skull size, abundant soft tissues**
  - **Pharyngeal abnormalities**
  - **Soft palate muscle abnormalities**
  - **Eustachian tube dysfunction**
- **PSOM/OME Cavalier King Charles Spaniel**

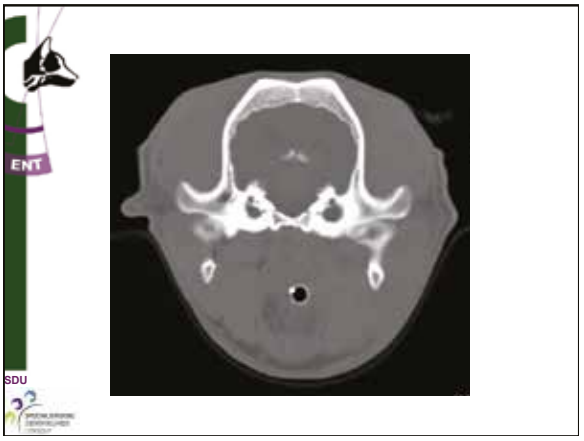
Hayes, G. M., Friend, E. J., & Jeffery, N. D. (2010). Relationship between pharyngeal conformation and otitis media with effusion in Cavalier King Charles spaniels. The Veterinary Record, 167(2), 55–58.
Oechtering, G. U. (2010). Brachycephalic syndrome - new information on an old congenital disease. Veterinary Focus, 20(2), 2–9.
Bluestone, C. D., & Swarts, J. D. (2010). Human evolutionary history: Consequences for the pathogenesis of otitis media. Otolaryngology--head and neck surgery; 143(6), 739–744.

CT-scan skull



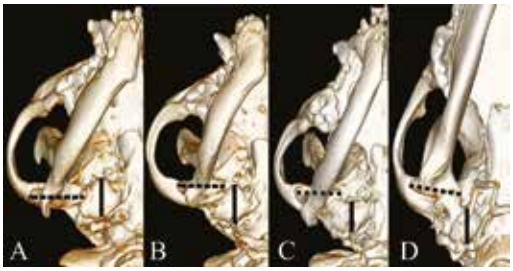
http://www.brachycephalia.com/info/521.pdf (Oechtering, Leipzig)





BOS History/Signalment

CT-scan bullae



CT bullae

- Study in 100 brachycephalics
  - CKCS significantly flatter tympanic bulla
  - French Bulldog and Pug had significantly more rostrally located tympanic bulla
  - Fluid was present in 47% of brachycephalics, but no association with TB shape & position
  - Presentation for BOAS was associated with a significantly higher incidence of effusion

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Mielke, B., Lam, R., & Haar, ter, G. Tympanic Bulla Morphology; Computed Tomographic Evaluation Of Tympanic Bulla Shape And Position In Brachycephalic Breeds. *Veterinary Radiology & Ultrasound 2016, in submission.*

Primary Secretory Otitis Media

- Cavalier King Charles Spaniel
  - Deafness
  - Nervous symptoms
    - Syringomyelia, HNP, PSOM

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Cole, L. K. (2012). Primary Secretory Otitis Media in Cavalier King Charles Spaniels. The Veterinary clinics of North America.Small animal practice, 42(6), 1137–1142.
Stern-Bertholtz, W., Sjostrom, L., & Hakanson, N. W. (2003). Primary secretory otitis media in the Cavalier King Charles spaniel: a review of 61 cases. The Journal of small animal practice, 44(6), 253–256.
Rusbridge, C. (2004). Primary secretory otitis media in Cavalier King Charles spaniels. The Journal of small animal practice, 45(4), 222

Etiopathogenesis PSOM

- PSOM is uncommon
- Etiopathogenesis unknown
- Predominantly CKC Spaniel
- Hypothesis
  - Dysfunction Eustachian tube
  - Increased production mucus

Etiopathogenesis PSOM

- PSOM is uncommon
- Etiopathogenesis unknown
- Predominantly CKC Spaniel
- Hypothesis
  - Dysfunction Eustachiun tube
  - Increased production mucus

Symptoms

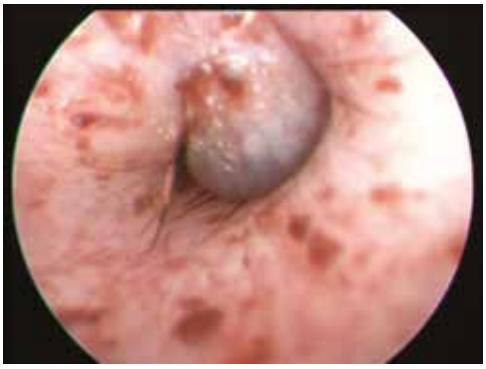
- Head and/or neck pain with episodes of yelping or crying
- Signs of otitis externa
  - Scratching, head shaking, otalgia, otic discharge
- Impaired hearing
- ! Cervical disk disease, syringomyelia!

Diagnosis

- Thorough and complete clinical examination!
  - General physical
  - Dermatologic
  - Neurologic (complete!)
  - Ear exam

Clinical Examination Ear

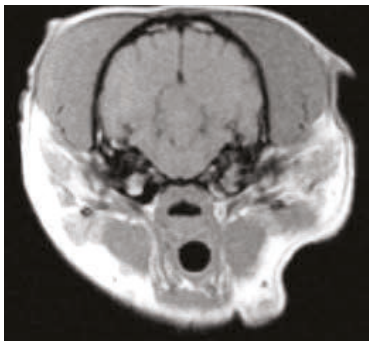
- Examination Ear
  - Hearing assessment
  - Inspection and palpation pinna and external ear canal
  - Otoscopy



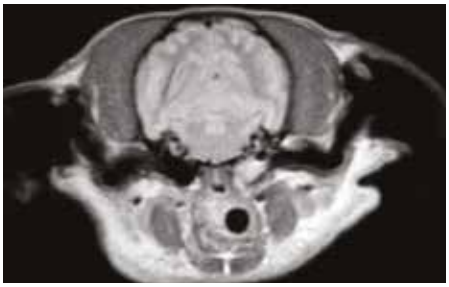
Clinical Examination Ear

- Examination Ear
  - Hearing assessment
  - Inspection and palpation pinna and external ear canal
  - Otoscopy (Flush!)
  - Diagnostic Imaging
    - CT versus MRI
  - Tympanotomy

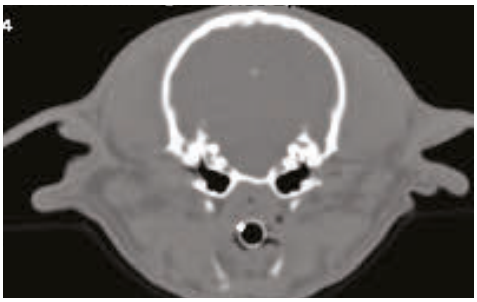
MRI CKSP PSOM bilateral



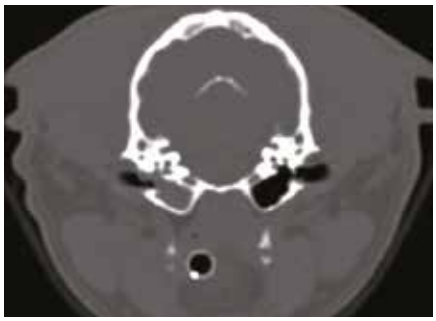
MRI CKSP PSOM unilateral



CT CKCS Normal



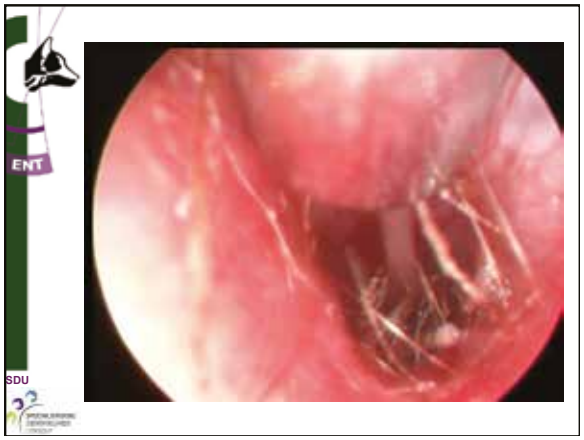
CT CKCS PSOM



Therapy

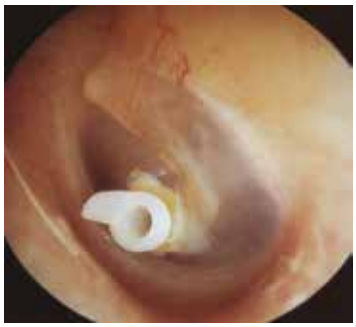
- Manual removal of mucoid effusion through myringotomy (1-5 times!)
- Topical or systemic corticosteroids
- Topical or systemic antibiotics
- Tympanostomy tubes

Corfield, G. S., Burrows, A. K., Imani, P., & Bryden, S. L. (2008). The method of application and short term results of tympanostomy tubes for the treatment of primary secretory otitis media in three Cavalier King Charles Spaniel dogs. Australian Veterinary Journal, 86(3), 88–94.



Ventilation tubes

Otitis Media Child



Therapy

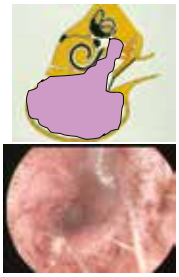
- Insertion
  - Endoscopy guided (operating microscope)

Otitis Media CKCS

Guerin, V., Hampel, R., & Haar, ter, G. (2015). Video-otoscopy-guided tympanostomy tube placement for treatment of middle ear effusion. Journal of Small Animal Practice.

Discussion

- PSOM in CKCS
  - Etiopathogenesis
  - Symptoms
  - Diagnostic Work-up
- Therapy
  - Myringotomy
  - Ventilation Tube




Questions?
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Diagnostic workup of patients with nasal disease

Gert ter Haar DVM, PhD, MRCVS, DECVS

SPECIALISTISCHE DIERENKLINIEK UTRECHT

Otorhinolaryngology

ENT

Diagnostic Work-up of Patients with Nasal Disease

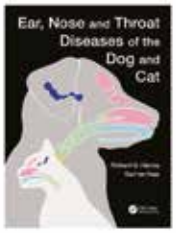
Gert ter Haar, DVM, PhD, DECVS
European Specialist in Small Animal Surgery
Ear, Nose & Throat, Brachycephaly related and Audiologic Diseases
Oncologic and Reconstructive Surgery
President of the IVENTA

AniCura

Introduction

ENT

- Nose, nasal cavity & sinuses
  - Diagnostic Procedures
    - Radiography
    - CT-scan/MRI
    - Endoscopy
  - Patients with diseases of nose
    - •Surgical treatment of nasal diseases



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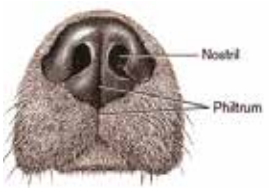
Anatomy & Physiology 1

ENT

- Nasal plane
  - Thick epithelium
  - Wet
  - Pigmentation
  - Nostril



Evans H.E. and de Lahunta A. Miller's Anatomy of the DOG, 4th edition, 2013; The Respiratory System.

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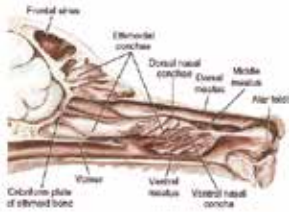
Anatomy & Physiology 3

ENT

- Neuroepithelium
- Conchae
  - Maxilloturbinates
  - Dorsal nasoturbinates
  - Ethmoturbinates
- Meatus



Evans H.E. and de Lahunta A. Miller's Anatomy of the DOG, 4th edition, 2013; The Respiratory System.

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Diagnostic Procedures 1

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- History!
  - Symptoms
    - Bilateral vs unilateral discharge (epistaxis)
    - Sneezing/reverse sneezing
    - Gagging/retching
    - Dyspnea (STRIDOR!)
    - Dysfagia
    - Systemic illness

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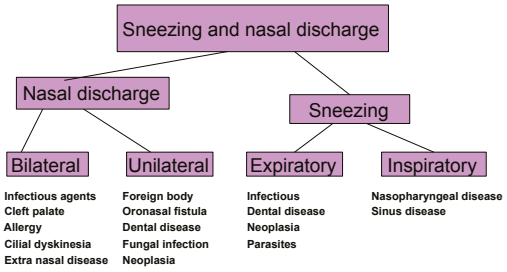
Diagnostic Procedures 2

ENT



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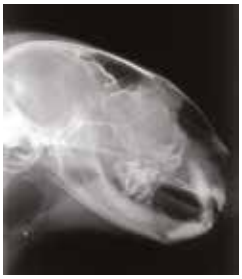
Diagnostic Procedures 3

- Clinical examination
  - Nasal plane (tumor, inflammation, depigmentation)
  - Air passage
  - Pain
  - Oral mucosa (dental problems)
  - Systemic abnormalities

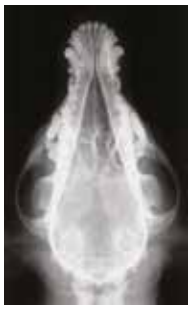
Diagnostic Procedures 4

- Radiography
- Lateral view
  - Frontal sinus
  - Nasopharynx



Diagnostic Procedures 5

- Radiography
- Dorsoventral view
  - Caudal nose
  - Foreign bodies

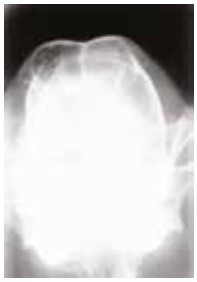


Diagnostic Procedures 6

- Radiography
- Intra-oral view
  - Most informative
  - Intranasal disease

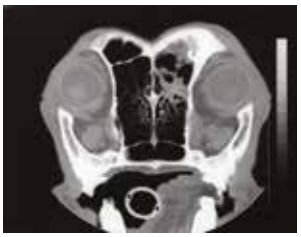
Diagnostic Procedures 7

- Radiography
- Frontal view
  - Frontal sinus



Diagnostic Procedures 8

- CT-scan/MRI
  - Expensive
  - Anaesthesia
  - Detailed info



Diagnostic Procedures 10

- Endoscopy
  - Inspection of mouth and throat under general anaesthesia with dental probing
  - Rigid endoscopy
    - Nasal sinuses
  - Flexible endoscopy
    - Nasopharynx

Diagnostic Procedures 11

- Rhinoscopy
  - Otoscope
  - Rigid scope

Diagnostic Procedures 12

- Rhinoscopy
  - Otoscope
  - Rigid scope
    - 2.7/3.5 mm
    - 15 cm
    - 25 °-vision

Diagnostic Procedures 13

- Rhinoscopy
  - Otoscope
  - Rigid scope
    - Camera
    - Recorder
    - Monitor
    - Light source

Diagnostic Procedures 14

- Rhinoscopy
  - Otoscope
  - Rigid scope
    - Hartmann forceps
    - Biopsy forceps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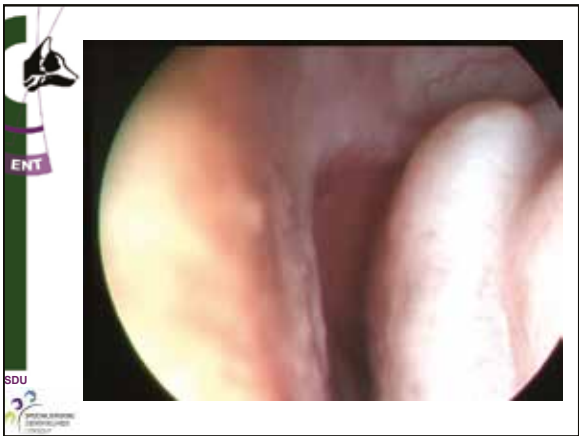
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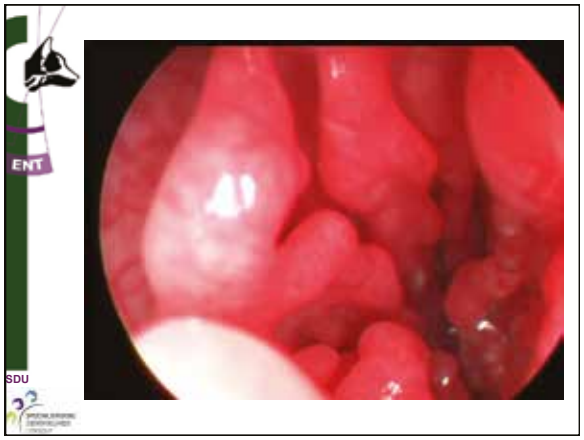
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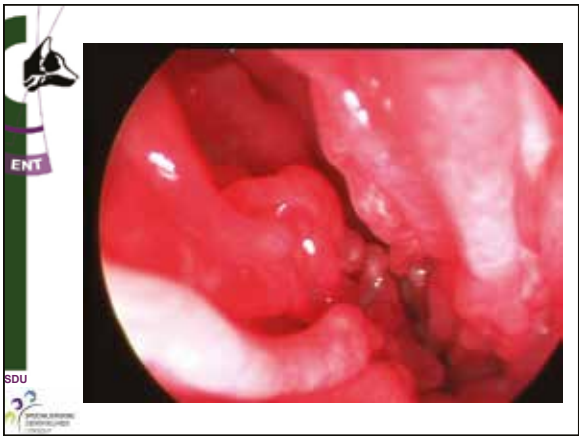
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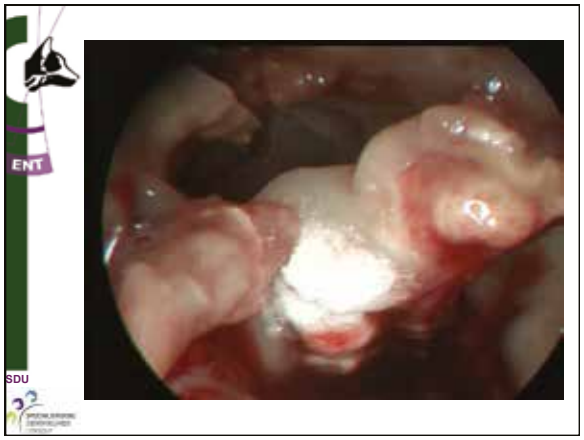
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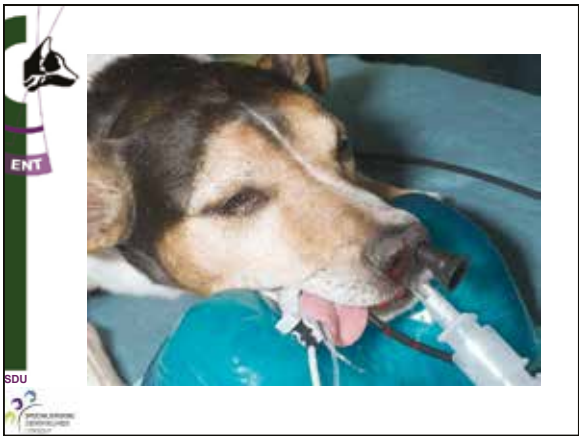
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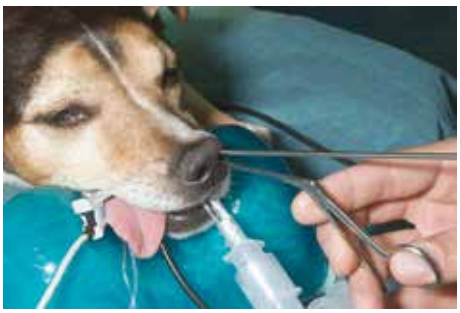


Diagnostic Procedures 20

- Haematology
- Urinary Analysis
- Culture
- Cytology
- Histology
- Surgical exploration

Diagnostic Procedures 21

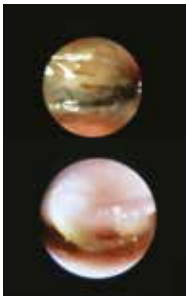
- Radiography, rhinoscopy and oral examination require general anesthesia!
- Proper planning
- Diagnosis and Therapy when possible

Patient 1

- Jack Russell, ♀, 2 years
- Since 2 weeks acute sneezing, now leftsided nasal discharge
- Physical examination no abnormalities
- Most likely diagnosis?

Patient 1

- Additional examination
  - Rhinoscopy
  - Radiology
  - Oral examination / nasopharyngoscopy



Patient 1

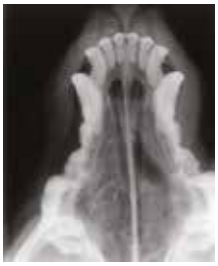
- Diagnosis: foreign body
- Therapy:
  - Rhinoscopic removal
  - Surgical removal
- Prognosis: good

Patient 2

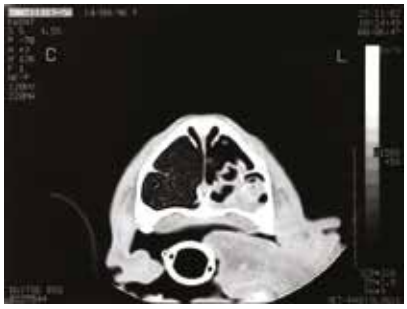
- Golden Retr, ♂, 4 years
- Left-sided nasal discharge of 3 month duration with occasional blood loss, sneezing
- PE: Depigmentation nasal plane, normal air passage
- Most likely diagnosis?

Patient 2

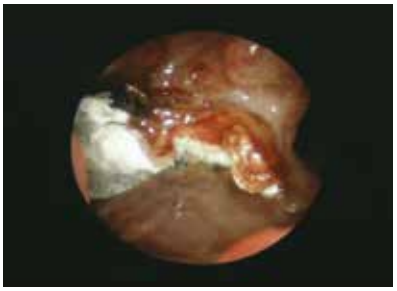
- Additional examination
  - Radiography skull
  - Rhinoscopy
  - Oral examination



Patient 2



Patient 2



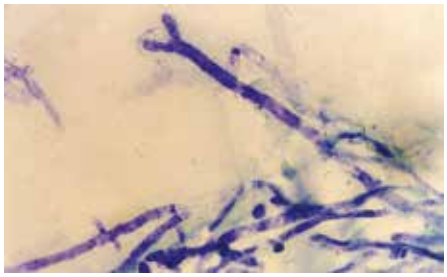
Aspergillosis

- Common in dogs
- Aspergillus and Penicillium species
- Rare in brachycephalic breeds, often in Golden Retriever and in Rottweiler dogs
- All ages, but rare in dogs < 1 year
- After exposure to large fungal inocula and/or decreased local resistance.
- Normal immunocompetent animals

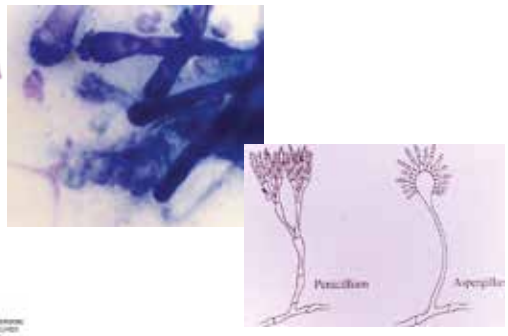
Aspergillosis

- Diagnosis:
  - History, signalement and clinical signs
  - Radiography; turbinate destruction, areas of increased and decreased radiolucency
  - Rhinoscopy: cavernous areas with fungal hyphae
  - Culture, serology (ELISA), histopathology
  - Trephination of the frontal sinus

Cytology 1



Cytology 2

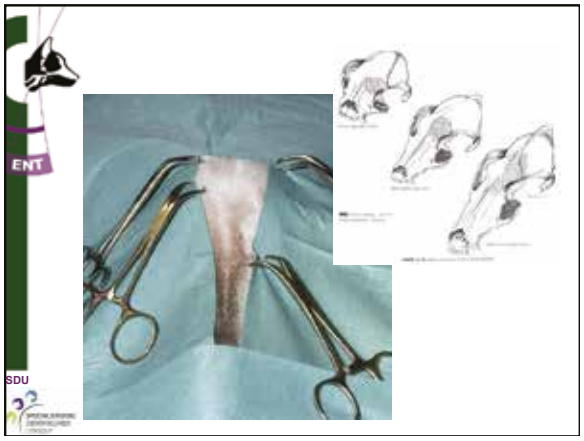


Aspergillosis

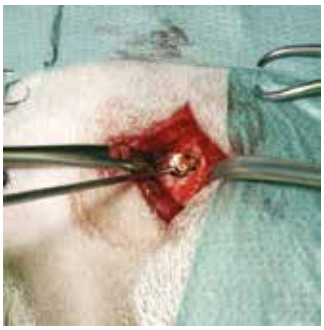
- Therapy:
  - ORAL: Thiabendazole, ketoconazole, itraconazole and flubendazole; expensive, side-effects, recurrence 50%
  - TOPICAL: Amphotericine B, lugol, clotrimazole and enilconazole; more invasive, expensive, less side-effects, recurrence 10-30%

Aspergillosis

- Therapy
  - Trephination SN + SF unilateral or bilateral
  - Tube placement
  - 2 dd instillation of 10 mg/kg enilconazole (Imaverol, Janssen Pharmaceutics), 1 : 10 diluted with sterile water for 14 days
  - Success rate of 87-92%



Enilconazole



Enilconazole

Clotrimazole – 1 hour infusion

- ECVIM meeting 1990 (Davidson)
- Journal of Veterinary Surgery
  - 1995 (Matthews)
  - 1996
- Now more and more reports (Sharp)

Clotrimazole – 1 hour infusion

- 1% solution of clotrimazole in polyethylene glycol
- Foley catheters occlude
  - Nasopharynx (with gauze sponges)
  - Nostrils
- Catheters for deliverance of drug in dorsal nasal meatus
- Rotation of dog every 15 minutes

Clotrimazole – 1 hour infusion

- Complications
  - Cribriform plate / meningoencephalitis
    - Diagnostic imaging!
  - Pharyngitis/laryngitis

Clotrimazole - 1 hour infusion

Clotrimazole - 1 hour infusion

Clotrimazole - 1 hour infusion

Conclusions

- **1st choice therapy:** clotrimazole soak and perendoscopic frontal sinus filling with cream
  - –Control rhinoscopy after 4 weeks
  - –2nd therapy if indicated
- **2nd choice therapy:** enilconazole after trephination (classic treatment)
- **Extranasal disease:** systemic antifungal therapy (flubendazole, voriconazole)

Patient 6

- Yorkshire Terrier, 12 years
- Right-sided mucopurulent nasal discharge, diminished appetite
- PE: peridontitis, plaque and calculus
- Most likely diagnosis?

Patient 6

- Additional examination
  - – Oral examination with dental probing
  - – Radiography of skull
  - – Rhinoscopy

- Diagnosis: Oronasal fistula

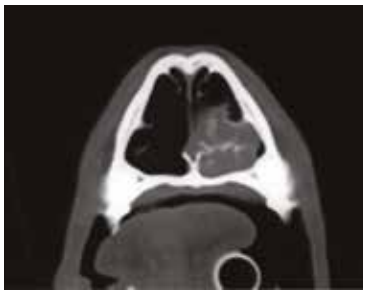
Patient 4

- Collie, ♀, 8 years
- Sneezing, right-sided nasal discharge for 3 months, with blood
- PE: No depigmentation, no air passage on the right
- Most likely diagnosis?

Patient 4

- Additional examination
  - Radiography skull/thorax
  - Rhinoscopy (RAB)
  - Oral examination

Patient 4



Nasal tumors

- Dog: 2/3 epithelial, ± 1/3 mesenchymal, some neuroendocrine
- Locally aggressive, metastasize late, micro-metastases have been reported however and can remain subclinically for 12-36 months
- Diagnosis: radiography/CT/MRI, RAB

Nasal tumors

- Radiotherapy:
  - Median survival of 8-36 months
- Systemic chemotherapy:
  - Cisplatine or cis-diamminedichloroplatinum II, 60 mg/m² iv every 4 weeks (palliative)
  - Malignant lymphoma: combination chemotherapy
- Median survival without therapy is usually < 6 months, surgery alone does not prolong survival

Questions?

Diagnostic workup of patients with nasal disease

Gert ter Haar DVM, PhD, MRCVS, DECVS

SPECIALISTISCHE DIERENKLINIEK UTRECHT

Otorhinolaryngology

ENT

Surgery & Reconstruction of the Nose

Gert ter Haar, DVM, PhD, DECVS
European Specialist in Small Animal Surgery
Ear, Nose & Throat, Brachycephaly related and Audiologic Diseases
Oncologic and Reconstructive Surgery
President of the IVENTA

AniCura

Nasal plane

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Nasal plane

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SDU

Nasal plane tumors

- Squamous cell carcinoma
- Mastocytoma
- Malignant histiocytoma / Sarcoidosis
- Fibrosarcoma
- Malignant lymphoma

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Nasal plane

- Therapy SCC
  - Radiation therapy
  - Hyperthermia
  - Intratumoral administration of carboplatin
  - Cryosurgery
  - Photodynamic therapy
  - Resection of nasal plane!!

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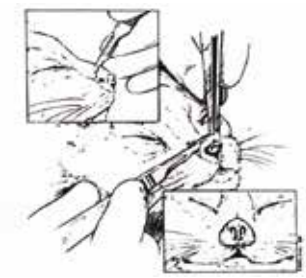
Resection Nasal Plane



From Withrow SJ, Straw RC. Resection of the nasal planum in nine cats and five dogs. J Am Anim Hosp Assoc 1990;26:219-222

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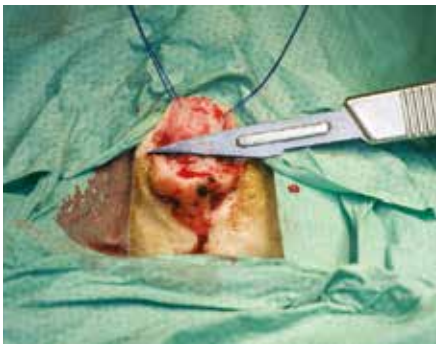
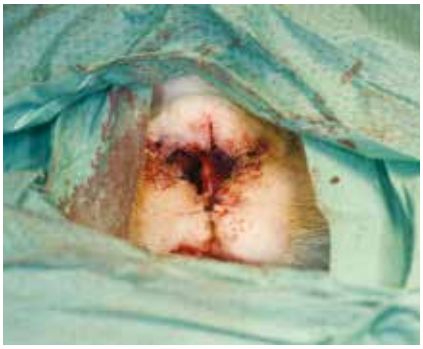
Resection nasal plane





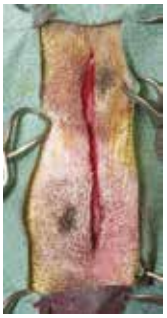
Rhinotomy

- Indications
  - Foreign body
  - Intranasal polyp
  - Chronic granulomatous inflammation in cats
  - Tumor biopsy
  - Tumor removal

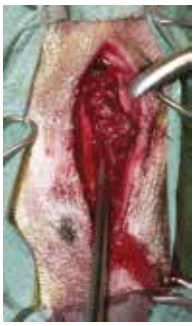
Rhinotomy

- Techniques
  - Dorsal rhinotomy
  - Ventral rhinotomy
  - Lateral rhinotomy

Dorsal Rhinotomy



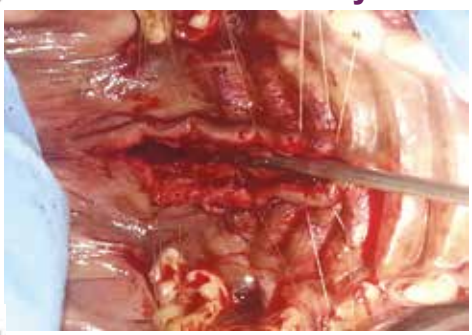
Dorsal Rhinotomy



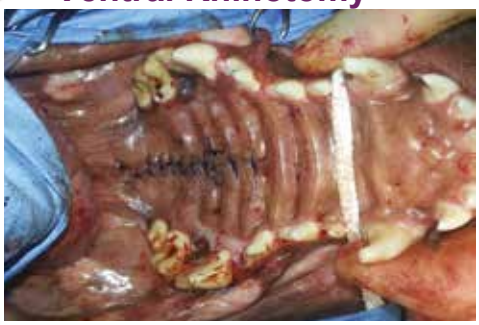
Rhinotomy

- Techniques
  - Dorsal rhinotomy
  - Ventral rhinotomy
  - Lateral rhinotomy

Ventral Rhinotomy



Ventral Rhinotomy



Rhinotomy

- Techniques
  - Dorsal rhinotomy
  - Ventral rhinotomy
  - Lateral rhinotomy

Lateral Rhinotomy in dogs

Hedlund CS: Rhinotomy techniques. In: Bojrab MJ; Current Techniques in Small Animal Surgery, 4th edition, 1998, fig. 20.15

Patient

- "Chip; Labrador Retriever
- Male, intact
- 9 years old
- 41.3 kg

Patient

- Dermatological problems
  - pruritis
- Epistaxis R
- No discharge
- Sometimes sneezing
- No other signs

Patient

- Physical exam:
  - No abnormalities
  - Ulcerative lesion inside nose, right side of nasal septum, cranially

Patient

- CT-scan
  - Nose & thorax
  - No abnormalities
- Rhinoscopy
  - Lesion extends 2 cm caudally, but is well circumscribed: Biopsy

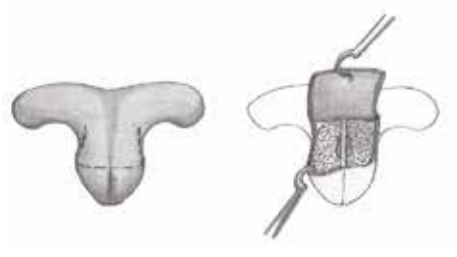
Patient

- Histopathology
  - Well differentiated squamous cell carcinoma
- Surgery

Modified Lateral Rhinotomy

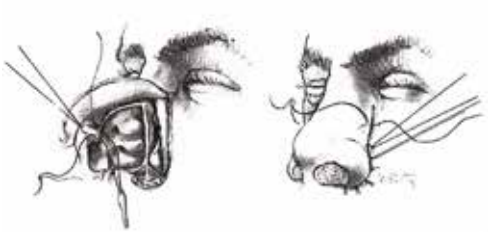
- For removal of nasal septal squamous cell carcinoma
- Combination of nasal septal resection technique (Pavletic) and lateral rhinotomy

Septal Resection Technique

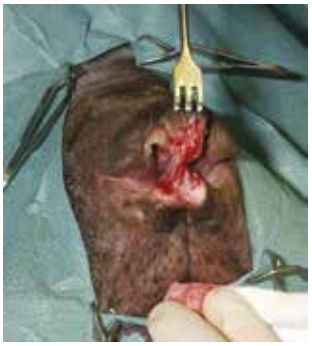


Pavletic MM. Septal Resection Technique. In: Pavletic MM, Atlas of Small Animal Reconstructive Surgery, 2nd edition 1999, Ch 19, nasal reconstruction techniques, page 400-401, plate 88.

Lateral Rhinotomy in humans

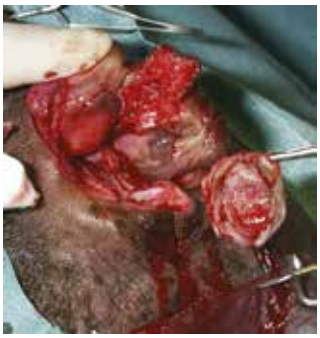


Modified Lateral Rhinotomy



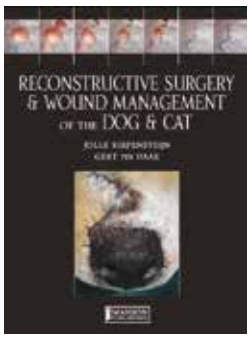
Modified Lateral Rhinotomy

Modified Lateral Rhinotomy



Introduction

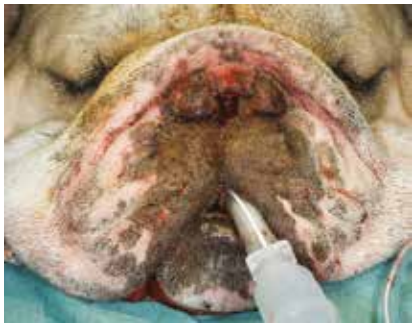
- Reconstructive surgery
- Face
  - Nasal plane
  - Nose
  - Remainder face



Oncologic / Reconstructive Sx

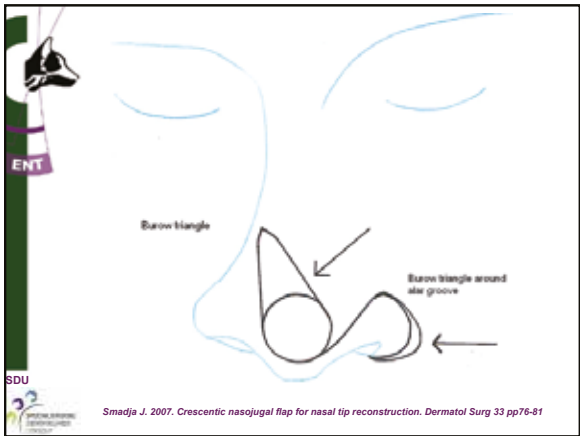
Oncologic / Reconstructive Sx

Oncologic / Reconstructive Sx

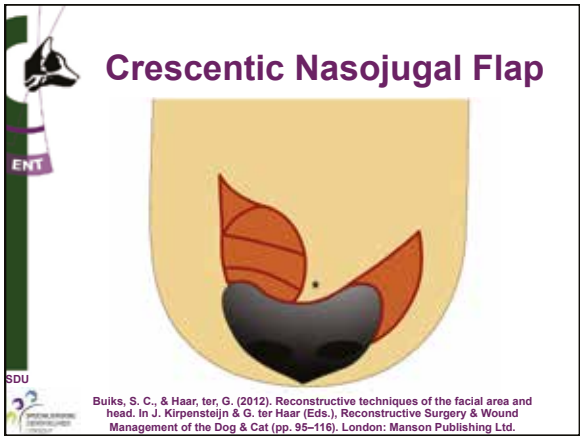


Crescentic Nasojugal Flap

- Carcinomas of the tip of the nose in people are very common
- the nasal tip defect is one of the most challenging ones to reconstruct
- There are numerous available procedures for reconstruction of the tip of the nose, but the nasojugal flap, due to its great reliability, yields excellent functional and cosmetic results


ENT
Burow triangle
Burow triangle around alar groove
SDU
Smadja J. 2007. Crescentic nasojugal flap for nasal tip reconstruction. Dermatol Surg 33 pp76-81


ENT
SDU


Crescentic Nasojugal Flap
ENT
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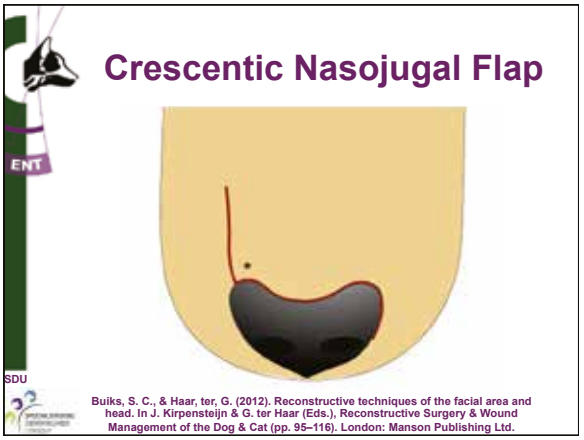
Buiks, S. C., & Haar, ter, G. (2012). Reconstructive techniques of the facial area and head. In J. Kirpensteijn & G. ter Haar (Eds.), Reconstructive Surgery & Wound Management of the Dog & Cat (pp. 95–116). London: Manson Publishing Ltd.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ENT
SDU


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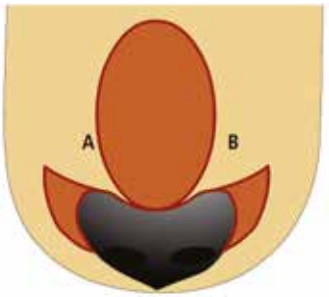
SDU

Crescentic Nasojugal Flap

- **Patient**
  - Bacio
  - Mixed breed cat, male, castrated
  - 2 years of age
  - Slowly expanding mass on dorsum of the nose
  - Histopathology: chronic pyogranulomatous dermatitis with intralesional fungal hyphae and spores, pseudomyocytoma
  - Plan: surgery

Crescentic Nasojugal Flap

Crescentic Nasojugal Flap



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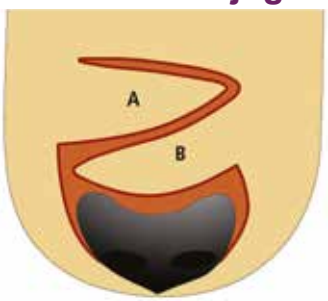
Crescentic Nasojugal Flap



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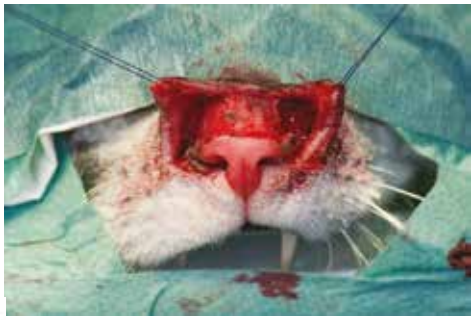
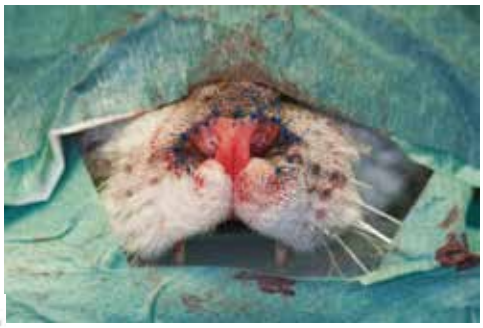


Crescentic Nasojugal Flap

Crescentic Nasojugal Flap

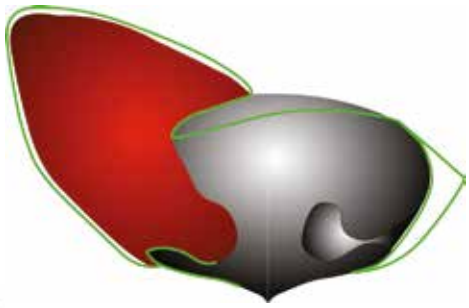


Crescentic Nasojugal Flap

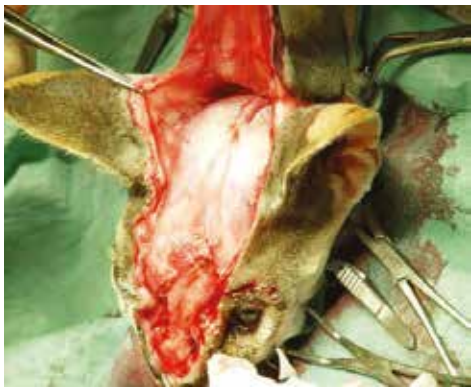
Crescentic Nasojugal Flap

- **Patient**
  - Chelo
  - Mixed breed dog, male, intact
  - 1.5 years of age
  - Brought from Portugal with lesion of nasal plane
  - Culture: no specific pathogens cultured
  - Histopathology: severe granulomatous inflammation with vasculitis and thrombosis
  - Plan: surgery

Crescentic Nasojugal Flap



Advancement or U-flap



Advancement or U-flap

Axial pattern flaps

- **Superficial temporal artery** (caudal nose)
- **Facial artery** (laterodorsal part of nose)
- **Direct cutaneous artery and vein**
  - Better perfusion than pedicle flaps

Superficial Temporal Artery

- For dogs and cats; cutaneous branch of the superficial temporal artery
- Maxilla, maxillofacial area and caudolateral side of the nose
- Base of flap determined by lateral orbital rim
- Width of flap is limited rostrally by the eye and caudally by the ear

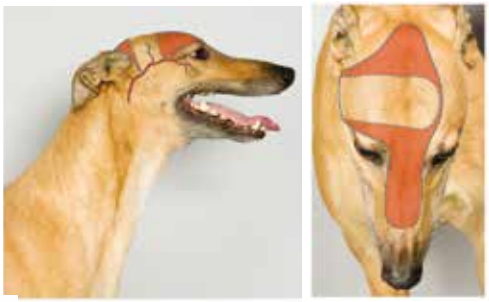
Fahie MA, Smith MM. Axial pattern flap based on the cutaneous branch of the superficial temporal artery in dogs: an experimental study and case report. *Vet Surg* 1999;28:141–147.
Fahie MA, Smith MM. Axial pattern flap based on the superficial temporal artery in cats: an experimental study. *Vet Surg* 1997;26:86–89.

Superficial Temporal Artery

- Width of flap is equal to length of zygomatic arch
- Length of flap limited to dorsal orbital rim of contralateral eye
- Brachycephalic breeds; limited amount of skin can be harvested!
- NB: necrosis distal end of the flap!

Fahie MA, Smith BJ, Ballard JB, et al. Regional peripheral vascular supply based on the superficial temporal artery in dogs and cats. *Anat, Histol, Embryol* 1998;27:205–208.
Pavletic MM. Axial Pattern Skin Flaps. In: Pavletic MM, ed. *Atlas of Small Animal Wound Management and Reconstructive Surgery*. 3rd ed. Wiley-Blackwell; 2010:357–402.

Superficial temporal a. a. p. f.



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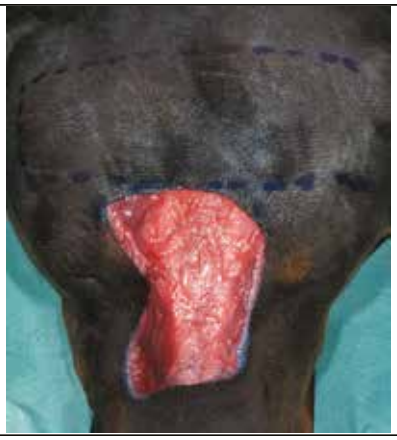


Superficial Temporal a.a.p.f.

Facial Artery

- Defects of the rostral and lateral nasal regions and maxilla
- Base of flap is located at the commissure of the lip
- Perfusion is by superior and inferior labial arteries
- Caudal border is lateral aspect of the atlas

Pavletic MM. Reconstructive surgery of the lips and cheek. *The Veterinary clinics of North America Small animal practice* 1990;20:201–226.
Pavletic MM. Facial Reconstruction. In: Pavletic MM, ed. *Atlas of Small Animal Wound Management and Reconstructive Surgery*. 3rd ed. Wiley-Blackwell; 2010:433–480.

Facial Artery

- Flap extending to the level of the vertical ear canal usually suffices however and reduces risk of necrosis of tip of the flap
- Lateral borders are the caudal mandible ventrally and ventral aspect zygomatic arch dorsally

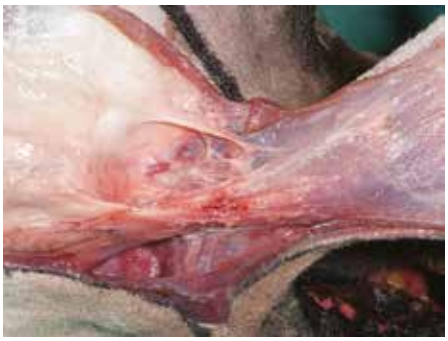
Yates G, Landon B, Edwards G. Investigation and clinical application of a novel axial pattern flap for nasal and facial reconstruction in the dog. *Aust Vet journal* 2007;85:113–118.
Milgram J, Weiser M, Kelmer E, et al. Axial pattern flap based on a cutaneous branch of the facial artery in cats. *Veterinary Surgery* 2011;40:347–351.

Facial artery axial pattern flap

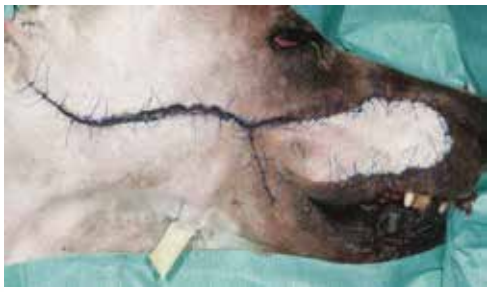
Buiks, S. C., & Haar, ter, G. (2012). Reconstructive techniques of the facial area and head. In J. Kirpensteijn & G. ter Haar (Eds.), Reconstructive Surgery & Wound Management of the Dog & Cat (pp. 95–116). London: Manson Publishing Ltd.

Facial artery axial pattern flap

Facial artery axial pattern flap



Facial artery axial pattern flap



Questions?

Exotics

Norin Chai (FR)

DVM, MSc, PhD

(Exotics)

Norin is currently the Deputy Director of the Ménagerie du Jardin des Plantes, the head of the veterinary department of the Ménagerie du Jardin des Plantes, as well as being the head vet of research facilities in the National Museum of Natural History. Norin's main research fields include: comparative medicine and pathology, infectious disease in amphibians and fish, and phyllogenetics of South East Asia wild bovids.

Update in anaesthesia and analgesia of small mammals

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57 rue Cuvier 75005 Paris, France

Abstract

What one should always have in mind is that these animals have a high heart and respiratory rates, and very stressables. Handling will be gentle and with calm, and should not be too long. Consulting rooms should be small, uncrowded, with no escaping doors and windows closed. Reduce the brightness of the room, avoid air draft. Clean the table between each visit to remove odors that could stress the animals. Beware of fast and dangerous hypothermia during anesthesia: think of heating in intra-and post-operative (water bottles, heating pads. ..). There is a rapid heat loss in the first 10 minutes of anesthesia. High metabolism means often limited reserves.

Fasting is not always needed, and when needed the length depend on species. Fasting does not significantly reduce gastrointestinal volume and may cause ileus in guinea pigs and other herbivores. High metabolic rates and small glycogen reserves predispose to hypoglycemia. In ferrets, fasting should not exceed 4 hours. Rabbits and small rodents should not be fasted because vomiting does not occur and hypoglycemia is a consequence. If fasting is not needed, it is important to always have a clean oropharynx.

Gas anesthesia with isoflurane is without contest the anesthetic of choice for pet rodents and lagomorphs. It is often challenging to intubate a little mammal: the author use an endoscope. V-gel is species-specific supraglottic airway device for general anesthesia and emergency resuscitation. Anatomic matching features combined with a soft gellike material to give a high-quality pressure seal that avoids laryngeal and tracheal trauma without the need for endotracheal intubation. V-gels are available for rabbits in 6 different sizes depending on patient body weight.

Resistance to many anesthetics is still seen making dose and induction time hazardous. Adding fixed drugs is dangerous (potentiation). Intranasal drug administration can be an effective and rapid route described in rabbits. The combination of ketamine with midazolam is commonly used in ferrets, rabbits, and Guinea pigs. When used appropriately, opioids can be administered safely to small mammals to effectively alleviate pain. They have a wide margin of safety and excellent analgesic properties. The author mainly uses butorphanol and buprenorphine. Buprenorphine is 35 times as potent as morphine and has a long duration of action (3–5 hours in mice, 6–8 hours in rats). An unusual effect of opioids in rodents is the production of pica behavior. This is thought to be analogous to vomiting in other species and has been reported following use of buprenorphine in rats. The incidence of pica appears very low, but if noted, analgesia should be provided using a nonopioid analgesic. Meloxicam has minimal side effects, although usually gastrointestinal when seen. Its ease of administration (owing to its commercially available liquid suspension), relative safety. It is the most used NSAID in small mammals. The author often uses this combination: MEDETOMIDINE OU DEXMEDETOMIDINE (0,2 mg/kg) + KETAMINE (5mg/kg) + MELOXICAM (0,5mg/kg) + BUPRENORPHINE (0,03mg/kg)

Patient monitoring helps detect early homeostatic imbalance before damage to organ systems become irreversible. Capillary refill time should be less than 2 seconds. Prolonged capillary refill time indicates poor tissue perfusion. The resting heart rate for small mammals is calculated using the allometric evaluation : Heart rate = 241 x $BW^{-0,25}$ (BW is body weight in kg). A heart rate greater than or less than 20% of the calculated value is considered tachycardia or bradycardia. If we use electrocardiogram (ECG), remember that electrical activity does not always ensure mechanical (pumping) activity of the heart. The author uses alligator clips attached to 25-gauge needles penetrating through skin. In case of severe bradycardia, reduce or terminate anesthesia and administer atropine or glycopyrrolate. Position of the probe of the pulse oximetry can be ear, tongue, buccal mucosa, vulva, prepuce, and proximal tail. Limitations are poorly perfused, pigmented, or vasoconstricted areas. For capnography, the mainstream method is not practical in small mammals because it adds more dead space.

Update in anaesthesia and analgesia of birds and reptiles

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Ménagerie du Jardin des Plantes, Muséum National d'Histoire Naturelle
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Reptiles

Reptiles especially aquatic animals, commonly exhibit species and individual differences in response to anesthetic drugs, dosages, and protocols. Thermoregulation in a healthy animal is accomplished by behavioral adaptations; however, sick animals often fail to effectively regulate their body temperature. The response to anesthetic drugs, as well as cardiopulmonary performance during anesthesia, is optimal if the patient is maintained within the species-specific preferred optimal temperature range. Most anesthetic drugs have cardiovascular depressant effects. Most agents, especially when used alone at high dosages, cause pronounced cardiopulmonary depression, prolonged induction and recovery times, and poor muscle relaxation during maintenance of anesthesia. Ketamine HCl is used in Reptiles for both immobilization and induction of anesthesia. It has a wide margin of safety and can be administered IM for immobilization and IV for induction of anesthesia. Ketamine alone provides poor muscle relaxation, minimal analgesia and, if used at high dosages, prolonged recovery times. Ketamine is rarely used alone because high dosages are required in most species to produce immobilization. Most commonly, ketamine is combined with benzodiazepines (i.e., diazepam, midazolam), opioids (i.e., butorphanol, buprenorphine) or the α2-adrenergic agonist medetomidine or dexmedetomidine. A combination will reduce the ketamine dose; this results in more rapid and smoother induction and recoveries, improves muscle relaxation, and provides analgesia. Tiletamine/zolazepam (Telazol) can be used for immobilization and induction of anesthesia. At high dosages (greater than or equal to 6 mg/kg IM) it is associated with prolonged recovery (greater than or equal to 48–72 hrs), especially in African spurr-thighed tortoises (Geochelone sulcata), and is not recommended. To facilitate handling of large chelonians (i.e., to gain access to a peripheral vein) a low dose of tiletamine/zolazepam (2–4 mg/kg IM) can be used. However, induction with propofol is then recommended. Medetomidine facilitates handling and concurrent administration of synergistic agents results in a plane of sedation effective for short procedures such as abscess debridement, shell repair procedures, and collection of diagnostic samples. It is most commonly combined with ketamine and an opioid (e.g., butorphanol). For reversal, atipamezole is administered IM at 5 times the medetomidine dose. Propofol has been used in a variety of reptile species for both induction and maintenance of anesthesia. It must be administered IV and the induction dosage depends on the health status of the chelonian and the type and amount of premedication. Propofol causes systemic hypotension, decreased myocardial contractility, and respiratory depression. Propofol (2 to 5 mg/kg IV) induces general anesthesia, especially following premedication with midazolam (0.5 to 1 mg/kg IV). Alphaxalone, is for the author a very useful drug ready for use. The author has found that 10 mg/ kg with IM injection give a very good sedation, fast, enough for quick procedures or intubation. Anesthetic induction with inhalational agents alone often results in prolonged induction times, especially in aquatic animals capable of prolonged breathholding. Isoflurane results in short recovery times and minimal cardiopulmonary depressant effects, and has limited effects on renal and hepatic function.

Injection of sedative and anesthetic drugs in the caudal body-half should be avoided if possible, since some drugs may undergo a hepatic first pass effect, which may be more important for most anesthetic drugs, compared to the more often discussed renal first-pass effect. Plasma levels and efficacy of anesthetic drugs can be greatly reduced and may require repeated drug administration.

Historically the intramuscular route has been recommended as the route of choice for non-vascular administration. However, the subcutaneous route provides a suitable alternative, in particular in animals with reduced muscle mass or if larger volumes of drugs need to be administered.

During anesthesia, respiration and heart rates are recorded regularly. Supportive care depends on preanesthetic clinical and laboratory findings, the size of the chelonian, and the procedure to be performed. Fluid therapy is accurately administered, even to small animals, with a syringe pump; most require maintenance fluid administration of a balanced electrolyte solution at 5 to 10 ml/kg per hour. The patient is kept within the preferred optimal body temperature range for the particular species. The anesthetic plane is frequently assessed for the presence or absence of reflexes such as righting, palpebral, cloacal, and limb and tail withdrawal reflexes. At a surgical plane the righting and palpebral reflexes are usually absent. Absence of the corneal reflex and no response to a surgical stimulus indicates a deep plane of anesthesia. Electrocardiography (ECG) is performed with leads attached in a conventional manner and detects changes in heart rate such as tachycardia, bradycardia and arrhythmias. However, it does not evaluate mechanical performance of the heart. The use of pulse oximeters in Reptiles to monitor heart rate, arterial oxygen saturation (SpO2), and diagnose hypoxemia during anesthesia is of limited value because all devices are calibrated based on the human oxygen hemoglobin

dissociation curve. However, at present, pulse oximetry may be more useful to detect trends in arterial oxygen desaturation.

Our knowledge on reptile analgesia continues to grow. In general most mu-opioid receptor agonists have been shown to provide analgesia in turtles and tortoises as well as lizards and crocodilians. Drugs such as morphine, hydromorphone, fentanyl and tramadol are most commonly used.

Birds

Anesthesia and analgesia are an important part of the daily routine associated with avian veterinary practice. These procedures differ from mammal medicine primarily because of different physiologic composition and different anatomical structures, which are described when relevant to anesthetic management.

Injectable anesthetic agents are typically metabolized in the liver and eliminated through the kidneys. Patients that are diagnosed with hepatic and/or renal disease may have reduced drug elimination, a long anesthetic recovery period, and concurrent cardiopulmonary depression. A surgical plane of anesthesia using injectable anesthetic agents is typically possible for up to 30 minutes, which can be used primarily for short surgical procedures, sedation for diagnostic purposes, and sampling during field studies. Local anesthesia is not commonly used in avian patients because small doses of local anesthetic drugs may have toxic effects and, most importantly, the patient remains conscious during a very stressful procedure. Ketamine should not be used as a single anesthetic agent, but may be used in combination with an alpha-2-agonist drug. In the same way, as alpha-2-agonists has cardiopulmonary depressive action, they should not be used as a monoanesthetic agent. Actually, midazolam is the most common drug used for sedation of pet birds and has a wide safety margin. Midazolam has sedative, muscle relaxing, anxiolytic, amnestic, and appetite-stimulating properties in birds. The injectable form of midazolam (midazolam hydrochloride, 5 mg/mL) or a more concentrated form (50 mg/mL) can be administered intranasally and/or intramuscularly without side effects. Dosages of midazolam commonly used in pet birds range from 0.5 to 3 mg/kg. The author routinely uses 2 mg/kg of midazolam in pet birds, if administered intranasally and as the sole sedative agent. Butorphanol is the most commonly used opioid analgesic in birds. Besides its analgesic effects, butorphanol also has sedative effects, which are potentiated by benzodiazepines (e.g., midazolam and diazepam). The combined administration of midazolam and butorphanol is recommended in birds for which midazolam alone provides an insufficient level of sedation or which require deeper sedation for certain clinical procedures (e.g., radiographic positioning). Butorphanol can be combined with midazolam into a single syringe and administered intramuscularly or intranasally. No side effects of intranasal administration of butorphanol at a dose range of 1 to 3 mg/kg have been reported in psittacines. The dose routinely used for intranasal or intramuscular administration is a combination of butorphanol (1 to 2 mg/kg) with midazolam (1 to 2 mg/kg) to pet birds.

When possible, inhalation is the method of choice for anesthesia in birds. Modern inhalation anesthetic agents have a low blood-gas solubility, which results in a rapid induction as well as a rapid recovery. Therefore, the concentration of administered anesthetic gas can be adjusted easily.

Basic considerations are similar to small mammal medicine and anesthetic circuits include nonrebreathing systems, pediatric circles, and adult circle systems. If necessary, the bird can be

ventilated using volume- or pressure-controlled application systems, the latter having major advantages because they are patient-size independent. The main risk of inhalation anesthesia, especially in smaller birds, is hypothermia because of the large surface area of the air sac system.

Butorphanol (1-3 mg/kg IM) is the current recommendation for opioid analgesia in parrots. Buprenorphine at 0.1 mg/kg IM in African grey parrots did not show an analgesic effect when tested by analgesimetry. However, clinical use of buprenorphine suggests it has an analgesic effect.

Some surgical procedures in birds and reptiles

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Reptile and Bird surgeries are extremely heterogeneous fields, with numerous species that have unique physiologic, anatomic, and pathologic differences. The purpose of this lecture is to describe common surgical techniques used in reptiles and birds.

Surgery of the skin in reptiles is indicated for wound management, cutaneous and subcutaneous abscesses, and removal of neoplasms.

In a study on wound healing in garter snakes (*Thamnophis sirtalis*) in which sutured and unsutured wounds were compared, unsutured linear incisions tended to have more rapid epithelial maturation and a less intense inflammatory response. On this basis, the investigators suggested that suturing small incisional wounds may not be advantageous.

Current recommendation for closure of coelomic breaches is to use absorbable synthetic monofilament suture material (eg, poliglecaprone 25, polyglyconate). Subcutaneous abscesses are a frequent finding in captive reptiles. The clinician has to remember that etiology may be more likely multifactorial. After surgical treatment, appropriate changes in management and diet are crucial to avoid recurrences. Esophagostomy tube placement is typically recommended in anorectic chelonians, given the difficulty of oral administration of food and/or medication. It may also be indicated in aggressive, chronically ill squamates. In case of foreign bodies, whenever removal from the oral cavity is not feasible, esophagotomy is performed. Surgical approaches to the coelom greatly vary depending on the species. In general, the size of the incision will depend on the indications for coeliotomy. During the opening of the coelomic membrane, the surgeon should pay attention for signs of free gas or liquid in the coelom, associated with gastrointestinal tract and urinary bladder perforation, respectively. After entering the coelom, all the organs that are visible should be carefully inspected. In chelonians, the two main approaches to the coelom are plastron osteotomy and prefemoral fossa coeliotomy. With the increased availability of laparoscopic and endosurgical equipment, prefemoral fossa coeliotomy has gained popularity due to the reduced invasiveness compared with plastron osteotomy. Coeliotomy will allowed reproductive surgery, cystotomy, gastrointestinal track surgery.

In Birds, in the author's experience, the main surgeries concern orthopedic surgery and skin surgeries. Skin surgeries are mostly wound repairs, neoplasia excisions. Soft tissue retractors, microsurgical instrumentation are often needed. It is fundamental to decrease the amount of blood loss. Coelomic surgery, like enterotomy or salpingotomy, has a high risk of resulting in peritonitis, airsacculitis, and pneumonia. In all cases, the author prefer minimal invasive surgery.

Reproductive disorders in birds and reptiles

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Reproductive tract disorders are common and include preovulatory stasis, postovulatory stasis, yolk coelomitis, ectopic eggs, and neoplasia, among others. In this lecture, we will mainly talk about dystocias which are the main reproductive disorders in birds and reptiles. Dystocia is a failure of an egg to pass through the reproductive tract within a normal period of time. Dystocia can be obstructive or nonobstructive. The subjects are reproductively active females and may concern all species.

The risk factors are the stress, adverse environmental conditions, inappropriate husbandry (e.g. lack of egg laying site), anatomic abnormalities, endocrine disorders, neoplasia, nutritional deficiency (e.g., calcium, vitamin E), obesity, malformed eggs, follicular Stasis, systemic/Metabolic disease, young or old animal... The history and chief complaint are anorexia, depression, weakness, dyspnea, nesting behavior for birds or unceasing digging behavior for reptiles. We may also see reluctance to fly or perch, drooped wings in birds and paresis in tortoise.

Clinical signs come from the fact that abnormally prolonged presence of an egg in the oviduct causes a multitude of complications. An egg lodged in the pelvis may compress the pelvic vessels and kidneys, causing circulatory disorders and shock. An impacted egg may cause metabolic disturbances by interfering with normal defecation and micturition, inducing ileus and renal dysfunction. The pressure necrosis to all three layers of the oviductal wall may ultimately lead to rupture.

Imaging (radiography and ultrasound) is the main diagnosis. It will aid in evaluation of the position and characterization of the egg(s). Eggs with noncalcified shell may appear similar to a mass in the caudal coelomic cavity. Multiple eggs may be identified owing to an obstruction distally or secondary to motility disorders. Eggs could be within the oviduct or ectopic due to oviductal rupture or retroperistalsis.

The goals of the therapeutic are first to stabilize and improve patient's physical condition by nursing care. For tortoise, the first step is to propose a nest.

Medical treatment is attempted first in nonobstructive cases of egg binding. If oviposition has not occurred within 2-12 hours, or there is indication of obstruction, surgical intervention should be considered. Medications include: Calcium gluconate, Prostaglandin E2, Oxytocin, Vitamin D3 and vitamin A (especially in aquatic turtles).

Before surgical delivery, we may attempt, manual delivery (be careful of cloacal prolapse), ovocentesis.

Feline cardiology

David Connolly

BSc BVetMed PhD CertVC CertSAM DipECVIM (cardiology) MRCVS

After qualifying from the Royal Veterinary College, David pursued a career in research with the Medical Research Council. Having obtained a PhD in molecular genetics, he continued research for three further years, investigating the molecular mechanism underlying early embryonic development.

Following a period working for the PDSA in the Midlands, he returned to the RVC and completed a Residency in the Medicine Service. During this time, David obtained the RCVS Certificates in Small Animal Medicine and Cardiology, and subsequently the ECVIM Diploma in Cardiology in 2003.

David is currently Head of the Clinical Cardiology Service at the Queen Mother Hospital for Animals.

David's main research interest is novel interventional techniques for the treatment of cardiovascular disorders and the application of cardiac biomarkers to the clinical case.

Diagnosis of heart disease in cats

David Connolly
BSc BVetMed PhD CertVC CertSAM DipECVIM (cardiology) MRCVS

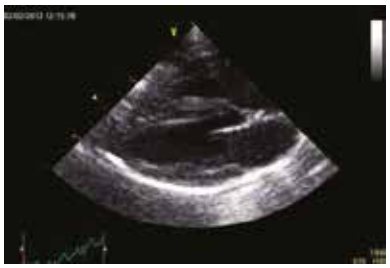
The Diagnosis of Cardiovascular Disease in Cats

David Connolly

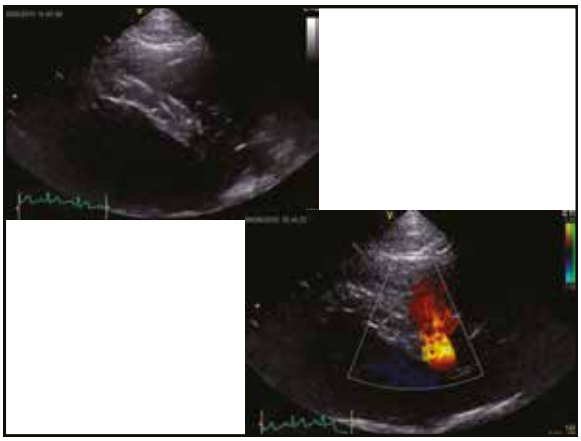
Royal Veterinary College
University of London

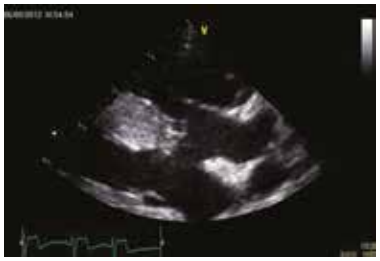
History

- Age
  - Congenital disease in young
    - VSD
    - PDA
    - Balanced AV canal defects – no murmur
  - Acquired diseases
    - HCM <6m to >12 yrs
- Breed
  - About 15-17% of non-pedigree cats have HCM.
  - The prevalence in pedigree cats is even greater- they include Main Coon, Ragdoll, British Shorthair, NFC, Burman, Sphynx
- Note However Many Young Cats have incidental murmurs in the absence of structural heart disease.



Normal heart in cat with murmur





History

- May have no clinical signs of disease
  - Particularly common with subclinical HCM
- Common historical complaints of symptomatic HCM include:
  - Dyspnoea/tachypnoea
  - Panting
    - Severe CHF, Severe dynamic LVOT obstruction causing chest pain.
  - Weight loss
  - Syncope
  - SCD
  - Rarely Ascites/ peripheral oedema
  - Cough – more frequently associated with broncho-constrictive disease.

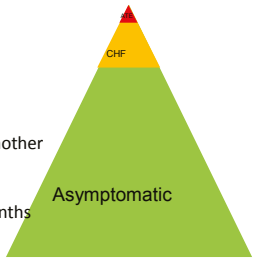
Presenting signs of HCM

HCM is a very heterogeneous disease.

Many cats present with mild asymptomatic HCM and never progress eventually dying of another condition

Cat can present in CHF at 6 months of age.

For many cats the first sign of HCM is SCD

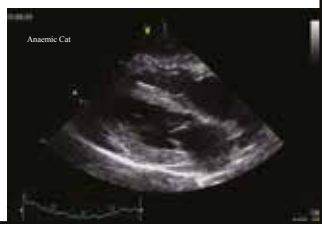


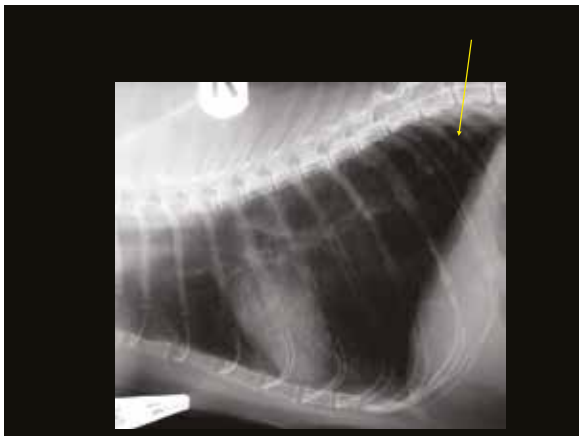
Differential diagnosis

- Historical signs of cardiac disease are not exclusive to cardiac abnormalities.

Need to also consider

- 1º Respiratory disease – allergic airway disease
- Hyperthyroidism
- Anaemia
- Hypertension





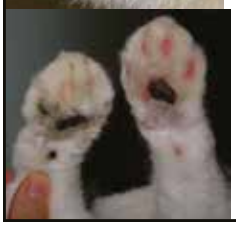
Physical examination

- Observation
- Auscultation
- Palpation

Observe

- Respiratory rate and effort
- Demeanour
- Jugular venous distension
- Mucous membranes
- Foot Pads
- Body condition
- Ascites
- Retinal lesions
  - Hypertension/Taurine deficiency

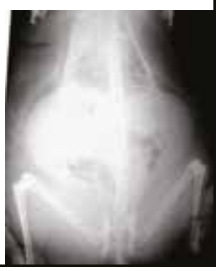
Pallor
Poor peripheral perfusion
Anaemia



Palpation

- Palpate precordium
  - Presence of thrill e.g. VSD if on right side
  - Often prominent in HCM
- Palpate abdomen
  - Ascites - ARVC/3rd degree AV block?
  - Concurrent disease – end stage kidney
- Palpate neck
  - Thyroid goitre
- Palpate distal limbs
  - Cold/painful/pulseless
- Palpate pulse
  - Is it regular - arrhythmia
  - Pulse deficits!
    - AF is rare VPCs are common

Cat with ascites.



No pulse in pelvic limb which will been cold to the touch

Auscultation

- Perform in a **quiet** room with minimal stress
- Cats can be retrained from purring by occluding the nares or gently running water in a nearby sink.
- If all fails then repeat the exam later when the animal has calmed down.

Auscultation

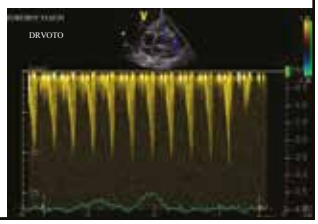
The two main cardiac sounds to consider are:

1. Heart murmurs
2. Gallop sound

Heart murmurs are the result of turbulent blood flow and are most commonly systolic in cats and frequently vary in intensity depending on heart rate.

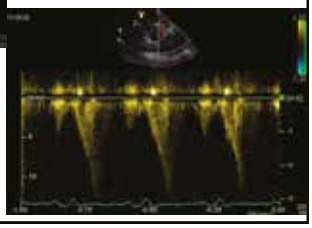
Causes of murmurs in cats:

- Functional
- HCM
- Other cardiomyopathies
- Hypertension
- Hyperthyroidism
- Anaemia
- Congenital heart disease
- Endocarditis



Functional murmurs can be caused by DRVOTO

Dynamic RVOTO



CatScan project

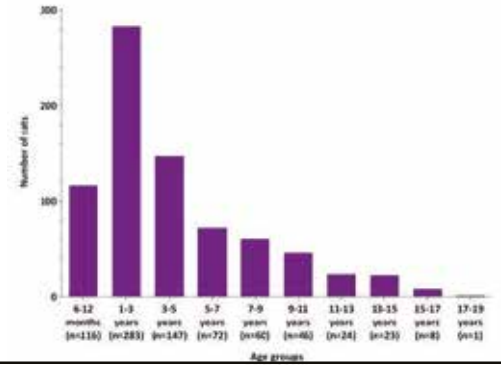
Study population

- 1,007 cats screened for inclusion
- 780 cats scanned

RVC

CatScan project

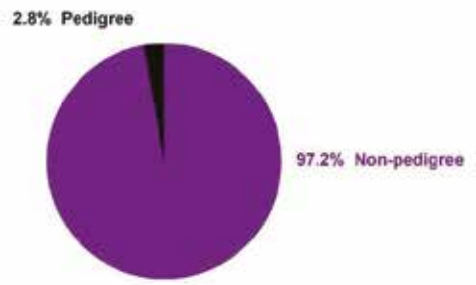
Age distribution



RVC

CatScan project

Breed distribution



RVC

CatScan project

Auscultation

- 1,630 auscultations of diagnostic quality
- Murmur prevalence
  - In this study = 40.8%
- Grade II/VI, left sternal border, variable

RVC

CatScan project

Auscultation

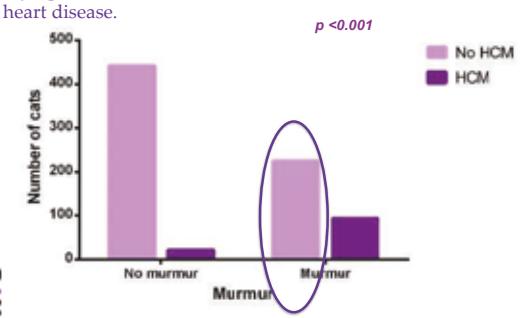
- Cats with murmurs…
  - More likely to be older
  - More likely to be male
  - Weighed significantly more

RVC

CatScan project

Murmur vs HCM

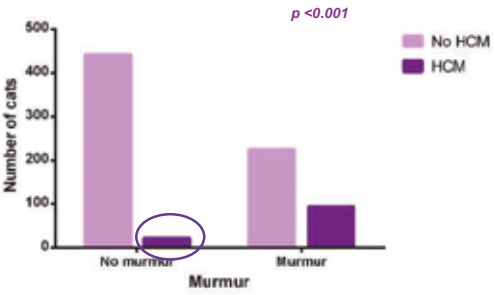
Therefore the detection of a systolic murmur in an asymptomatic cat is NOT an accurate indicator of structural heart disease.



RVC

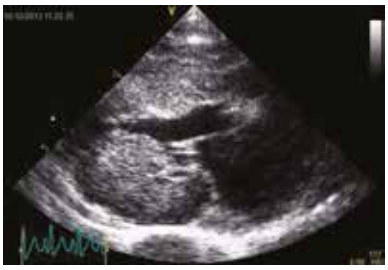
CatScan project

Murmur vs HCM



RVC

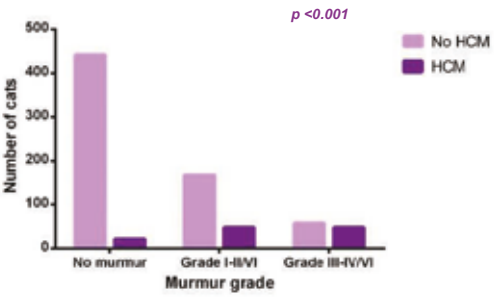
- Equally it is possible to have a cat with HCM and marked thickening of the LV without a murmur.



CatScan project

Murmur vs HCM

The higher intensity of the murmur the greater chance of HCM

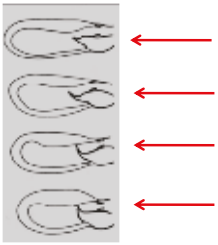


RVC

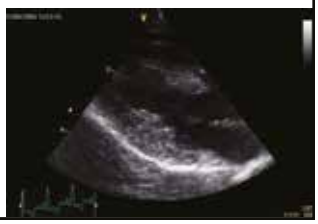
Common causes of murmurs in cats:

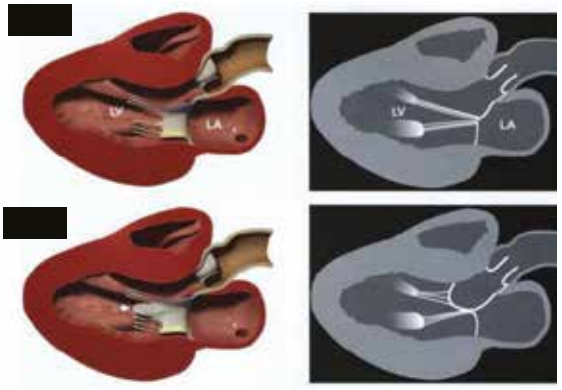
- HCM
- Functional
- Hyperthyroidism
- Anaemia
- Other cardiomyopathies
- Hypertension
- Congenital heart disease
- (Infective endocarditis)

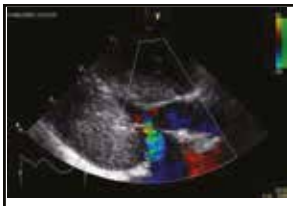
Remember murmurs in cats with HCM are frequently variable in intensity due to dynamic left outflow tract obstruction.

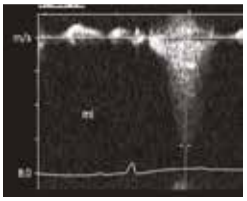


Systolic anterior motion

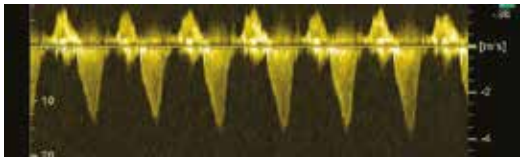








Dynamic LVOTO

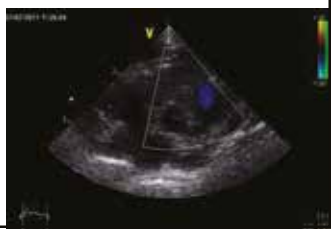


HCM:

- Any age
- Can have prominent apical impulse
- variable murmur intensity (sternal) or none
- Can be associated with open mouth breathing

Functional murmurs:

- Any age
- Louder when stressed
- Variable intensity
- Generally lower grade



Systemic hypertension:

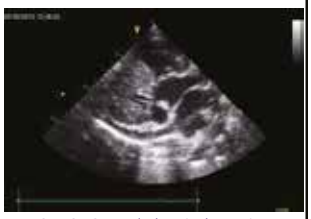
- renal disease
- retinal lesions
- Murmur may be variable

Hyperthyroidism:

- Usually >10y
- Concurrent clinical signs
- Variable intensity murmur
- Open moth breathing
- May also have arrhythmias

Congenital heart disease:

- May not always be very young
- Palpable thrill suggestive
- Cardiomegaly may be pronounced
- Echo can be challenging



ASD, VSD, Supravalvular mitral stenosis

Gallop sound

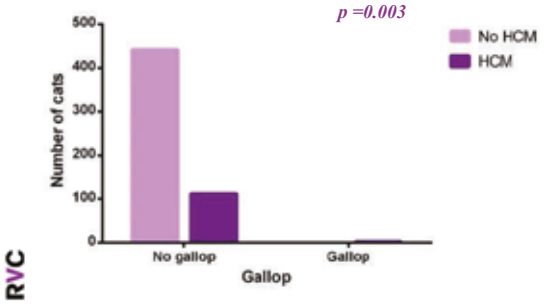
- Additional diastolic heart sound, that is not a murmur.
- It generally represents the heart sound S_3.
- It is a strong indicator of poor diastolic relaxation of the left ventricle
- It is nearly always associated with significant structural heart disease.

The presence of a gallop sound is a significant indicator of underlying heart disease in the cat.

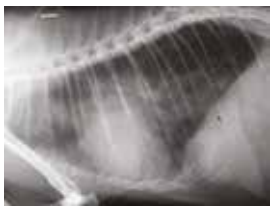
Detection of a gallop and arrhythmia would indicate advance cardiac disease.

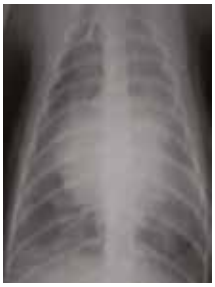
CatScan project

Gallop vs HCM

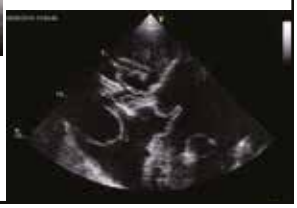


Tachypnoea ± crackles can be associated with CHF or primary lung disease





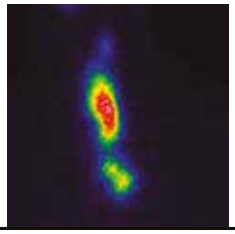
Dullness ventrally, or absent breath sounds suggest pleural effusion.

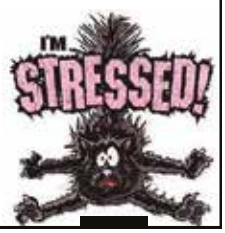


Note also that in cats with HCM:

Additional 'stresses' such as:

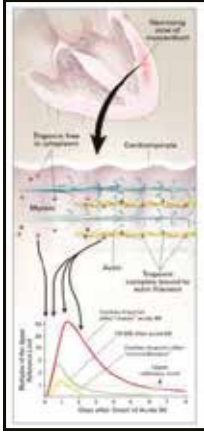
Surgery, fluid therapy, GA, systemic disease or environmental changes can result in rapid decompensation of a stable animal.





The use of Biomarkers in diagnosing feline cardiac disease.

- The 2 best studied biomarkers in feline cardiology are cTnI and NT-proBNP.
- To date these biomarkers have been shown to
  - Enable detection of clinical and sub-clinical disease
  - Enable discrimination between diseases presenting with similar clinical signs.

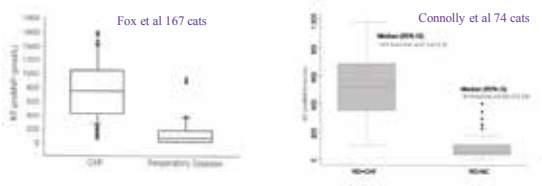


Concentrations in normal cats: Using an assay with an analytical sensitivity of 0.03ng/ml. Range <0.03-0.16ng/ml, mean 0.04ng/ml and median of 0.03ng/ml.

Cardiac biomarkers to differentiate between cardiac and non-cardiac causes of respiratory distress in cats.

Biomarker	Number of cats	Conclusions	Citation	Evidence category
cTnI	43 cats with dyspnea (31 cardiac)	Able to identify cardiac causes of dyspnea: AUC 0.84.[3] Overlap between groups	Herndon[4]	P;M;C;E
cTnI	53 cats with dyspnea (23 cardiac)	Significant difference between cardiac and non-cardiac: AUC 0.84.[5] Overlap between groups	Connolly[5]	P;M;C
NTproANP NTproBNP	85 dyspneic cats (44 cardiac)	Both NTproANP[6] and NTproBNP[6] were able to discriminate between cardiac and non-cardiac patients, but NTproBNP better performance (cut-off 220 pmol/L, AUC 0.96)	Connolly[6]	P;C;B
NTproBNP	162 dyspneic cats (101 cardiac)	Reliable discrimination between cardiac and respiratory causes of dyspnea: cut-off 207 pmol/L, AUC 0.98[6]	Fox[7]	P;M;C;E
NTproBNP	21 cats with pleural effusion (11 cardiac disease)	NTproBNP successfully discriminated between cardiogenic and non-cardiac causes of pleural effusion: cut-off 258 pmol/L, AUC 1.0[8]	Hassdenteufel[8]	P;C;B;E
NTproBNP	40 cats with pleural effusion (22 cardiac)	Plasma NTproBNP reliably identified cats with cardiogenic pleural effusion: cut-off 214 pmol/L, AUC 0.91 **Pleural fluid** NT-proBNP cut-off of 322·3 pmol/mL AUC 0.95	Humm[10]	P;C;
Point of care NTproBNP	78 cats 43 with cardiogenic pleural effusion	**Pleural fluid** POC test had good sensitivity (100%) but low specificity (64.7%). AUC 0.824		

Studies have shown that NT-proBNP can distinguish cats with CHF from those with Non cardiac dyspnoea.



	Fox et al median (IQR) plasma NT-proBNP (pmol/l)	Connolly et al median (IQR 25th and 75th percentiles) serum NT-proBNP (pmol/l)
Respiratory dyspnea	76.5 (24-180)	45 (75, 26-101)
CHF	754 (437-1035)	532 (336, 347-683)
Cut-off and sn and sp	265 sn 90.2% sp 87.9%	220 sn 93.9% sp 87.8%

Summary of the studies reporting the use of cardiac biomarkers to detect occult heart disease in cats

Test	Number of cats	Conclusions	Citation	Study design
NT-proBNP	201 cats: 99 normal, 9 equivocal HCM, 15 mild HCM, 17 moderate HCM, 61 severe HCM	No difference in NTproBNP between equivocal and healthy cats.[1] Severe HCM had significantly higher NTproBNP than other groups. Cut-off for mild HCM detection 100 pmol/L, AUC 0.96	Wess[1]	P;C;B;E
NT-proBNP	227 cats: 114 normal, 87 HCM, 22 UCM, 3 UCM, 1 DCM	NTproBNP effectively discriminated between normal cats and those with occult cardiomyopathy.[2] Cut-off 99 pmol/L, AUC 0.92. Correlation of NTproBNP with LV wall thickness and LA size.	Fox[14]	P;M;C;B ;E
NT-proBNP	146 cats: 43 normal, 16 equivocal, 50 mild heart disease, 37 moderate/severe	NTproBNP SNAP test can be used to help exclude moderate to severe occult cardiomyopathy; negative predictive value 94%[1]	Machen[1] [2]	P;M;C;B ;

The use of NT-proBNP in diagnosing feline heart disease.

Who should I run an NT-proBNP test on	Cardiopet® proBNP (pmol/L)	SNAP® Feline proBNP	Interpretation of test result
Cat with respiratory signs in which the cause of the signs in not obvious despite other appropriate diagnostic tests	< 100	Normal	Does NOT support at diagnosis of CHF
	100-270	Abnormal	CHF is possible; review the balance of evidence from the other tests
	> 270		Supports a diagnosis of CHF
Asymptomatic cat with cardiac risk factors (e.g. murmur, arrhythmia, gallop heart sound)	<100	Normal	Significant heart disease can be ruled out with a high degree of accuracy
	Increase of >100	Abnormal	Increased risk of having significant heart disease; recommend an echocardiogram

NT-proBNP can also be affected by:

i. Thyroid status,
ii. Systemic and pulmonary hypertension,
iii. Sample handling
iv. BUT not significantly by renal function.

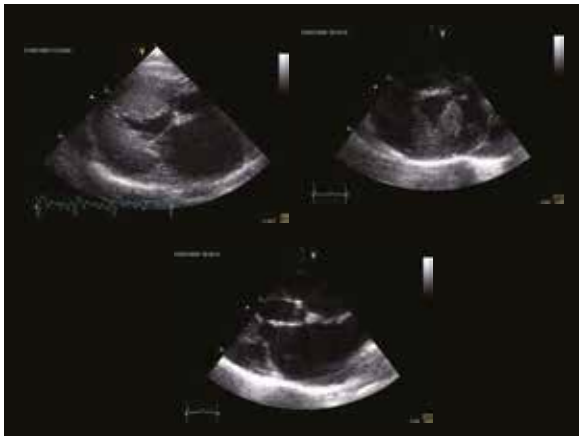
Review Articles on Biomarkers in Feline Heart Disease.

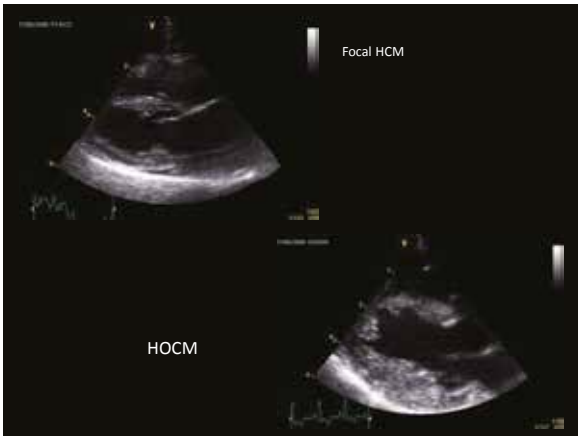
- Oyama MA, Boswood A, Connolly DJ et al. Clinical usefulness of an assay for measurement of circulating N-terminal pro-B-type natriuretic peptide concentration in dogs and cats with heart disease. J Am Vet Med Assoc. 2013 Jul 1;243(1):71-82.
- Borgeat K, Connolly DJ, Luis Fuentes V. Cardiac biomarkers in cats. J Vet Cardiol. 2015 Dec;17 Suppl 1:S74-86
- Hezzell MJ, Rush JE, Humm K, Rozanski EA, Sargent J, Connolly DJ, Boswood A, Oyama MA. Differentiation of Cardiac from Noncardiac Pleural Effusions in Cats using Second-Generation Quantitative and Point-of-Care NT-proBNP Measurements. J Vet Intern Med. 2016;30(2):536-42.

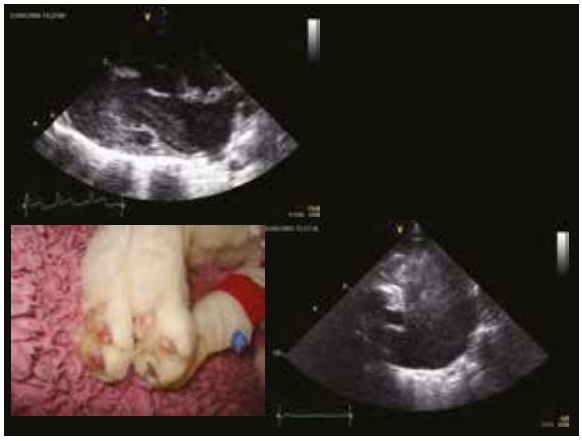
Conclusion:

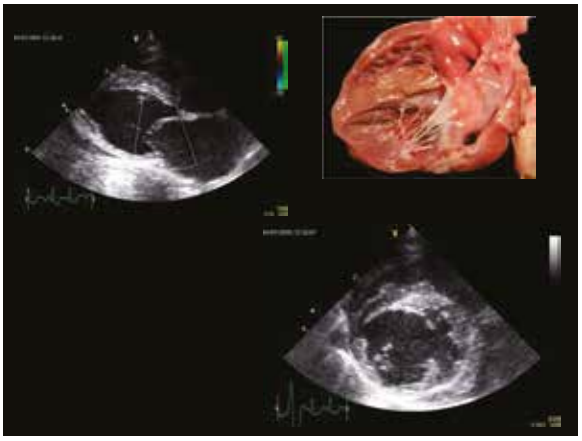
i. Cats are not small dogs.
ii. Systolic murmurs are not diagnostic for heart disease
iii. Gallop sound and arrhythmia are strong indictors for structural heart disease
iv. Biomarkers have significant diagnostic use in cats – in particular the have a significant negative predictive valve.
v. i.e. they rule out heart disease

Echocardiographic examples of recognised feline cardiomyopathies.









Prognostic indicators in cats with HCM

David Connolly
BSc BVetMed PhD CertVC CertSAM DipECVIM (cardiology) MRCVS

Royal Veterinary College
University of London

Prognostic indicators in cats with HCM

David Connolly

CatScan project

Study population

- 1,007 cats screened for inclusion
- 780 cats scanned

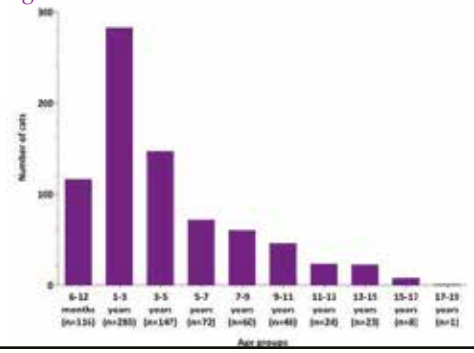
Payne J, Borgeat K, Connolly DJ, Boswood A, Dennis S, Wagner T, Menaut P, Maerz I, Evans D, Simons V, Brodbelt D, Luis Fuentes V. Prognostic indicators in cats with hypertrophic cardiomyopathy J Vet Intern Med 2013 Nov-Dec;27(6):1427-36

Payne JR, Borgeat K, Brodbelt DC, Connolly DJ, Luis Fuentes V. Risk factors associated with sudden death vs. congestive heart failure or arterial thromboembolism in cats with hypertrophic cardiomyopathy. J Vet Cardiol. 2015 Dec;17 Suppl 1:S318-28.

RVC

CatScan project

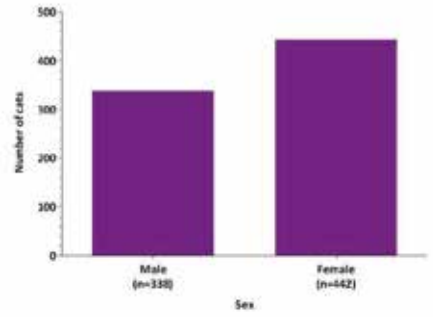
Age distribution



RVC

CatScan project

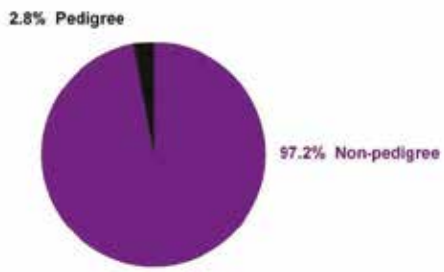
Sex distribution



RVC

CatScan project

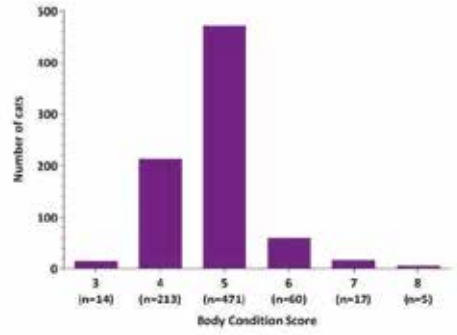
Breed distribution



RVC

CatScan project

Body condition score distribution



RVC

CatScan project

Echocardiography

Normal

635

111 'Low-risk' HCM

4 'High-risk' HCM

2 PPDH
1 VSD
1 RCM
1 Supravalvular mitral stenosis
1 abnormal papillary muscle

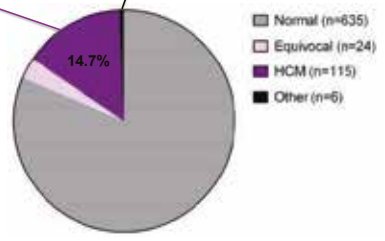
RVC

CatScan project

Echocardiography

- 2 PPDH
- 1 VSD
- 1 RCM
- 1 Supravalvular mitral stenosis
- 1 abnormal papillary muscle

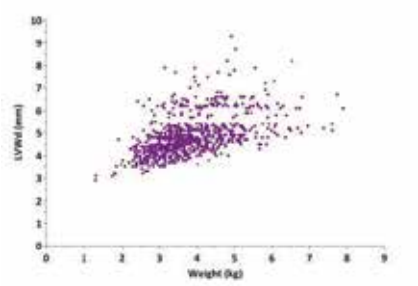
- 111 without left atrial enlargement
- 4 with left atrial enlargement



RVC

CatScan project

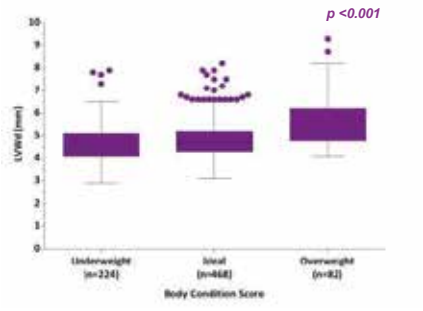
Echocardiography



RVC

CatScan project

Echocardiography



RVC

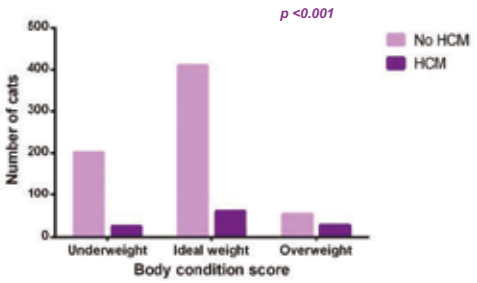
CatScan project

Sex vs HCM

RVC

CatScan project

Overweight vs HCM



RVC

CatScan project

HCM

- Irrespective of cut-off used, cats with HCM…
  - Older
  - More likely to be male
  - More likely to be overweight
  - More likely to have a murmur
  - More likely to have a gallop sound

RVC

RVC Royal Veterinary College University of London

Survival

Results

Population characteristics

- 285 cats
- 6.26 (0.37 – 17.40) years
- 75.8% male
- 80% domestic

RVC

Results

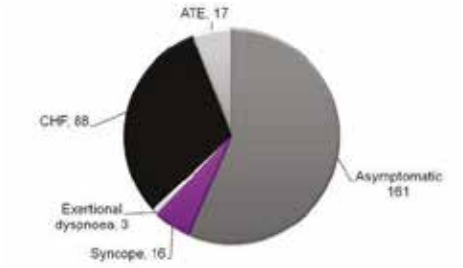
Population characteristics

- 73.0% murmur
- 23.5% gallop
- 17.5% arrhythmia (auscultation)
- 27.0% arrhythmia (ECG)

RVC

Results

Population characteristics



RVC

Results

Survival

285 cats

RVC

Results

Survival

285 cats

3 lost to follow up

282 with follow up

Median follow up time: 729 (0-2,755) days

RVC

Results

Survival

285 cats

3 lost to follow up

282 with follow up

Median follow up time: 729 (0-2,755) days

119 Alive

106 Deceased (cardiac)

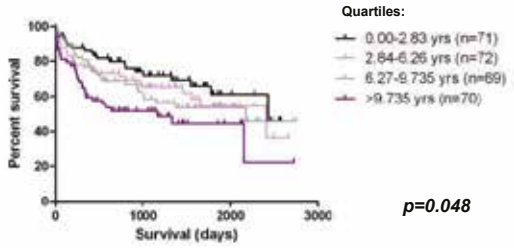
57 Deceased (non cardiac)

Median survival time: 2,171 (0-2,755) days

RVC

Results

Survival – age at presentation

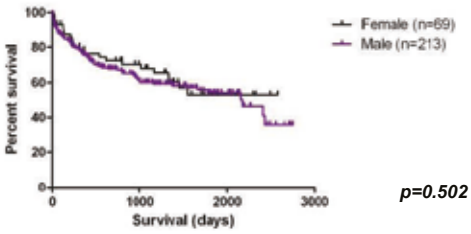


p=0.048

RVC

Results

Survival – sex

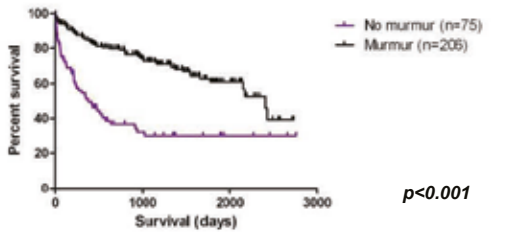


p=0.502

RVC

Results

Survival – murmur

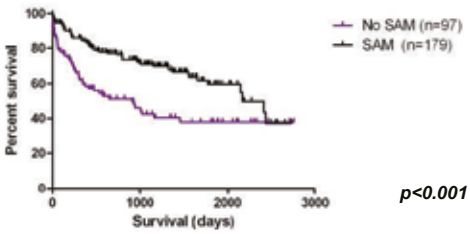


p<0.001

RVC

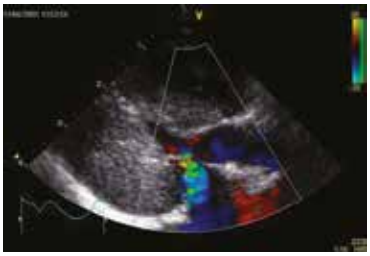
Results

Survival – Systolic anterior motion of the mitral valve



p<0.001

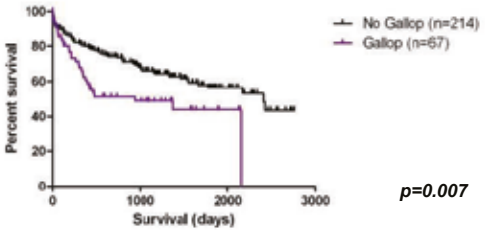
RVC



RVC

Results

Survival – gallop

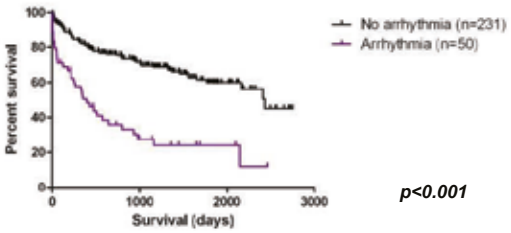


p=0.007

RVC

Results

Survival – arrhythmia (auscultation)

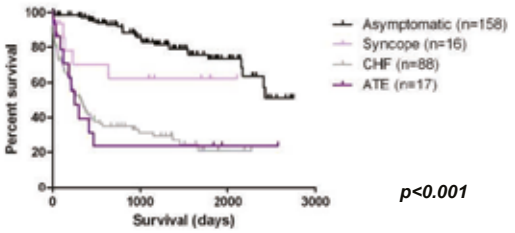


p<0.001

RVC

Results

Survival – symptomatic status

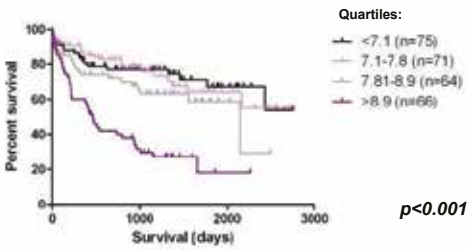


p<0.001

RVC

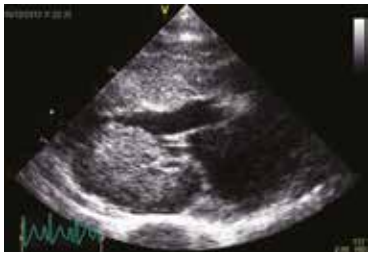
Results

Survival – Max diastolic wall thickness



p<0.001

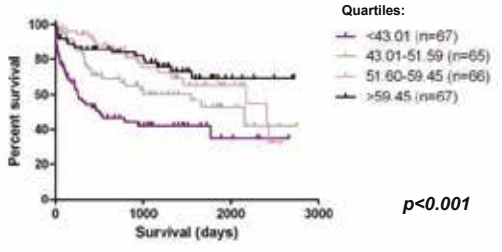
RVC



RVC

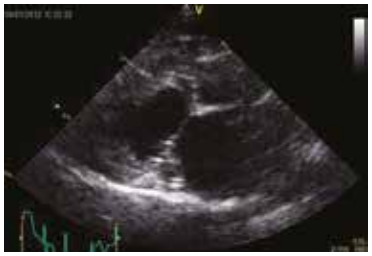
Results

Survival – Fractional shortening (FS%)



p<0.001

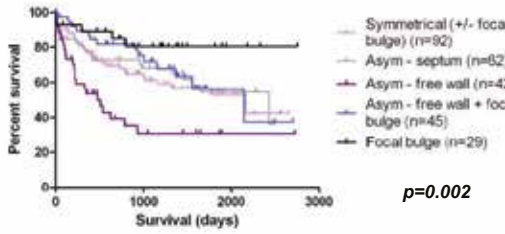
RVC



RVC

Results

Survival – distribution of hypertrophy

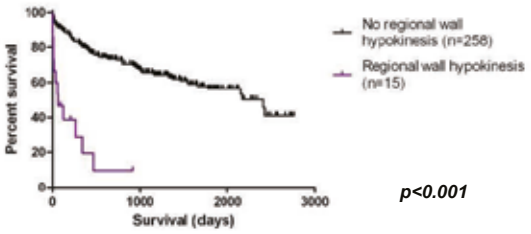


p=0.002

RVC

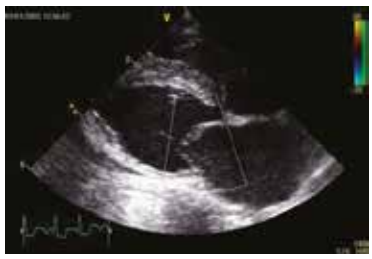
Results

Survival – regional wall hypokinesis



p<0.001

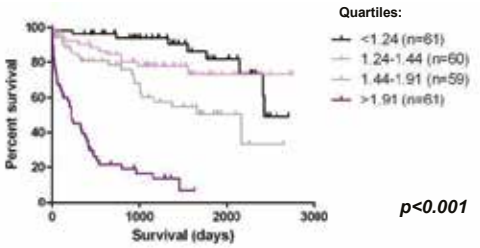
RVC



RVC

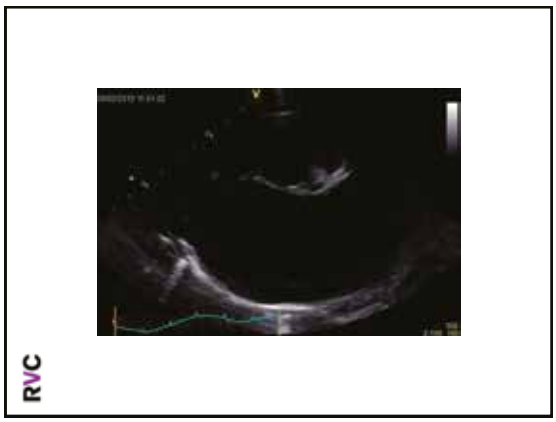
Results

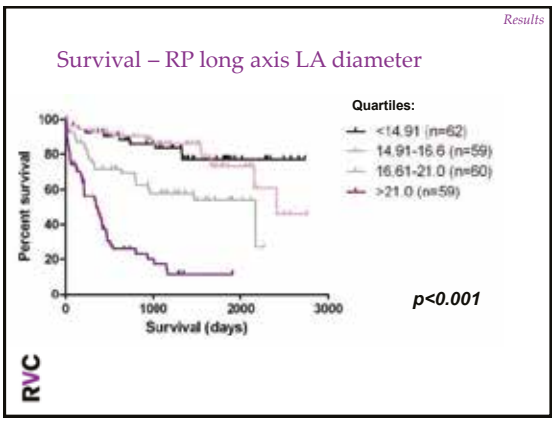
Survival – short axis LA:Ao

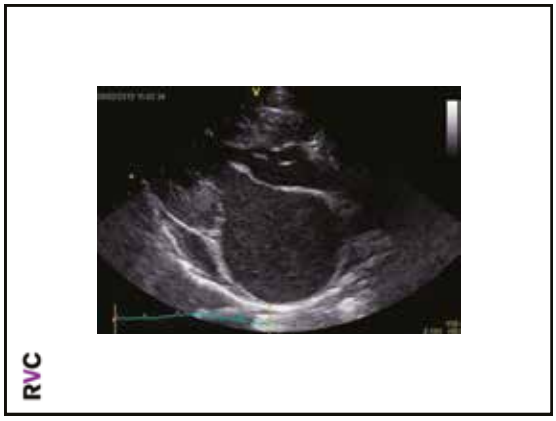


p<0.001

RVC


RVC


Results
Survival – RP long axis LA diameter
Quartiles:
<14.91 (n=62)
14.91-16.6 (n=59)
16.61-21.0 (n=60)
>21.0 (n=59)
Percent survival
Survival (days)
p<0.001
RVC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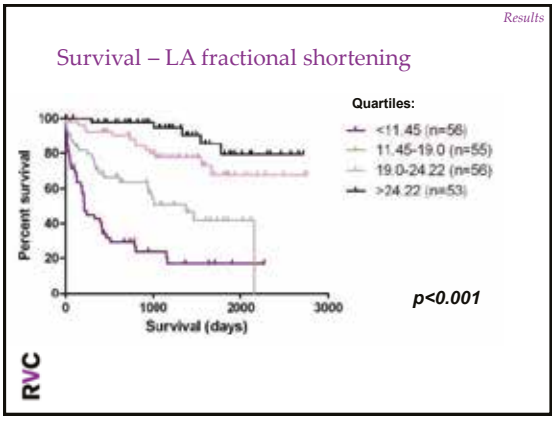
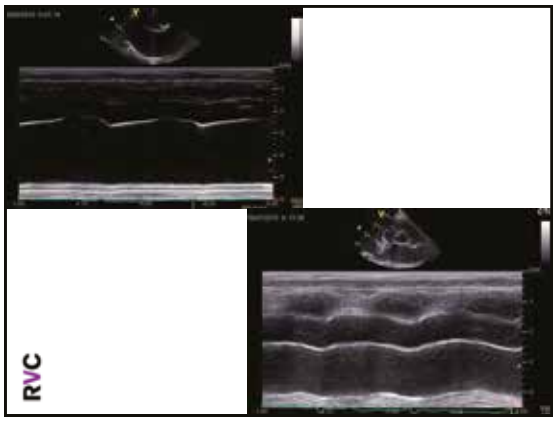

Results
Survival – LA fractional shortening
Quartiles:
<11.45 (n=58)
11.45-19.0 (n=55)
19.0-24.22 (n=56)
>24.22 (n=53)
Percent survival
Survival (days)
p<0.001
RVC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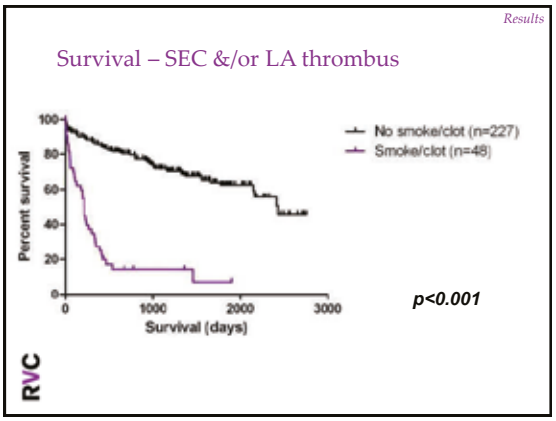
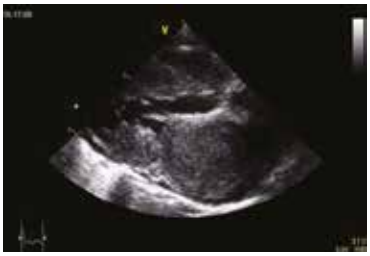

Results
Survival – SEC &/or LA thrombus
No smoke/clot (n=227)
Smoke/clot (n=48)
Percent survival
Survival (days)
p<0.001
RVC



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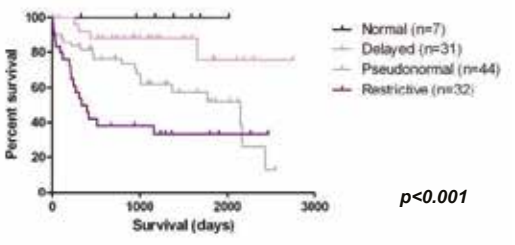
Results

Survival – Diastolic filling pattern



p<0.001

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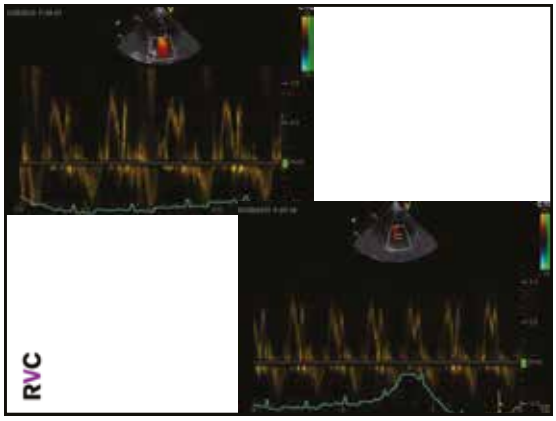

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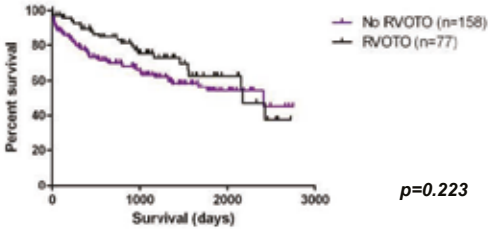
Results

Survival – Right ventricular outflow tract obstruction



p=0.223

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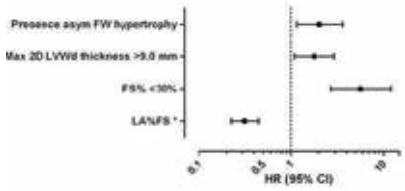
HCM survival study

Survival – multivariable Cox model



*for a unit increase of 10%

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Summary of the studies reporting prognostic capability of cardiac biomarkers in cats

Test	Number of cats	Conclusions	Citation	Study design
NTproANP	68 cats: 25 heart disease/CHF, 26 heart disease/NO-CHF, 17 controls	Increased NTproANP was associated with reduced survival time in univariable analysis, but lost significance in multivariable analysis when included with LA size[8]	Zimmering[17]	P;B;C;E
cTnI cTnT	36 cats with HCM (10 cardiac death) 23 healthy controls	cTnI and cTnT were both higher in non-survivors than survivors. cTnI correlated with LVFW thickness at diagnosis[36]	Langhorn[38]	P;B;E
NTproBNP cTnI	41 cats with HCM (21 cardiac death)	cTnI provided prognostic information, independent of heart failure status and the presence of left atrial dilation. NTproBNP was significantly associated with prognosis only if heart failure status or LA size was not accounted for[39]	Borgeat[39]	P;B;E

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Treatment of feline acute heart failure

David Connolly
BSc BVetMed PhD CertVC CertSAM DipECVIM (cardiology) MRCVS

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Royal Veterinary College
University of London

Management of Acute Heart Failure in the cat

Definitions

Acute heart failure is defined as the rapid onset of signs secondary to cardiac disease.

It may occur with or without previous cardiac disease. A or A on C

It is often life threatening and requires urgent treatment.

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STRESS in CATS With HCM

- Many Cats with occult HCM live on the edge of CHF
- They can be stable for month to years but then acutely decompensate into fulminant CHF
- Stressors include – new diaereses e.g. Hyper t4, cat bite abscesses
- New environmental stress – new cat in household etc.
- Avoid too many re-exams

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Potential Causes of AHF

- Decompensation of pre-existing CHF due to myocardial disease
- Infectious valvular disease
- Acute arrhythmia
- Cardiac tamponade
- Volume overload due to congenital disease
- High output syndromes (anaemia, Hyper T4)

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Medical management of AHF

Respiratory rate is the most important parameter to constantly measure.

Remember cat with CHF generally do not cough.

Remember many cat present with pleural effusion.

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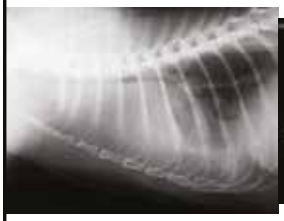
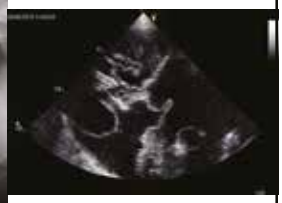
Thoracocentesis

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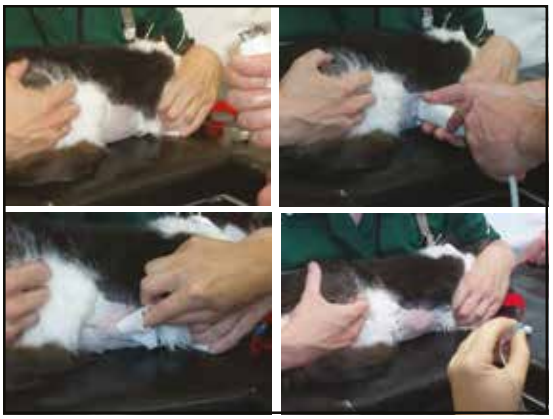

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Medical management of AHF

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Medical management of AHF

- T = Thoracocentesis ± pericardiocentesis
- O = Oxygen
- C = Contractility
- R = Rate and rhythm
- A = Afterload
- P = Preload

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Medical management of AHF

If and when the animal is sufficiently stable then measure

- Systolic blood pressure.
- Rectal temperature (cats).

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Acute heart failure -Feline HCM, no previous treatment

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Acute heart failure –Feline HCM, no previous therapy

- Resp rate >60 bpm ± crackles
- Sinus rhythm
- Systolic BP >120 mmHg

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Acute heart failure -HCM, no previous therapy

- **O_2** (45-60%)
- **Furosemide 1-2 mg/kg IV or IM q15/30mins,** repeated until resp rate decreases
- **[OR continue with Furosemide CRI 1mg/kg/hr]**

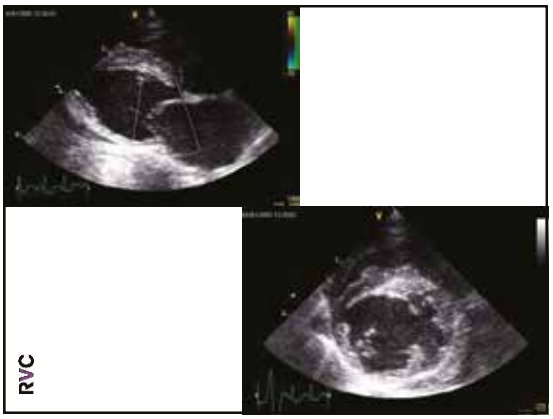
Acute heart failure -HCM, no previous therapy

- If unresponsive, or resp rate >80 bpm + crackles
- Systolic BP >120 mmHg

- **O_2** (60%)
- **Furosemide 1-2 mg/kg IV or IM q15/30mins,** repeated until resp rate decreases
- Consider Dobutamine/pimobendan if becomes hypotensive/cold

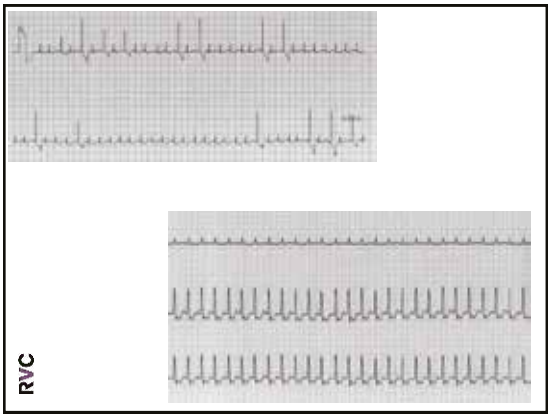
Acute heart failure –*HCM* **already on** *previous therapy*

- O_2 (45-60%)
- **Furosemide 3-4 mg/kg IV or IM q15/30mins,** repeated until resp rate decreases
- **Consider pimobendan if systolic dysfunction or infarct present**
- **Continue with other current Tx**



Acute heart failure -*HCM, arrhythmias*

- **AF** no treatment
- **V Tach** IV lidocaine (0.5mg/kg slow IV) – Sotolol
- **SVT** IV diltiazem - oral diltiazem



HCM: following response to initial therapy

- **Furosemide 1mg/kg IV** as needed
  AND
- **Furosemide PO** 1mg/kg q8-12 hours
- **Benazepril PO** (half dose if previously hypotensive?)
- **± Pimobendan**

HCM: discharge therapy

- **Furosemide PO** 1-2mg/kg q12 hours
- **Benazepril PO**
- **Antiplatelet therapy** (clopidogrel and or aspirin)
- **± Pimobendan**

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Thromboembolism

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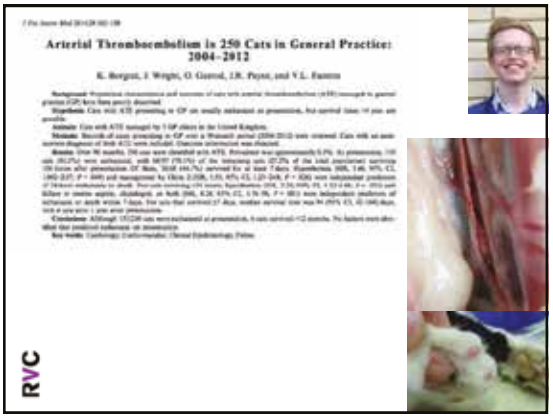

Arterial Thromboembolism in 250 Cats in General Practice: 2004–2012

K. Borgeat, J. Wright, O. Garrod, J.R. Payne, and V.L. Faunes

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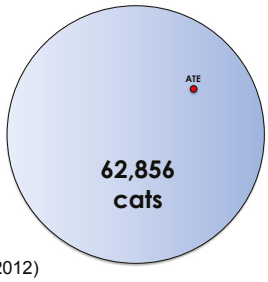
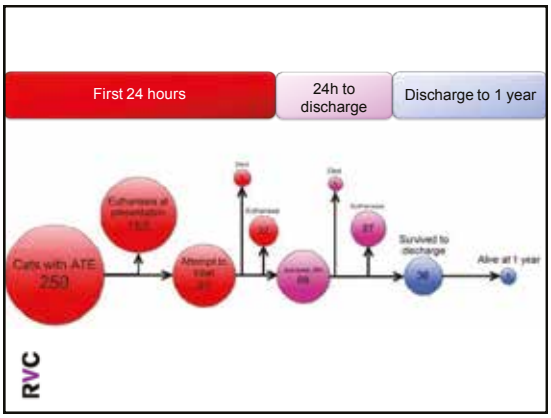
0.26% incidence arterial embolism over 8 years



•62,856 unique cats (2004-2012)
•166 cats developed ATE



Aortic thromboembolism

- Teat cardiovascular shock if present Dobutamine and/or pimobendan)
- ANALGESIA (opiods)
- Treat congestion if present (furosemide IV/IM/PO)
- Management of electrolyte/ acid-base disturbances (insulin/glucose)
- Heparin
- Physiotherapy
- Nutrition (PEG tube, pharyngostomy tube)

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Prevention of systemic thromboembolism

- Clopidogrel
- Aspirin
- Aspirin and clopidogrel
- LMW heparins
- ?Warfarin
- ?Pimobendan

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J Vet Cardiol 2015 Dec;17 Suppl 1:S306-17. doi: 10.1016/j.jvc.2015.10.004.
Secondary prevention of cardiogenic arterial thromboembolism in the cat: The double-blind, randomized, positive-controlled feline arterial thromboembolism; clopidogrel vs. aspirin trial (FAT CAT).
Hogan DF, Fox PR, Jacob K, Keene B, Laste NJ, Rosenthal S, Sederquist K, Weng HY
Abstract
OBJECTIVES:
To determine if clopidogrel administration is associated with a reduced likelihood of recurrent cardiogenic arterial thromboembolism (CATE) in cats compared to aspirin administration. Secondary aims were to determine if clopidogrel administration had an effect on the composite endpoint of recurrent CATE and cardiac death and to identify adverse effects of chronic clopidogrel or aspirin therapy.
ANIMALS:
Seventy-five cats that survived a CATE event.
METHODS:
Multicenter, double-blind, randomized, positive-controlled study. Cats were assigned to clopidogrel (18.75 mg/cat PO q 24 h) or aspirin (81 mg/cat PO q 72 h). **RESULTS:**
Clopidogrel was also associated with a significantly reduced likelihood of the composite endpoint of recurrent CATE or cardiac death (p = 0.033) with a longer median time to event [346 (95% CI 146-495) days vs. 128 (95% CI 58-243) days].
CONCLUSIONS:
Clopidogrel administration significantly reduces the likelihood of recurrent CATE compared with aspirin in cats; both drugs were well tolerated.

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Prevention of systemic thromboembolism

- Clopidogrel acts by inhibiting the ADP receptor on platelet cell membrane –reducing platelet function.
- Our favoured antithrombotic
- Dose 75mg SID
- Used:
  - Enlarged LA
  - Reduced atrial systolic funtion
  - Enlarged LA + spontaneous contrast
  - Used in combination with aspirin where a thrombus is visible or following an ATE episode.

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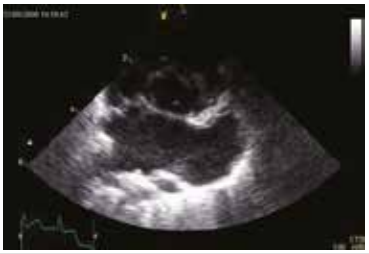
Prevention of systemic thromboembolism

- Clopidogrel – disliked by many cats
- Care with aspirin in cat with severe azotemia



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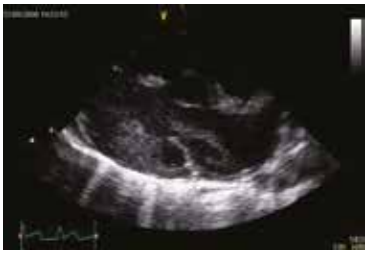

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Imaging

Frances Barr (UK)

MA VetMB PhD DVR DipECVDI MRCVS

Frances Barr graduated in 1979, and worked for four years in mixed practice before moving to the University of Bristol to specialise in diagnostic imaging. She was awarded the RCVS Diploma in Veterinary Radiology in 1985, and became a Foundation Diplomate of the European College of Veterinary Diagnostic Imaging in 1994. She was awarded the BSAVA Blaine award in 1994 for outstanding contributions to the advancement of small animal veterinary medicine or surgery, and the BSAVA Woodrow award in 1999 for outstanding contributions in the field of small animal veterinary medicine.

In 2010 Frances took on the role of BSAVA academic director, with the remit of developing the BSAVA Postgraduate Certificate qualifications. There are currently over 300 enrolled postgraduate students studying for these qualifications.

X rays and ultrasound for the investigation of the dyspnoeic cat

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BSAVA Head of Education
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Diagnostic imaging can play a crucial part in the investigation of the dyspnoeic cat. Such cats are in a fragile clinical state, and they can deteriorate rapidly if stressed by handling or treatment. It is therefore very important that cats with dyspnoea are placed in a calm environment, that handling is gentle and kept to a minimum, and that procedures are carefully planned.

An initial thoracic radiograph is often a useful screening tool. A cat which is struggling to breathe can usually be positioned in sternal recumbency on the X ray cassette with no, or minimal, restraint. Ideally the forelimbs and hind limbs should be placed symmetrically on each side of the body - but imperfect positioning in this situation is preferable to stressing the patient. A single dorso-ventral radiograph of the thorax allows an evaluation of:

- Thoracic wall injuries, including rib fractures
  - look for gas or radiopaque foreign material in the soft tissues
  - check each rib in turn to look for fractures
  - if rib fractures are seen, check for multiple segmental fractures which result in an unstable section of thoracic wall and a 'flail' chest
- Pneumothorax
  - radiolucent (dark) space separating the margins of the lungs and the thoracic wall
  - since the lungs are partially collapsed, they will appear more opaque than normal
- Pleural fluid
  - radiopaque (soft tissue opacity) space separating the margins of the lungs and the thoracic wall
  - fluid separates the individual lung lobes
  - the mediastinum may appear widened
- Diaphragmatic rupture, with abdominal viscera lying within the pleural space
  - the heart may be displaced from its usual position or obscured
  - look for gas or faeces filled intestinal loops in the thorax
  - check whether the stomach is in its normal position in the cranial abdomen
- Cardiac size and shape
  - normal heart size on the DV thoracic radiograph - the maximum width of the heart should not be more than 2/3 width of the thorax.
  - normal heart shape on this projection is oval; look particularly for an abnormal 'valentine' shape
- Lung collapse or consolidation
  - look for soft tissue opacity within lung lobes
  - look for volume loss (suggests the soft tissue opacity is due to collapse) or volume increase (suggests the soft tissue opacity is due to consolidation or a mass)
  - 'consolidation' suggests fluid or cells are within the alveoli; the fluid can be oedema fluid, inflammatory fluid, or blood and all have the same radiographic opacity

Subtle abnormalities can easily be missed on a single DV radiograph, but major abnormalities which require urgent treatment can usually be identified.

Thoracic ultrasound is an alternative quick screening tool for cats with dyspnoea. TFAST (Thoracic focussed assessment with sonography for trauma) is a rapid 5-point ultrasound examination of the thorax which was first developed for triage of trauma patients. Its wider use in the emergency situation is now well accepted[1]. The ultrasound transducer is placed

- at the chest tube site (right and left). Check particularly for pneumothorax or for consolidated lung. Normally the lung is aerated and the surface of the lung lobe slides to and fro under the thoracic wall during respiration. With pneumothorax this respiratory movement is lost.
- at the pericardial site. Check for pleural or pericardial fluid and perform a rapid screen for cardiac disease. Fluid appears echolucent (black), although echoes may be seen with highly cellular fluid or debris. Pericardial fluid surrounds the heart but remains contained within the sac of the pericardium. In cats it is more common to see pleural fluid, which surrounds the heart, and surrounds and separates the lung lobes.
  A rapid screen of the heart includes evaluation of atrial size and the thickness of the left ventricular wall, as well as a subjective assessment of cardiac movement
- at the hepatico-diaphragmatic site. Check for pleural or pericardial fluid.

TFAST is traditionally performed with the patient in lateral recumbency, but in the dyspnoeic cat it is often better to keep the patient in sternal recumbency.

In a dyspnoeic patient, if the chest appears to be normal on radiography and TFAST, it is important to consider an upper respiratory cause of the dyspnoea. Indeed the respiratory pattern and respiratory noise found on initial examination may allow you to focus on the upper respiratory tract from the outset. Bear in mind that cats are nasal breathers and may become dyspnoeic due to nasal or naso-pharyngeal disease. Dyspnoea may also arise due to laryngeal or tracheal disorders.

It is rarely possible to achieve an X ray of the head and pharynx in the conscious patient which is of diagnostic quality. Therefore radiography should be part of a planned investigation under general anaesthesia. A lateral projection of the head and pharynx allows evaluation of the pharynx and larynx, although this may be limited if an endotracheal tube is in place. If the neck is included in the field of view, the cervical trachea can also be assessed. Remember to check the soft tissues of the neck for evidence of emphysema - free air tracking within the soft tissue planes and under the skin.

Specialised radiographic projections are required to view the nasal cavities.

Ultrasound is of limited benefit for evaluation of the upper respiratory tract, but can be useful for evaluation of the larynx. The transducer can be placed on the ventral aspect of the larynx and if there is mucosal swelling or a discrete mass reducing the laryngeal lumen, this can be appreciated. Minimal restraint is required for this procedure and the cat can remain sitting or standing.

References

1. Boysen SR, Lisciandro GR. The use of ultrasound for dogs and cats in the emergency room: AFAST and TFAST. Veterinary Clinics of North America Small Animal Practice. 2013; 43(4: 773-797

X rays and ultrasound for the investigation of the coughing dog

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Coughing in the dog is commonly due to airway disease (either lower or upper airways) or to cardiac disease. Pleural space disorders (such as pneumothorax) less commonly cause coughing unless tracheal compression occurs. A careful history and a thorough physical examination often help to decide whether the cough is primarily cardiac or primarily respiratory. However in dogs with multiple concurrent conditions it can be difficult to be certain without further investigation.

X rays are an important part of the diagnostic plan for investigation of coughing in the dog. It is worth taking time to plan the X ray projections needed and taking care to ensure that all X rays are of diagnostic quality in order to get the maximum diagnostic information from the procedure.

- Ideally take thoracic radiographs under general anaesthesia, with the opportunity to fully inflate the lungs as each exposure is made. Lesions within the lung become much more obvious when surrounded by air filled alveoli (ie at full inspiration).
- A minimum of three projections should be taken; in right lateral recumbency, in left lateral recumbency, and in either dorsal or sternal recumbency. A dorso-ventral view (with the dog in sternal recumbency) is preferable if a cardiac cause of the cough is suspected. The heart then lies in a more natural position and shape is easier to evaluate. **Never turn an animal on its back for radiography if it is in respiratory distress.**
- If the general clinical status of the patient is poor or unstable, then it may be preferable not to use general anaesthesia. In such cases handle the dog gently and calmly, and take radiographs as near to full inspiration as you can. The lungs will not be fully inflated but major abnormalities may be seen; you need to be aware that subtle lesions may be missed.
- Radiographs of the head and neck may be useful in investigation of upper airway coughing. Lateral projections of the skull and pharynx should be carefully positioned to avoid any tilting or rotation. In particular, rotation of the skull around the sagittal plane results in obliquity of the pharynx, which makes interpretation of radiographs difficult.

When evaluating radiographs of the thorax in a coughing dog, it is important to check for:

- An abnormal lung pattern. In the normal dog, with fully inflated lungs, the pulmonary blood vessels are clearly seen. The larger bronchi, near the hilus of the lung, may also be visible as fine radiopaque rings or lines - which are parallel or gradually converging. It is usual for bronchi to become more prominent as the animal ages but the bronchial walls should remain thin.
  - An increased **bronchial pattern** results in more numerous bronchial lines and rings, extending also to the periphery of the lungs. The walls of the bronchi may be thickened, or may be ill defined as a result of peribronchial infiltration. An increased bronchial pattern is most commonly a result of inflammatory disease of the lower airways (bronchitis or bronchopneumonia). Occasionally the bronchi become dilated and the lumen can become irregularly sacculated; this indicates bronchiectasis, which is challenging to manage. It is important to remember that acute bronchitis can exist without radiographic changes.
  - An increased **alveolar pattern** occurs when the alveoli are no longer filled with air. Partial or total collapse of lung lobes results in an increased radiopacity of the affected lobe. An alveolar pattern also occurs if the alveoli become flooded with fluid (inflammatory, oedema, blood) or cells (neoplasia). Depending on the extent of alveolar disease, small ill-defined patches of soft tissue opacity appear, and then coalesce. The pulmonary blood vessels become obscured. The distribution of the alveolar pattern may help to narrow down the differential diagnoses. For example, cardiogenic oedema is most likely to be perihilar in distribution, while aspiration pneumonia is most likely to affect the cranio-ventral lung lobes.
  - An increased **interstitial pattern** results in a diffuse hazy opacity across the lung fields; sometimes the pattern can appear like a fine honeycomb or as tiny scattered nodules. An increased interstitial pattern can be the result of under inflation of lungs (radiographs taken on expiration, or lobes which are partially collapsed). However, this pattern can also indicate fluid accumulation in the interstitium (oedema), cellular infiltration or fibrosis.
- Enlarged lymph nodes. The lymph nodes at the hilus of the lung (the bronchial lymph nodes) are not usually visible on thoracic radiographs. However, if they become enlarged they may become visible as soft tissue masses at the hilus which may compress the trachea or the main stem bronchi.
- The size and shape of the heart. In the dog, a 'cardiac' cough is most often associated with left sided heart enlargement. A dilated left atrium may physically compress the main stem bronchi.

- The size and shape of the tracheal lumen. The lumen should be air filled, and of an even diameter. Abnormal opacities in the lumen can be due to a mass, foreign material, or mucus/exudate/blood. Narrowing of the lumen can be dynamic (eg tracheal collapse) or static (eg tracheal stricture).
- The upper airways. Check that the naso- and oro-pharynx are air filled. Check the relative positions of the soft palate, epiglottis and larynx. Check the integrity and alignment of the hyoid apparatus. Nasal disease in the dog can sometimes result in a cough but there are usually also other signs indicative of nasal disease.

Ultrasound has limited application in the investigation of a coughing dog where cardiac disease is not the cause, and X rays will normally be the imaging modality of choice. However, follow-up ultrasound may provide useful additional information. For example;

- If a lung mass is seen on X rays, and lies sufficiently close to the thoracic wall, it may be examined ultrasonographically to determine whether it is solid or fluid filled. Fine needle aspirates may be taken of the mass under ultrasound guidance.
- The presence of enlarged lymph nodes at the hilus may be confirmed. Enlarged retropharyngeal lymph nodes may also be identified and in this location can be aspirated under ultrasound guidance.
- Narrowing of the cervical trachea may be confirmed and the dynamic or static nature of the narrowing established.
- Ultrasonographic evaluation of the larynx may be useful. This can be carried out in the conscious dog by placing the transducer transversely on the ventral surface of the larynx. The arytenoid cartilages should be visible as symmetrical paired echogenic foci, which abduct on inspiration. In laryngeal paralysis abduction may be absent or asymmetrical.

When radiographs (in combination with historical and physical findings) are indicative of cardiac disease as a cause of the cough, ultrasound is a very useful addition to the investigation. Ultrasound allows the relative size of the 4 chambers of the heart to be assessed, and the thickness of the left and right ventricular walls to be measured. An evaluation of myocardial movement can be made. The structure and movement of the atrioventricular, aortic and pulmonary valves can be examined. The most common cardiac causes of coughing in the dog are mitral valve disease and dilated cardiomyopathy. Typical ultrasonographic features of these 2 conditions are as follows.

- **Mitral valve disease**.
  - Dilation of the left atrium.
  - Irregular thickening of the mitral valve leaflets. If Doppler is available, the a jet of mitral regurgitation from the left ventricle into the left atrium during systole may be visible
  - Myocardial contractility is usually normal or increased, except in the later stages of the disease when it may become reduced
- **Dilated cardiomyopathy**
  - Typically dilation of the left atrium and left ventricle. The wall of the left ventricle may also appear thinner than usual
  - Reduced myocardial contractility
  - Arrhythmias may be apparent, resulting in irregular and uncoordinated myocardial contractions. However an ECG is required to confirm this and to define the nature of the arrhythmia
  - Mitral valve regurgitation may be apparent if the dilation of the atrioventricular ring results in a failure of the valve leaflets to meet.

Further reading

BSAVA Manual of Canine and Feline Thoracic Imaging (2008). Eds. Schwarz T and Johnson V. ISBN 978 0 905214 97 9

X rays and ultrasound for the investigation of dysuria and haematuria

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A variety of terms are used to indicate abnormalities on urination. Dysuria is strictly pain on urination, but may be used more widely to suggest difficulty on urination. Stranguria denotes difficulty on urination, often with straining. Pollakiuria indicates increased frequency of urination. This talk considers dogs and cats exhibiting any of these clinical signs, with or without the presence of blood in the urine.

Difficulties in urination with or without the presence of blood are most often indicative of lower urinary tract disease - the bladder, prostate or urethra.

The bladder

Ultrasound is an excellent imaging modality for the bladder. Ultrasonographic examination can be performed in the conscious patient, with the patient either standing or in lateral or dorsal recumbency. A high frequency transducer (around 7.5 MHz) is placed in the caudal midline (or in a paraprepucial position in the male dog), with the sound beam directed dorsally and parallel to the spine to produce a sagittal image. Rotation of the transducer through 90^0 produces a transverse image. Care must be taken to sweep the sound beam throughout the whole volume of the bladder to ensure a thorough examination.

The normal bladder is a rounded, anechoic (black) structure with thin, well defined walls. It tapers caudally towards the urethra / prostate, forming the neck of the bladder. Bear in mind that the bladder is distensible, and size therefore is variable. If the bladder is very small, the walls of the bladder may appear subjectively thickened but this is not necessarily abnormal. In cats and dogs presenting with dysuria and haematuria it is important to check for:

- Thickening of the bladder wall. While the thickness of the bladder varies with the degree of bladder filling, wall thickness generally remains the same in all areas. If one part of the wall is significantly thicker than another then this indicates an abnormality. Thickening may be inflammatory or neoplastic. The site of thickening may suggest neoplasia is more (or less) likely. For example, irregular bladder wall thickening at the neck of the bladder is a common site for transitional cell carcinoma. However a definitive diagnosis is not possible without a tissue biopsy.
- Bladder wall masses. Focal masses may be neoplastic or polypoid. Occasionally an adherent blood clot may mimic a mass, and this is worth remembering in a patient with frank haematuria.
- Cystic calculi. Cystic calculi appear echogenic (white) on ultrasound, irrespective of the mineral composition. Acoustic shadowing is commonly seen deep to each calculus. Remember that calculi will settle in the dependent part of the bladder, so it is important to make sure that the sound beam sweeps through this part of the bladder during your examination.
- Other luminal material. A sediment of fine echoes may be found in the dependent part of the bladder; gently ballottement disturbs the sediment, which then gradually settles again. Sediment is not necessarily abnormal. Small, irregularly shaped structures of soft tissue echogenicity may float within the urine. These may be strands of mucus, blood clots, or collections of cellular debris.

A urine sample may be taken under ultrasound guidance for urinalysis and culture.

If ultrasound is not available, radiography may be used to evaluate the bladder. Survey lateral radiographs of the caudal abdomen will confirm the size, shape and position of the bladder. Cystic calculi may be seen but it's important to appreciate that radiopacity varies with mineral composition. Therefore struvite and oxalate calculi are radiopaque and should be seen on radiographs, but urate and cystine calculi are more variable in their radiopacity and may be missed.

Wall thickening / masses are not evident on survey radiographs as the soft tissue of the bladder wall and the urine within are of similar radiopacity. In order to evaluate the bladder wall it is necessary to perform a contrast study.

- Pneumocystogram. Empty the bladder of urine, and introduce room air via a urinary catheter until the bladder is moderately distended. Repeat the radiograph; air within the bladder lumen highlights the inner surface of the bladder wall and allows thickening or masses to be detected.
- Double contrast cystogram. Empty the bladder of urine, and introduce a small volume of water soluble, iodinated contrast medium. Roll the patient to ensure even distribution of the contrast, then introduce air into the bladder as above. Repeat the radiograph. A small pool of contrast in the dependent part of the bladder will highlight calculi or other luminal structures. The air highlights the bladder wall, but furthermore, the iodinated contrast may stick to any irregular or eroded areas of wall.

NB. If you plan to undertake radiographic and ultrasonographic examination of the bladder, do the ultrasound first. This ensures that the air of the radiographic contrast study does not interfere with the ultrasound examination.

The urethra

If the bladder appears to be normal in a dog or cat with dysuria/haematuria, it is important to evaluate the urethra.

Radiography is preferred to ultrasound for evaluation of the urethra. This is because a significant portion of the urethra (as it runs through the pelvis) is not easily seen with ultrasound.

Survey radiographs of the pelvic and perineal regions may be helpful in identifying urethral calculi. Comments regarding radiopacity of calculi (see above) remain relevant. Common sites for urethral calculi to lodge are proximal to the os penis (dogs), at the ischial arch (dogs and cats) and the tip of the penile urethra (cats).

Evaluation of the urethral lumen requires positive contrast urethrography. The patient is preferably under general anaesthesia or heavy sedation.

- In the male dog or cat, one option is to pass a urinary catheter into the bladder. Inject water soluble iodinated contrast medium through the catheter, at the same time withdrawing the catheter. Make the X ray exposure just before the tip of the catheter emerges from the penis. Alternatively place the tip of a small balloon catheter in the tip of the penile urethra in a male dog, inflate the balloon to hold in position, and inject the contrast medium. Make the X ray exposure as the contrast injection is made. A similar technique is possible in the male cat, with the catheter held in place within the penis by atraumatic clamps.
- In the female dog or cat, a urinary catheter can be placed in the bladder. Contrast is injected as the catheter is withdrawn, as in the male. If catheterisation is difficult then vaginourethrography can be considered. The tip of a catheter is placed inside the lips of the vulva, held in place with a balloon and clamps (dog) or clamps alone (cat). As contrast is injected, the vestibule and vagina fill first, and then the urethra. Make the X ray exposure towards the end of the injection.

Whatever the technique chosen, pay attention to radiographic safety of personnel.

The contrast column within the urethra should be smooth and continuous. Filling defects in the contrast may occur due to calculi - or to air bubbles. Irregularity in the outline of the contrast column is suggestive of inflammatory or neoplastic disease of the urethra.

The prostate

The shape, size and position of the prostate may be evaluated on survey radiographs; sometimes the prostate itself is clearly seen, but in other cases its outline may be inferred from the bladder position and the path of the descending colon/rectum. During contrast urethrography, contrast may spill into the prostate, indicating cavitating disease.

Ultrasound however provides more information about the prostate. The normal prostate is a smoothly rounded or bilobed structure lying caudal to the neck of the bladder. The parenchyma is finely speckled with an even texture. Check for:

- Abnormal enlargement and/or asymmetry. Asymmetry is often due to a solid or fluid filled mass. Enlargement alone may not be clinically significant, depending on age and neutering status.
- Fluid filled cavities within the prostate. Small fluid accumulations may be normal, but larger cavities may be intraprostatic cysts, abscesses or the necrotic centre of a neoplasm. Sampling of the fluid under ultrasound guidance may help to differentiate these possibilities.
- Heterogeneity of the parenchyma. Echogenic (bright) foci may occur due to fibrosis or calcification. Darker foci may occur with small fluid accumulations. It is difficult to differentiate diffuse inflammatory disease and diffuse neoplastic disease from the ultrasonographic appearance alone, but fine needle aspiration and/or tissue core biopsy may be performed under ultrasound guidance.
- Enlargement of the iliac lymph nodes. These lie adjacent to the aorta at the level of the bifurcation.

X rays and ultrasound for the investigation of acute vomiting

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There are many causes of vomiting in the dog and cat, which may be broadly divided into primary causes (conditions which primarily affect the gastro-intestinal [GI] tract) and secondary causes (conditions which primarily affect other organs or which are systemic). It is therefore important to take a careful history and perform a thorough physical examination. In particular it is useful to establish that the signs described are due to vomiting, and not to regurgitation.

There will many cases where further investigation is not warranted - at least immediately. It may be entirely appropriate to manage the patient conservatively. However, if the vomiting is severe and/or persistent and the patient is unwell, diagnostic imaging plays an important part in the plan for investigation.

Survey abdominal radiography is often a useful initial step. A right lateral recumbent projection allows an initial evaluation, although the orthogonal ventro-dorsal projection is necessary to confirm localisation of any abnormalities noted.

- Check for presence of radiopaque foreign material within the GI tract. Remember that the presence of foreign material does not necessarily indicate obstruction.
  - Foreign material in the stomach is common in dogs, and may be vomited or pass on uneventfully. Foreign material which remains in the stomach may result in intermittent vomiting.
  - Foreign material in the colon will usually pass on uneventfully
  - If foreign material is located in the small intestine [SI], it is important to look for radiographic evidence of obstruction (see below)
- Look for evidence of SI obstruction. Complete obstruction usually results in distension of SI loops proximal to the site of obstruction. SI distal to the obstruction will usually remain a normal diameter. It may be useful to compare the diameter of the dilated small intestinal loops with the height of the midpoint of the vertebral body of L5. A ratio >1.6:1 is suggestive of mechanical obstruction. Take care to identify the colon so that this is not mistaken for dilated SI.
  - An obstruction of the duodenum may be difficult to identify as there is only a very limited length of SI proximal to the site of obstruction
  - Remember that foreign material may be radiolucent and therefore difficult to identify (eg cloth).
  - Remember that dilation of intestinal loops may occur for reasons other than mechanical obstruction. (eg aerophagia, peritonitis, electrolyte disturbances). In such cases it is more usual for the SI to be distended throughout its length.
- Check the size and contents of the stomach; in a patient with vomiting the stomach is usually empty. Look for presence of formed faecal material in the colon; fluid contents may indicate associated diarrhoea.
- Look for evidence of free abdominal fluid. This is more readily detected using ultrasound (see below) but moderate to large quantities of fluid can be seen radiographically. Free fluid obscures the outlines of the abdominal viscera, resulting in a homogenous opacity throughout the abdomen. Remember that a lack of abdominal fat (eg a very young animal or a cachexic animal) can produce a similar radiographic appearance. However if abdominal fluid is present, a sample can be collected for analysis.
- Evaluate the radiographic appearance of other abdominal structures - in particular the liver, kidneys, bladder and (in the entire female) uterus.

Abdominal ultrasound is often preferred over radiography for imaging of the abdomen. It certainly has the potential to provide more information if a careful and systematic examination of the abdomen is made. It's important not to dismiss the value of radiography however, particularly for those not very experienced with the use of ultrasound.

A systematic ultrasonographic examination of the abdomen can be done with the patient in lateral or dorsal recumbency. This is largely a matter of personal preference, but should also take into account the comfort of the patient. Careful preparation of the scanning surface is helpful, and should entail clipping hair from the ventral abdomen, cleaning the skin, and applying liberal quantities of acoustic gel. The order in which structures are examined also varies between individuals. The author's personal approach in the vomiting dog or cat is as follows.

- Place the transducer caudal to the xiphisternum and angle the sound beam dorso-cranially to view the liver. Make a subjective evaluation of liver size. Check that the liver parenchyma is evenly granular in texture. Locate the gall bladder and check the contents are fluid and the wall thin and smooth. Look for evidence of distended hepatic veins or dilated bile ducts. Check for evidence of free fluid between the liver lobes or between the liver and the diaphragm.

- Slide the transducer caudally to examine the stomach. Check the contents of the stomach and pause to watch for peristaltic or segmental contractions. Identify the pylorus and follow the gastric outflow tract into the descending duodenum.
- Evaluate the pancreas. The right limb of the pancreas lies alongside the descending duodenum, while the left limb of the pancreas lies caudal to the gastric fundus. The normal pancreas can be difficult for the inexperienced user to find; ancillary ultrasonographic features indicative of pancreatitis include hyperechoic (very bright) fat in this region, and pockets of anechoic (black) fluid. The diseased pancreas itself tends to enlarge and become heterogeneous in echogenicity, but it is not usually possible to differentiate between inflammatory and neoplastic disease.
- Move the transducer to and fro over the ventral abdominal surface to examine the small intestine. Look for abnormal distension (which may be gas or fluid) and search for evidence of foreign material. Foreign material generally has an echogenic appearance, and may result in acoustic shadowing of the deeper tissues. Check for evidence of abnormal gathering or pleating of intestinal loops, which may occur around linear foreign bodies. Watch for peristaltic contractions. Nb. It is probably easier for the inexperienced user to identify an intestinal foreign body on a radiograph than by using ultrasound. However the advantage of ultrasound is that foreign bodies can be localised whether or not they are radiopaque.
- Check the small intestinal wall. Normally the walls have a layered appearance, and are uniform in thickness. Thickening and/or loss of layering of a section of wall is abnormal, and suggestive of inflammatory or neoplastic disease.
- Identify the colon and decide whether the contents are fluid or formed.
- During the evaluation of the small and large intestine, look also for evidence of free abdominal fluid. If only small pockets of free fluid are present, ultrasound can be used to guide a needle to collect a sample for analysis.
- Check the lymph nodes at the root of the mesentery. It is common for these nodes to be prominent in vomiting patients, but reactive nodes usually remain a normal smooth, elongated shape. If nodes are very large and rounded, perhaps forming clusters, consider the possibility of neoplasia.
- The kidneys can be examined from the ventral abdomen (mid to cranial abdomen, each side of the midline) or from the flank. The normal kidneys are smooth in outline and oval or bean shaped. The internal architecture is characteristic, with cortex, medulla and renal pelvis clearly defined. It's important to be aware that the kidneys may look entirely normal in acute renal disease, but chronic renal disease (with acute decompensation and vomiting) is often associated with renal fibrosis, which obscures the normal internal architecture.
- Move the transducer caudally to check the bladder. Make sure there is no abnormal bladder distension which might suggest urinary retention or obstruction. In the female dog or cat, check dorsal and cranial to the bladder for evidence of fluid distension of the uterine horns, indicative of pyometra.

Diagnostic imaging can provide essential information in the investigation of the patient with acute vomiting. However, the results should always be considered in conjunction with the findings from the history and physical examination, and the results of other diagnostic tests - and not in isolation.

Further reading

BSAVA Manual of Canine and Feline Abdominal Imaging (2009). Eds. O'Brien R and Barr F. ISBN 978 1 905319107

Keynote lecture

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Emerging canine and feline vector- and snail-borne diseases in Eastern Europe

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Canine and feline medicine is among the most dynamically advancing field of clinical veterinary medicine. New diagnostic and therapeutic techniques become progressively available worldwide, mainly due to scientific development and economic growth in certain countries, together with an increased number of companion animals. Additionally, an increased trend in companion animal movement (tourism, international adoptions, pet trade) associated with climatic and land-use changes have led to emergence of new diseases in certain territories and/or significant or even dramatic changes in the local epidemiology. Vector-borne and snail-borne diseases are typical examples of emerging diseases and due to the local ecological conditions, historical and socioeconomic context, Eastern Europe represents a textbook example. Moreover, an increased awareness among owners and clinicians has led to new findings even in case of diseases historically present but so far neglected.

The lecture will discuss several examples of vector-borne diseases (hepatozoonosis, cytauxzoonosis, babesiosis, filariases, thelaziosis, leishmaniosis, borreliosis) and snail-borne diseases (canine and feline lungworms), their local epidemiology and risk factors and their implications in the clinical approach in small animal patients.

The lecture will discuss also the diagnostic approaches to canine and feline vector- and snail-borne diseases, with their advantages and limitations.

Management

Nadia Kostadinova (BG)

DVM, MBA

Team management and motivation

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"Teamwork makes the dream work"

Every veterinarian has a dream, especially if you own the business that you work in. This dream is to come at work and find a happy, relaxed, concentrate in their work people. Not just a group of people, but a team. Your team.

There are moments and days when you simply think – "this is impossible", and then there are moments when you are proud of your team.

The definition of a team:

The team is a group that has a common goal in which the contribution of individuals is complementary to each other.

No man can be perfect, but one team can be.

The differences between a working group and a team are:

Working group:

- Members are gathered together for administrative reasons only. The work of each member is independent and linked to the end of working hours.
- The members are not involved in the planning of common goals. They look to work from the position of "leased labor"
- Members are told what they should do and what should be the best approach in their work.
- Expressing an opinion or disagreement is considered an obstacle that is not contributing to the work
- The members are too cautious in what they say, so true sharing and understanding are impossible
- Members can participate in training related to their work, but the application of what has been learned in practice is temporary
- Conflicts between members are solved by the leader, and this is done after the conflict has grown in size and influence
- Members do not participate in decision-making

Team:

- Members are independent and accept the independence of others. Time is not wasted in „wrestling for something" or in achieving personal success at the expense of others
- Among the members there is a feeling of community and partnership because they have committed themselves to the goals by taking part in their placement
- Members help and contribute to achieving goals by using their unique talent and knowledge
- The open expression of ideas, opinions, and disagreement is encouraged. Questions are welcomed
- Communication between members is frank and sincere. They make an effort to understand the other's point of view
- Members are encouraged to develop their skills and training and implement them in practice. They receive support and assistance from the team.
- Members accept the conflict as a normal part of human relationships. They try to resolve the conflicts in a timely and constructive manner
- Members are actively involved in discussing decisions, recognizing that the team leader is the one who takes the final decision

What is motivation?

Motivation is what makes a team successful. Motivation is a process, not a one-time event. Therefore, motivating factors and effects require constant monitoring, analysis and evaluation. To check your understanding of employee motivation, decide what you think about these statements:

1. Motivation is all about what you say and do to your employees. True or false?
2. Hire enthusiastic self-starters and you won't need to worry about motivation at all. True or false?

Answers: 1 – false; 2 – false

The five factors of motivation are fulfillment of basic needs, a healthy work environment, security, knowledge and ability to do the job, and knowledge of the practice's employee policies and procedures. These elements must be present before anyone can become motivated.

Management expert Frederick Herzberg says the factors that contribute to job satisfaction fall into the following categories: achievement; recognition; the work itself; responsibility; advancement; growth.

Money isn't everything! At least six factors outweigh salary:

1. Agreeable working conditions
2. Recognition
3. Fringe benefits
4. Lifestyle
5. Challenge
6. Personal fulfillment

Motivational techniques

Encourage, recognize and reward. Offer competitive salaries. Appropriate benefits – make a benefit package. Consistent and frequent rewards. Making people feel important. Getting people involved – according to Herzberg, the best form of motivation is the kind that gets people involved with what they do, making them feel that their efforts result in worthwhile contributions to the organization.

Action Items for Staff Motivation and Teamwork

1. Hire quality people who demonstrate a service-oriented attitude
2. Talk with each of your employees to understand what motivates each one to perform effectively
3. Write accurate, realistic job descriptions based on the tasks that have to be accomplished in your practice
4. Provide challenging work, and encourage career development
5. Offer competitive salaries and benefits that meet your employees needs
6. Provide ongoing recognition, appreciation, and participation in decision making
7. Keep communication flowing, formally and informally
8. Empower your staff to make decisions and to contribute to the practice in meaningful ways
9. **Take time for fun!**

A good manager must plan, organize, lead and monitor the use of human and material resources in his practice. This is how the time you spend in the clinic should look like:

- Administrative work ~ 10%
- Commercial Management ~ 10%
- Customer Relationships ~ 20%
- Clinical work ~ 20%
- **Training, motivating, monitoring and evaluating the team (veterinarians and other staff) form the rest of the time, up to 60%**

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Dr Goran Cvetkovic (SR)

DVM, MBA

Dr. Goran Cvetkovic received his veterinary degree from the Veterinary Faculty University of Belgrade, Serbia in 1997. Until 2002 he worked in several practices. Afterwards he finished his DVM theses at Vetsuisse Faculty in Zurich based on 1-year reasearch project: "Inhibition of endogenous sources of arginine in arginine depleted dogs". From 2004 until 2007 he studied business GSBA in Zurich and at University of Marylend. In 2007 he joined KYON AG in Zurich as CMO. In addition, Goran is leads a team of eleven people at Mediport doo, Serbia and is an avid reader and dedicated writer and speaker.

How healthy is your clinic?

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Every veterinary practice is a business (provided it is not a nonprofit organization).

Like any other business, the veterinary clinic is constantly facing problems, risks and opportunities for growth. The capability of practice management is reflected in its vigour to diagnose problems, to anticipate risks, and to use the most profitable growth paths for its practice.

In this lecture we will learn together how to determine the health of your clinic for all business aspects - finance, strategy, marketing, operations, HR, knowledge management and leadership by using an effective and precise diagnostic tool for small and midsize businesses.

The result will be a list of the most important problems, risks and growth opportunities in your practice.

THE BUSINESS DIAGNOSTIC TOOL

Financial health

1. Revenue, ATV, APF
2. Gross margin, net margin
3. Expenses
4. Cash flow, liquidity and solvency
5. ROIC and NPV

Business operational health

1. Processes
2. Meetings
3. P&D
4. A&L
5. Infrastructure

Strategic health

1. Life cycle
2. Vision & purpose
3. Core values
4. Constraints
5. Blue ocean

Marketing

1. Customer segmentation
2. Ideal customer
3. Offering
4. Lead generation
5. Lead conversion

People and knowledge

1. Hiring
2. Growth
3. Appraisal
4. Job description
5. Underperformers

Leadership

1. Types of leaders
2. Openness and vulnerability
3. Results orientation
4. Popularity or accountability?
5. Harmony or conflict?

By answering these thirty questions, a very clear and actionable picture of the business side of your clinic will emerge.

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1. **Valuation**, Measuring and Managing the Value of Companies, McKinsey & Company Tim Koller, Marc Goedhart, David Wessels
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4. **Marketing Management**, Philip Kotler, Kevin Lane Keller
5. **Blue Ocean Strategy**, W.Chan Kim, Renée Mauborgne
6. **Playing to Win**, A.G. Lafley, Roger L. Martin
7. **Human Resource Management,** Sean R. Valentine, Robert L. Mathis
8. **Operations Management,** Lee J. Krajewsky, Larry P. Ritzman, Manoj K. Malhotra

Business tools – is common sense all what we need?

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Once we have identified a precise list of the most important problems, risks and chances for growth, it is necessary to localize them in organs (business anatomy) and the functions of your practice (business physiology).

To thoroughly understand and to manage each of the identified problems, risks and opportunities, we need to learn a set of very effective analytical tools and frameworks.

Common sense is just not enough since a big number of pitfalls and potentials are counterintuitive and not easily visible to the untrained eye. The other main reason for using the frameworks listed below is that they will force us to take a systematic and holistic approach.

Veterinary diagnostics and business diagnostics have just so much in common.

TOOLS AND FRAMEWORKS

1. Business Anatomy
2. Business Physiology
3. Value Generators
4. Revenue Generators
5. Financial Statements
6. Strategy Discoverer
7. Marketing – One Page
8. Marketing – Buying Process
9. Marketing – Going To The Vet™
10. Marketing – Execution
11. Processes Definer
12. The Vision Organizer
13. Infrastructure Check

Once we have mastered these tools and frameworks it will be crystal clear what problems, risks and opportunities need to be focused upon, where they should be attacked and how they should be treated.

REFERENCES

1. **Valuation**, Measuring and Managing the Value of Companies, McKinsey & Company Tim Koller, Marc Goedhart, David Wessels
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8. **Operations Management,** Lee J. Krajewsky, Larry P. Ritzman, Manoj K. Malhotra

Business growth – a step by step guide to grow your clinic

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In this lecture, we will now apply all the tools and frameworks mentioned and discussed in the previous lectures, to a real practice in our region, which I helped turnauround from a very desperate position into a prosperous and profitable clinic.

THE LIVING CASE

Introduction

After my presentation at the first EEVC in Belgrade last year, a well known practice owner from our region approached me with the question of whether an MRI would be the right investment for his clinic. My answer was that any investment, not to mention one of this magnitude, needs careful analysis and considered review.

Analysis

An objective and disciplined deployment of The Business Diagnostic Tool into this clinic's setting has helped the owner and myself to compile a list of the most important problems the practice was encountering. None of those was the lack of a new MRI machine!

PRO™

Problems:

1. Stagnating revenue, increasing expenses
2. Poor strategic choices
3. Dysfunctional team

Risks:

1. Upcoming serious financial distress
2. Some of the key employees might leave imminently

Opportunities:

1. Excellent specialist knowledge – surgery, internal medicine, imaging
2. Flawless processes regarding delivery of the veterinary services

Recommendations:

1. Reconsider the current strategy
2. Bring the team back into alignment
3. Financial restructuring

Epilogue

A newly formulated strategy was presented to the team and to the bank partners.

Carefully instructed team members realigned their behaviours and activities with the new strategy. This has immediately started to create some momentum and results. These results have ignited a new enthusiasm, trust and energy...

After a short period of time the clinic has dramatically improved its approach to marketing and operational activities.

Very solid financial results have started to follow...

REFERENCES

1. **Valuation**, Measuring and Managing the Value of Companies, McKinsey & Company Tim Koller, Marc Goedhart, David Wessels
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Medicine

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Assistant Professor Bogdan Alexandru Vitalaru graduated the Faculty of Veterinary Medicine of Bucharest in 2004, PhD ever since October 2009, and he has participated since then in numerous national and international conferences, both as a doctor and as a lecturer. Since January 2008, he is an University Assistant and, from January 2016, Assistant Professor at the Faculty of Veterinary Medicine of Bucharest. Over the years he has written more than 60 scientific papers on topics related to oncologic surgery, dialysis, hemodialysis and emergencies. Since January 2014, in collaboration with BBraun Romania, within the Faculty of Veterinary Medicine, he opened the first Veterinary Hemodialysis Clinic in Romania and one of the few in Eastern Europe. Starting 2013, he established the Romanian Association of Veterinary Nephrology, Hemodialivet, whose Chairman he is. In the last 7 years, Dr. Vitalaru has been a lecturer for Hills Romania and one of the keynote speakers in Romania and Turkey regarding urology, nephrology and renal replacement therapies. He is also leading the emergencies Department of the Faculty of Veterinary Medicine of Bucharest since 2015. Starting 2014, Dr. Vitalaru has initiated a program of workshops and seminars regarding renal patients all over Romania (București, Iași, Cluj, Timișoara, Brașov, Constanța) and in Turkey (Istanbul, Ankara). He was awarded with the Continuing medical education prize - AMVAC/RoSAVA, Sinaia 12-14 November 2015 and SPEAKER OF THE YEAR - Romanian College of Veterinarians, Bucharest, 17 December 2015. He has written 3 books on emergencies in small animals and he is a member with some of the most prestigious publications in Romania: Cat life, 2015, Romanian Journal of Veterinary Orthopedics and Imagistic, 2015, Romanian Journal of Veterinary Medicine & Pharmacology, 2016. In 2016, Dr. Vitalaru has received the Special Prize for the best veterinary initiative of the year - Innovation in Health. The greatest achievements of his life are his two beautiful daughters: Natalia and Sofia.

Blood gases and electrolytes – key elements in critical renal patients

VIȚĂLARU BOGDAN ALEXADNRU
DVM, PhD

Understanding of acid-base and electrolyte chemistry and physiology is both an important and valuable knowledge base for all veterinarians, and especially those in emergency and critical care or specialty practice.

Assessment of acid-base status provides insight into three physiologic processes: alveolar ventilation, acid-base status, and oxygenation. Evaluating acid-base status has become an integral part of the emergent/critical care patient workup and should be performed as a baseline on all emergent patients. Deviation from normal acid-base balances is indicative of clinical disease processes and can aid the clinician in identifying underlying causes of illness in the patient. Venous samples can provide most of the information needed regarding acid-base status and even alveolar ventilation.

The balance of acid-base in the body is regulated by metabolic, respiratory, and renal pathways. In terms of acid-base discussion, generally either a metabolic or a respiratory derangement occurs with the renal or respiratory system compensating for either/both.

There are four primary acid-base disturbances that may occur in the body: metabolic acidosis, metabolic alkalosis, respiratory acidosis, and respiratory alkalosis. When evaluating a patient's acid-base status, the following parameters are primarily needed: pH, HCO_3 (or TCO_2), and pCO_2.

Typically the body's electrolytes are distributed intra- and extracellularly, and it is important to understand in which compartment they mostly reside. Sodium, the body's primary and most abundant cation, resides extracellularly. In addition, sodium's counterpart, chloride, and bicarbonate reside mainly extracellularly. The abundant intracellular electrolytes include: potassium, calcium, magnesium, and phosphorus/phosphate.

An ABG analyzer measures the pH and levels of both oxygen and carbon dioxide in the blood from an artery. This testing is used to determine acid-base balance, and also assess how well the patient's lungs are performing. The most important values in ABG analysis, utilizing the traditional approach, are pH, PCO_2 and HCO_3. Most blood gas analyzers directly measure pH and pCO_2, and calculate the HCO_3 concentration. CO_2 concentration results vary considerably depending on factors associated with sample collection, handling, timing, and the specific equipment used for analysis. Each of these factors is of critical importance and can affect the accuracy of pH and CO_2 measurements. Venous samples can also be used for blood gas evaluation, but there is a significant difference in the value for pO_2 between venous and arterial samples.

Anion gap (AG) is another useful tool for analyzing a patient's acid-base status. To maintain electroneutrality within the ECF, the sum of the concentrations of the positively charged ions (cations) must equal that of the negatively charged ions (anions). Cations that we typically measure are sodium (Na^+) and potassium (K^+). Other, often unmeasured, cations such as calcium (Ca^{++}) and magnesium (Mg^{++}) are present in much lower concentrations and are not used in the calculation of AG. Measured anions are chloride (Cl^-) and bicarbonate (HCO_3). There are many unmeasured anions, including blood proteins, phosphates, and sulfates. The AG is the difference between the measured cations and the measured anions.

Normal AG is 12–24 mEq/L in dogs and 13–27 mEq/L in cats. An elevated anion gap is usually a result of a decrease in bicarbonate levels and metabolic acidosis.

Early detection and treatment of any acid-base derangement is key. Since the respiratory system responds to metabolic derangements quickly, metabolic blood gas disorders are usually diagnosed once the compensatory response systems have already kicked in. If the underlying disease, as well as primary disturbance, is promptly and properly treated, the compensatory response and any other clinical signs should resolve accordingly. Prognosis varies and is ultimately dependent on the underlying cause and concurrent diseases present. Treating electrolyte abnormalities in conjunction with the acid-base disturbance and understanding compensatory mechanisms is a critical skill for diagnosis and treatment of any metabolic acid-base disorders.

Colloids vs crystalloids – key elements in critical renal patients

VIȚĂLARU BOGDAN ALEXADNRU
DVM, PhD

Many truths and myths exist about the role of intravenous fluids in prevention and management of kidney injury. Although there is a substantial body of literature about the impact of various management strategies in the kidney at risk, there is also a lack of definitive evidence for many of the assumptions that we as clinicians take for granted. There are some excellent reviews on the subject.

It is well documented that large volume 0.9% saline administration causes a hyperchloremic metabolic acidosis (HCMA). Hyperchloremia causes afferent renal artery vasocontstriction in an animal model, suggesting a possible role in kidney function.

Recently, growing interest has also focused on the role of bicarbonate alkalinization of the intravenous fluid administered.

The administration of fluid and the maintenance of plasma volume are most likely the only strategies that can reliably diminish the incidence of renal dysfunction in surgical patients, victims of trauma, and the critically ill. Aggressive crystalloid resuscitation in a variety of clinical circumstances may increase the risk of abdominal compartment syndrome and may thus impair renal function. As colloids maintain the plasma volume efficiently, they ought to be associated with a reduction in the incidence of renal failure. However, there is no conclusive evidence that this is the case and, under certain circumstances, it appears that the colloids may have a detrimental effect on renal performance.

Acute renal failure following the infusion of colloids was first described with the dextrans, notably dextran 40. A number of possibilities were considered to explain the renal injury including direct toxicity of the dextran molecule and the accumulation of hyperoncotic molecules within the plasma. The chemical toxicity of dextran is very low, but the occurrence of vacuoles in proximal tubular cells, referred to as osmotic nephrosis-like lesions, has been described. Although these lesions were initially thought to be responsible for impaired renal function, they have subsequently been described in association with a wide variety of intravenously administered substances,including mannitol, gelatins, and hydroxyethyl starches, as well as agents not associated with renal failure. This led to the concept of hyperoncotic renal failure, and the syndrome has been reported not only with dextrans but also with hydroxyethyl starch (HES), gelatin, and hyperoncotic albumin.

Neither albumin nor gelatin is thought to have any direct toxic effect on the renal tubules, but a variety of reports have raised the possibility that HES may be associated with renal injury.

There is a large body of literature demonstrating an absence of adverse renal effects with a variety of HES products, but particularly related to the tetrastarches.

Careful use of appropriate doses of all colloids solutions may offer some renal protection through optimal circulating volume expansion, and there is some evidence that HES solutions may protect renal endothelium to a greater extent than other colloids. There seems to be very little, if any, place for the use of hyperoncotic colloids for volume replacement. Wherever colloids are used for volume resuscitation, it is essential that adequate volumes of crystalloid are provided with a minimum of the daily water requirement being given as crystalloid solutions. Given the fact that all of the studies that have suggested possibility of an adverse renal effect of HES solutions, there is some basis for suggesting that HES may be best avoided in renal patients.

Many developments have occurred in fluid management in the last few years. Greater understanding of the harm of fluid overload is now appreciated, both in the surgical and critical care patient, and especially in those with oliguric AKI. It is now better understood that the risk of abdominal compartment syndrome and generalized edema are greater than was previously realized.

Elevation of abdominal pressures clearly can lead to a reduction in perfusion gradients of the gut and the kidney, further compounding previous injury. Paradigm shifts in fluid management with regards to saline-based volume therapy and HCMA are now occurring, especially when considering the type of fluid in renal patients. It is better to switch from saline-based volume therapy to balanced electrolyte formulations, even if they contain potassium.

The risk of HCMA is no longer being considered innocuous; however, actual adverse patient outcome has not been proven as yet.

The HES preparations were thought to have been implicated in AKI in these patients, but now it appears that the oncotic load of any colloid (including albumin) has a significant impact.

Finally, it is most important to consider basic understanding of physiology, pathophysiology, and pharmacology when using fluids in the critically ill patient.

Oncology

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Other: ESAVS Course Master Oncology and Supervisor for the ESAVS Master Programme; Part-time Business Mediator; Part-time Managment Trainer; Member of Toastmasters International.

Biopsies and tumour cytology – potential and pitfalls

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Simple Approach to Tumour Cytology

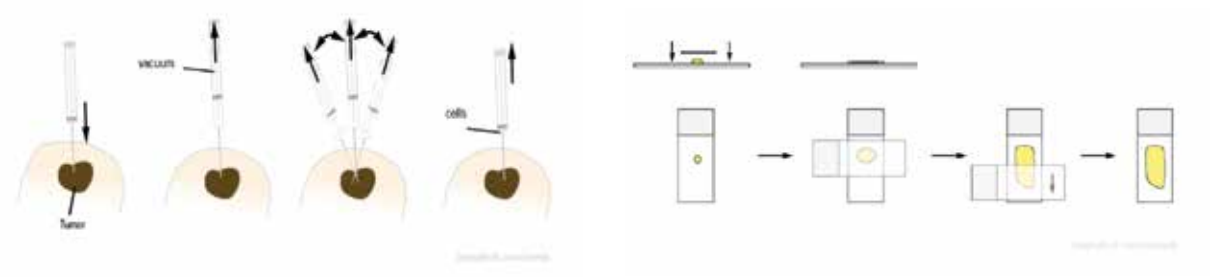
Cytology allows rapid, initial assessment of a suspicious lesion and helps narrow down the list of differential diagnoses. It is cheap, minimally invasive and does not require surgical skills. Cytological assessment of a mass involves retrieving cells from the mass (e.g. via fine-needle aspiration), preparing a smear, fixation and staining (in-house or at external lab) and microscopic evaluation (in-house or at external lab). However, the usefulness of cytology for diagnosis of a tumour depends on the quality of the sample, the type of tumour and the experience of the cytologist. *Have reasonable expectations!* Cytology is not a substitute for histology! In many cases cytology does not allow for a specific and/or definitive diagnosis, and histology may be required. However, often cytology helps ruling out important differential diagnoses!

Pros of cytology: Minimal equipment needed; minimally invasive. Often no sedation/anesthesia required. Rapid (no surgical prep needed in most cases). Cheap. Repeatable. High level of client acceptance. Allows ruling out important differential diagnoses.

Cons of cytology: Sometimes non-diagnostic. Often only a presumptive diagnosis and no *definitive* diagnosis. Often grading is not possible, but requires histology. Limited number of tissue stains. Limited immunocytochemistry. One cannot assess tissue architecture (e.g. invasion). Potential risks of bleeding, trauma (e.g. pneumothorax when aspirating lung lesions). Potential risk of tumour seeding.

Aspiration cytology

For tumour aspiration a 5 or 10 ml syringe, needles (size 20-23 G) and microscope slides are used. An aspiration pistol („Cyto-gun") can be useful. After aspiration the needle is removed from the syringe, air is loaded into the syringe and the needle is reattached. Afterwards the material inside the needle is expelled onto a glass slide and a smear is produced with a second slide or a cover slip (see below). When obtaining cytologic samples without aspiration (capillary method), the needle is inserted (with no syringe attached) into the mass and forwarded and retracted in the tumour several times, thus "punching" out material. After retraction from the tumour, the needle is then attached to an air-filed syringe and the material expelled onto a glass slide.



For preparation of a smear a second slide is carefully placed on top of the slide with the material and, after the drop has doubled in size from the surface tension between the slides, pulled apart without "squeezing" the cell material.

General Approach to Cytological Interpretation

1)Is the sample potentially diagnostic or is it a non-diagnostic sample (e.g. insufficient or destroyed cells or excessive blood contamination)? 2) Identify the types of nucleated cells present. 3) Does it look normal or abnormal? 4) Is there evidence of inflammation? 5) Does it look cystic? 6) Does is look neoplastic? 7) Is it epithelial, mesenchymal or round cell? 8) Is it benign or malignant?

General cytology of epithelial, mesenchymal and round cell tumours

Epithelial tumours (e.g. adenoma, carcinoma) are typically large, oval to pleomorphic (not spindle shaped) cells in clusters with distinct cell borders. Exfoliation is typically good. Malignant mesenchymal tumours (sarcomas) are classically spindle-shaped, individualized cells with indistinct cell borders. Cell exfoliation is mostly poor.

Round cell tumours (lymphoma, mast cell tumour, plasmacytoma, histiocytoma, transmissible venereal tumor, (melanoma)) are round, individualized cells that can contain vacuoles or granules. Exfoliation is typically excellent.

Cytologic diagnosis of malignancy

While benign tumours tend to be monomorphic and resemble the tissue of origin, malignant tumours are pleomorphic and have criteria of malignancy. Some tumours are invariably *potentially* malignant regardless of cytological appearance.

Histologic biopsies

The prerequisite for a correct tissue diagnosis is a representative sample. For this, there are four general rules: First, one has to choose a *representative part of the mass*. The more samples from different areas of the tumor are taken, the higher is the diagnostic yield. Avoid ulcerated surfaces and necrotic tumor centers. Invasiveness can be assessed best in the periphery of a tumor mass. Second one has to choose the *appropriate biopsy instrument and technique (*depends on tissue type and location of the tumour). In general, the least invasive technique is chosen. Diagnostic imaging may aid in finding the appropriate sampling location. Third, one has to avoid *contamination of the surrounding tissue* with tumor cells. Also, any biopsy tract must be removed during definite resection. Fourth, biopsies must be taken carefully as not to squeeze or tear the tissue with your instruments. Never use electrocautery for the excision of biopsy tissue.

Biopsy techniques:

1. *Superficial tumours* can be biopsied with a *skin biopsy punch*.
2. *Tru-Cut needle (e.g. 14G needle) is* the most common instrument for subcutaneous or abdominal tumours. Although designed for single use, Tru-Cut biopsy instruments can be disinfected or gas sterilized and reused.
3. *Incisional biopsy* is defined as taking a wedge-shaped piece of tissue from a tumour using a scalpel blade. This usually requires general anaesthesia. Since a larger piece of tissue is obtained, a more reliable diagnosis is possible.
4. *Excisional biopsy* is defined as the complete removal of the tumor with subsequent pathological examination. It is performed far more often than indicated. It should only be employed as long as the extent of the surgery would not change if one knew the histological tumour type (e.g. pulmonary or splenic tumors).

Location-specific biopsy methods

Biopsies of nasal tumours

Once a nasal tumour is suspected based on rhinoscopy, radiographs or CT, biopsy is indicated to assess its histological type (e.g. carcinoma vs sarcoma vs lymphoma).

While cytology (cyto brush, impression smears, FNA through lytic bone) can be used to diagnose nasal tumours, due to concurrent inflammation the false negative rate can be high. Prior to taking biopsies, please ensure adequacy of the coagulation system first (platelet count, PT, aPTT) and intubate the patient (to prevent blood aspiration). Biopsies can be taken normograde (through the nares), retrograde (via the choanes), or through the nasal dorsum. *Normograde biopsy* is the preferred technique most tumours. The biopsy is typically taken under direct visualization during rhinoscopy or CT-guided or "blindly" with alligator forceps. The biopsy instrument should never be introduced beyond the level of the medial canthus of the eye due to the risk of penetration the cribriforme plate.

Bone biopsy

For sampling bone lesions a small gauge bone trepan (3-5 mm) or a Jamshidi-Needle can be used. Please bear in mind that osteosarcomas arise from the medullar cavity and they are usually surrounded by a thick layer of reactive bone, cartilage and soft tissue. Therefore, if the biopsy is not deep enough, false negative results are common. Also, in contrast to most other tumor types, biopsies from bone lesions should be taken from the *center* of the lesion.

Bone marrow biopsies

Bone marrow biopsies are useful in the workup or diagnosis of haematopoietic tumours or infectious diseases, to assess cytopaenias or abnormalities on the blood smear or in patients with unexplained fever, gammopathy or hypercalcaemia. Bone marrow cytology is more commonly assessed than histology. Biopsy requires a spinal needle or a 15-18 G needle (e.g. Jamshidi) with a mandarin. Good locations for bone marrow biopsy are the proximal humerus or femoral bone, the iliac crest, or the ribs. Bone marrow fluid coagulates rapidly so a smear must be made immediately.

Endoscopic biopsies (Gastrointestinal, respiratory, and urogenital tract, nasal cavity)

The advantage of these technique are the low degree of invasiveness, however, since samples are small and superficial, disease processes in the submucosal layer (eg. diffuse GI-lymphoma) can be missed.

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Canine and feline nasal tumours

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Summary

Neoplasia in the nasal cavity is an important differential diagnosis in an older dog or cat presenting with chronic sneezing and/or epistaxis. Although the vast majority of nasal tumours are malignant, local invasion and destruction are more common than distant metastasis. Treatment of choice is radiation therapy; however, rhinotomy combined with chemotherapy and/or NSAIDs may be an option if radiation therapy is not available.

Epidemiology

Nasal tumours are rare in dogs and cats accounting for approximately 1% of all tumours. However, they occur much more commonly than in man. Medium to large breed dogs and especially dolichocephalic breeds are more commonly affected by nasal tumours. The median age of onset is 9-11 years.

Tumour types and biological behavior

The vast majority of nasal tumours are malignant (>90%). The most common histological types are carcinoma (60% in dogs, 30-60% in cats), sarcomas (30% dogs, <10% cats) and lymphoma (<5% dogs, 50% cats). Other tumours (e.g. esthesioneuroblastoma, mast cell tumour, melanoma, plasmacytoma, transmissible venereal tumour (dogs), benign tumours (e.g. fibroma, osteoma, hemangioma) are rare. Nasal carcinomas typically originate from the caudal 2/3 of the nasal cavity and are locally invasive and destructive. At least 50% of nasal tumours are bilateral at presentation. Death is usually due to local disease progression, e.g. invasion of the cribiform plate or the retro-orbital space. Intractable or recurrent epistaxis may also be a reason for owners to elect euthanasia.

Metastasis is generally uncommon (<20%) at initial presentation, however, in end-stage patients metastases may be present in as much as 40-50% of patients with nasal carcinomas and 20% of patients with sarcomas. The most common sites of metastasis are the regional lymph nodes (i.e. mandibular and medial retropharyngeal) and the lungs. Nasal lymphomas may be confined to the nasal cavity (more commonly) or they may be part of a multicentric lymphoma. Paraneoplastic hypercalcemia and erythrocytosis have rarely been reported.

History and clinical signs

Nasal tumours usually cause non-specific clinical signs compatible with nasal disease, e.g. intermittent and progressive unilateral (or later bilateral) nasal discharge and/or epistaxis, sneezing or epiphora. Signs more suggestive of a nasal tumour are facial deformation or exophthalmos. Neurological signs (e.g. seizures) due to progression of a nasal tumour into the central nervous system are very rare at initial presentation. Clinical signs have been present for a median of 2-3 months prior to diagnosis. It is not uncommon to see transient clinical improvement upon treatment with NSAIDs or antibiotics.

Diagnosis

In an older dog with recurrent epistaxis a nasal tumour is the most important differential diagnosis. A variety of diagnostic imaging modalities exist to localize a nasal tumour. CT is probably the best option. Facial deformation or presence of osteolysis and mass lesions in the nasal cavity are suggestive of a tumour. Nevertheless, biopsy is necessary for confirmation of a tumour and to establish the exact histological type. Prior to biopsy coagulation testing (including platelet count, prothrombin time, activated partial thromboplastin time) are indicated.

Diagnostic Imaging Options for Workup of Nasal Tumours

Standard radiographs can show signs suggestive of a tumour: e.g. loss of trabecular structure and other evidence of osteolysis, increased soft-tissue opacities and osteolysis. Overall, however, it has low sensitivity and specificity. Superimposition of structures is a problem and the extent of a tumour cannot be accurately assessed. *Rhinoscopy* can directly visualize a mass. Biopsies can be taken under direct visualization. However, often visibility is poor due to secretions or blood. Also, one can only detect the surface of a mass and neither its full extent, nor if it is invasive. Endoscopic biopsies are generally only small (and potentially non-diagnostic). Rhinoscopy should be done after CT, otherwise iatrogenic bleeding may interfere with CT interpretation. *CT* has superior detail compared to conventional radiographs as there is no superimposition of structures. It is excellent for detecting osteolysis and to assess the 3D extent of the disease. Iodine contrast enhances soft-tissue contrast. It is faster than MRI. Biopsies can be guided by CT. It is essential for radiation therapy planning. Downsides of CT include the need for general anaesthesia, cost, availability, the inferior soft-tissue contrast compared to MRI and the inability to differentiate between different histological types of tumour. *MRI* provides good detail as there is no superimposition. It is better for soft-tissue detail than CT and better than CT to assess invasion into the central nervous system. Downsides of MRI include: it is not as sensitive for bony detail (osteolysis) compared to CT. It cannot differentiate between different histological types of tumour. Cost! Longer general anaesthesia compared to CT. Unless invasion into CNS is suspected, there is little extra benefit compared to CT.

Differential diagnoses

Differential diagnoses include chronic rhinitis, foreign body / trauma, dental disease (e.g. tooth root abscess), infectious disease (e.g. dogs: aspergillosis, leishmaniasis, ehrlichiosis, nasal mites (Pneumonyssoides Caninum), cats: aspergillosis, cryptococcosis), coagulopathy, hypertension, polyps (cats>dogs), naso-pharyngeal stenosis and others.

Biopsy options

Several biopsy options exist to establish a diagnosis. Complications include bleeding and accidental brain biopsy. The former is self-limiting in most cases. To avoid the latter, biopsy instruments should not go further caudal than the medial canthus of the eye.

1) Biopsy through the nares with *Alligator forceps (commonly used and most often the preferred method!), b*one curette, TruCut biopsy device, endoscopic biopsies and fine-needle aspiration. 2) Biopsy through lytic bones: TruCut biopsy device or fine-needle aspiration. 3) Nasal flushing with saline (=attempt to dislodge part of the mass): often non-diagnostic. 4) Through the nasal bone after drilling a 3mm hole: causing extra trauma. 5) Open surgical biopsies in the course of a rhinotomy

Staging

1. Palpation of regional lymph nodes plus aspiration cytology if enlarged
2. Thoracic radiographs or CT (however, it is rare to find distant metastasis at presentation)
3. CT to assess medial retropharyngeal lymph nodes (however, most often unremarkable)

Therapy and prognosis

The best therapeutic option depends on the size and stage of the tumour. With NSAIDs (e.g. meloxicam) alone median survival times are only 3 months. Generally, radiation therapy plus long-term oral NSAIDs (e.g. meloxicam at standard doses) is regarded as the treatment of choice for canine nasal carcinomas.

For carcinomas with a "curative" radiation protocol (16-19 small fractions, Mondays-Fridays)

median survival time ranges from 9-18 months (25% live >2 years). With a "palliative" radiation protocol (4-5 large fractions, 1-2x per week) median survival times of 5-10 months have been described. Surgical debulking (rhinotomy) is regarded as palliative and by itself does not improve survival times (median 3-6 months) compared to patients treated with NSAIDs. However, if radiation therapy is not available, surgical debulking (rhinotomy) followed by chemotherapy (e.g. carboplatin: 200-300mg/m^2 IV, every 21-28 days; total of 4-5 doses)) and/or NSAIDs (e.g. Meloxicam) may be an option. Another primary or adjuvant treatment option is a trail with tyrosine kinase inhibitors (toceranib, Palladia™ (Zoetis), 2.5-2,8mg/kg, PO, EOD, long-term). Unfortunately, toceranib is very expensive and only little data is available on its use in nasal tumours showing remissions in a few patients. If extensive disease is present, palliative therapy with NSAIDs may be the most suitable (palliative) option. Feline nasal lymphomas can be treated with radiation therapy or chemotherapy. Generally, radiation therapy is thought to be the superior option. However, multi-agent chemotherapy as for other forms of lymphoma (see handout on lymphoma) is a reasonable alternative. The prognosis is highly variable and depends on the treatment and several other factors.

Adverse effects of radiation therapy for nasal tumours

Acute adverse effects with a „curative" protocol are mainly inflammation (incl. blepharitis, conjunctivitis, keratoconjunctivitis sicca (KCS), oral mucositis, rhinitis, dermatitis, etc). Chronic adverse effects are rare with a curative protocol, but more common with a "palliative" (hypofractionated) protocol: e.g. cataracts, KCS, osteonecrosis, permanent alopecia, fibrosis of the skin, etc.

Negative prognostic factors in canine nasal tumours

Negative prognostic factors include: age >10 years, epistaxis at initial presentation, large tumours, metastases, squamous cell carcinoma or undifferentiated carcinoma, lack of remission, invasion through the cribiform plate and palliative radiation (compared to curative radiation).

Recommended reading

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Thyroid tumours

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Canine Thyroid Tumours

Epidemiology, history and clinical signs:

Thyroid tumours represent 1-4% of all canine tumours. It is the most frequent endocrine neoplasia. The median age of onset is 9-10 years. Clinically apparent cases are >85% malignant (carcinomas), the remaining tumours are mostly adenomas, but those are clinically silent nodules found on post-mortem as an incidental finding. Commonly affected breeds include boxer, beagle and golden retriever. Thyroid tumours typically present as a palpable pain-free mass at the cranial neck that was noticed by clients or vets. Less common are clinical signs such as coughing, swallowing problems, voice change, Horner syndrome, facial swelling (prae-caval syndrome). In case of rare hyperthyroidism: polyphagia, weight-loss, polyuria, polydipsia, etc. Very rarely thyroid tumours appear at ectopic location: anywhere from the base of the tongue up to the heart (neoplastic transformation of thyroid tissue scattered during embryogenesis).

	dog	**cat**
Incidence	rare	common
% malignant	80-90%	<5%
% hyperthyroid	<10%	>95%

Biological behaviour:

They typically originate from the follicular epithelium. 60% are euthyroid, 30% hypothyroid and 10% hyperthyroid. 10-45% of cases are bilateral.

Carcinomas grow more rapidly than the (rare) adenomas and they are potentially *locally invasive* into the trachea, larynx, carotid artery, jugular vein, etc. 33-50% are mobile, the rest is fixed to surrounding tissues (and typically non-resectable). 15-40% show metastases at initial presentation depending on tumour size (higher in tumours >5cm): esp. regional lymph nodes (retropharyngeal, mandibular, prae-scapular nodes) and lung. According to one study, bilateral tumours are 16x more likely to have metastases at presentation (but this has not been confirmed in other studies). The risk of metastases correlates with tumour size resp. volume: >15% in tumours <20cm^3, 75% in tumours 21-100cm^3 and 100% in tumours >100cm^3. Vascular invasion is negatively prognostic. There is a high risk of **tumour recurrence** in case of incomplete resection

Recommended diagnostic/staging tests:

- Palpation (mobile vs fixed? Unilateral vs bilateral?)
- Cytology from fine-needle aspiration. If non-diagnosticà incisional biopsy
- CT (size, invasion, metastases)
- T4/TSH, ionised calcium
- Assessment of lymph node metastases (retropharyngeal, mandibular, prescapular LNN): CT or ultrasound and potentially FNA & cytology
- Assessment of pulmonary metastases: radiographs or CT

Differential diagnoses:

1. Mucocele of the salivary glands
2. Abscess, foreign body granuloma
3. Lymphadenopathy (metastasis? E.g. from tonsillar squamous cell carcinoma)
4. Soft-tissue sarcoma
5. Other tumours (lipoma, hemangiosarcoma...)

	PROS	CONS
Palpation	rapid, good for assessment of mobility vs fixation (ideally under GA), but not always reliable	Presumptive diagnosis
X-rays (neck)	none	Waste of money
CT	Highly specific! Assessment of size, extent and invasion (MRI even better for assessment of invasion). Detection of mets	costs general anaesthesia
Ultrasound	Can help differentiate thyroid tissue from LNN. Can assess size & extent. Assess invasion (but inferior to CT or MRI)	Requires experience. Cost (requires >10 MHz probe). Not specific!
Cytology	Rapid, cheap. If diagnostic, one can often rule out other differentials (e.g. fat, LNN) and diagnose endocrine neoplasia	Often much blood contamination *Only approx. 50% diagnostic* Generally, cannot differentiate benign from malignant tumours
Incisional biopsy	Allows definitive diagnosis Histology = gold standard	Does not always allow assessment of invasion. Potential for severe hemorrhage. General anaesthesia Ideally sonographic guidance
Surgical exploration	Allows diagnosis and gold-standard for assessment of mobile vs. fixed.	Potentially difficult area to operate on (nerves, vessels...) Only useful if distant metastases have been ruled out.

Therapeutic options:

Therapy depends on the tumour size, invasion/mobility and the presence/absence of metastases.

Surgical resection:

Surgery is the treatment of choice if tumour is mobile. One can sacrifice unilaterally the recurrent laryngeal nerve, the jugular vein, carotid artery and both ipsilateral parathyroid glands (à unilateral Horner syndrome). Median survival is ~3 years following resection of mobile tumours. Median survival is ~6-12 months following resection of invasive tumours. Bilateral tumours: resection is possible but requires supplementation with thyroxin and possibly vitamin D and calcium.

Radiation therapy:

Radiation is (possibly) the best option for fixed (non-mobile) tumours (alternatively radioiondine treatment). Maximal effect is often only evident after several months (8-22 months). With „curative protocol" in one study with macroscopic carcinomas (12 fractions with 4 Gy per fraction; Mondays-Wednesdays-Fridays) there was a 80% progression-free survival after 12 months; 72% after 3 years. 50% had acute adverse effects (mucositis, dermatitis, coughing) for 2-3 weeks. Less than 20% have late effects (fibrosis of the skin, alopecia, chronic tracheitis). Hypothyroidism: up to 50% following radiation therapy. Local recurrence is approximately 25%. With a „palliative protocol"

(4 fractions, 9Gy per fraction (=36Gy) at weekly interval) in 10 dogs there were 9 partial remissions and 1 complete remission. Median survival time was 2 years.

Radioiodine treatment (^{131}I):

Potentially effective if the tumour takes up the iodine (median survival: 30 months).

Chemotherapy:

This is less effective, but may be useful in some: e.g. Doxorubicin: 30-50% response rate with macroscopic tumours, but short-lived and uncertain if survival benefit: median survival 33 weeks (~ 8 months). Alternatives are Carboplatin (Cisplatin), Mitoxantrone, Actinomycin D. The tyrosine kinase inhibitor Toceranib (Palladia®, Zoetis) achieved measurable remissions of macroscopic thyroid tumours and their pulmonary metastases in a few cases. NSAIDs: uncertain if effective. Probably not. But may have a place combined with metronomic chemotherapy. Metronomic chemotherapy (Leukeran (chlorambucil) 4mg/m^2/day PO): partial remissions and stable disease for several months have been reported in a few cases.

Post-operative thyroxine-Supplementation:

This is theoretically useful (blocking endogenous TSH secretion, which could act as a tumour growth factor).

Feline thyroid tumours

Feline hyperthyroidism is common, but carcinomas are rare: >95% adenomatous hyperplasia (>70% bilateral), <5% carcinomas (mostly unilateral). The vast majority are hyperthyroid. The median age is 13 years (only 5% are <10 years. Adenomas: 12.4 years, carcinomas: 15.8 years). Benign hyperthyroidism has a multifactorial etiology. Carcinomas may arise from long-standing benign disease. Treatment of benign hyperthyroidism can involve drugs (methimazole or carbimazole), surgical thyroidectomy, radioactive iodine (131I) or nutritional therapy. The prognosis is generally good. Carcinomas are usually larger than adenomas. They frequently show infiltration of trachea, larynx or esophagus. Metastasis occur in 40-70% (regional LNN & lungs). Features suggestive of thyroid carcinoma are 1) lack of response to high doses of methimazole or carbimazole (> 15-20 mg/day), 2) treatment failure or recurrence of hyperthyroidism after surgery or radioiodine treatment, 3) locally invasive thyroid tumor (at surgery), 4) signs of distant metastases on radiography or thyroid imaging (usually pulmonary). Histology is the only definitive way to confirm the diagnosis. Treatment is ideally done with high doses of radioactive iodine or sometimes with surgery. Prolonged survival times (181-2381 days) are possible.

Recommended reading

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Canine mast cell tumours

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Summary

Mast cell tumours are one of the most common and clinically important skin tumours in dogs. The clinical behavior is highly variable and can include local extension, metastasis and paraneoplastic disease due to release of vasoactive substances. Depending on the tumour grade and other prognostic factors a multimodal treatment approach is necessary.

Introduction

Mast cells (MC) originate from the bone marrow, but migrate to peripheral tissues (esp. skin, GI tract, lungs) where they differentiate and mature. MC are part of the body's immune system and involved in allergic and inflammatory reactions. MC have abundant cytoplasm containing a wide variety of vasoactive substances (e.g. histamine, heparin, etc.) stored in characteristic granules. The stored vasoactive cytokines can be released upon stimulation Neoplastic accumulations of mast cells are called mast cell tumours (MCT). MCT are one of the most common cutaneous tumours in dogs: up to 20% of all canine cutaneous tumours. MCT are usually solitary skin tumours. Clinical presentation is extremely variable: they may be ulcerated or pruritic; some even feel like *lipomas*. MCT show a highly variable biologic behaviour.

Most are clinically silent skin nodules, however mast cell degranulation and release of vasoactive substances can cause local or systemic/paraneoplastic reactions including swelling, pruritus, erythema, oedema, wound healing problems, local coagulopathies or even systemic complications (gastric hyperacidity and ulceration; hypotension, anaphylactic shock, vomiting/diarrhoea, melaena, systemic coagulopathies).

Diagnosis

Fine-needle aspiration cytology allows diagnosis of MCT in >90% of cases. MC are round cells with a round to oval nucleus and abundant cytoplasm containing cytoplasmic metachromatic granules in >90% of cases. Poorly differentiated MCT may lack granules and histology and potentially immunohistochemistry may be required for diagnosis. Cytology can give a *vague* direction whether the tumour is well differentiated or poorly differentiated. However, the degree of tissue invasion, which is a key criterion for accurate MCT staging cannot be assessed cytologically. Therefore, histology is always recommended for assessment of tumour grade (and surgical margins)

Biological behavior

MCT can cause problems due to local extension, metastasis and paraneoplastic disease. Several tests on histological specimens exist to predict the biological behaviour of MCT including:

1. *Classical histological grading system* (Patnaik 1984): 3 grades (grade 1 being good)
2. *New histological grading system (Kiupel, 2011):* 2 grades (low-grade *vs* high-grade)
3. *Mitotic Index* (high mitotic index being associated with poor prognosis)
4. *Ki-67 expression* (high expression being associated with poor prognosis)
5. *Immunohistological intracellular c-kit expression:* 3 staining patterns (patterns 2 or 3 are associated with a poor prognosis).

Tests 2-5 are especially useful for grade 2 tumours according to Patnaik.

Staging

All cutaneous MCT are potentially malignant, however the likelihood of metastasis is grade dependant. MCT can metastasize lymphogenously (i.e. to the regional lymph nodes) and/or haematologenously (predominantly to the liver, spleen and bone marrow). Metastasis to the lungs is very uncommon. Useful staging tests include:

1. palpation and aspiration cytology of regional lymph nodes *(see handout on lymph node cytology)*
2. ultrasound of the abdomen with a focus on liver, spleen and lymph nodes
3. potentially aspiration cytology of liver and spleen: matter of debate
4. chest radiographs (mainly for MCT in the cranial half of the body, to assess intrathoracic lymph node enlargement)

Not every patient requires full staging, many oncologists only recommend full staging in cases of poorly differentiated MCT, multiple MCT or MCT with confirmed lymph node metastasis.

Treatment

Therapeutic options depend on the number of MCTs present, the tumour grade, stage and location**.** Because of their unpredictable biological behavior, in general, MCT should not be "monitored"

Surgery is the treatment of choice for localized, non-metastatic cutaneous MCT. *It is essential to know before surgery if a mast cell tumour is present or not.* MCT grow invasively into surrounding tissues and therefore wide resection is necessary. Marginal resection leaves tumour cells behind and tumour recurrence is likely. A repeat surgery at that stage is, if at all possible, generally more difficult and invasive. For the majority of grade I and II MCTs, a lateral margin of 2 cm and a deep margin of 1 fascial plane is sufficient to achieve complete excision. The deep margin should always include the panniculus muscle (if present), the underlying fascia or, in its absence, the superficial layer of the underlying musculature. If complete excision is achieved, most grade 1 or grade 2 tumours can be cured. All MCT larger than 3 cm and all grade 3 MCT should be excised with a lateral margin of at least 3 cm plus the deep fascial plane.

Whenever surgical excision is incomplete, there are 2 options:

1. 2-3 cm re-resection around the scar including the deep fascial plane.
2. radiation therapy to "sterilize" the tumour bed

At some locations wide surgical resection may not be feasible: e.g. at the distal limbs. Here, marginal/debulking resections followed by adjuvant radiation therapy are recommended. In case of high-grade or metastatic mast cell tumours, adjuvant chemotherapy or tyrosine kinase inhibitors are recommended. Inoperable tumours can be treated with radiation therapy or chemotherapy. In some cases such treatment can shrink the mass and make these tumours resectable. For MCT grade 1 or grade 2 with lymph node metastasis marginal resection and adjuvant radiation therapy of primary tumour bed and lymph node bed can achieve median disease-free intervals or ~40 months.

In case of multiple, unrelated mast cell tumours (n<5-6) all mast cell tumours should be excised as if they were solitary MCT. If there are too many simultaneous MCT to resect, systemic therapy (chemo/TK-inhibitors) may be more appropriate.

Radiation therapy is recommended in cases of:

a. incompletely resected, microscopic MCTà radiation therapy of grade 1 or grade 2 tumours is most often curative. Local control can often be achieved in grade 3 MCT, but most succumb to metastatic disease
b. primary irradiation of macroscopic tumoursà in most cases only palliative (partial response, stabilization).

MCT are generally moderately radiosensitive. Generally, fractionated ("curative") radiation protocols are superior to hypofractionated ("palliative") protocols.

Systemic chemotherapy is recommended as primary or adjuvant treatment for metastasized and/or poorly differentiated MCT (i.e. grade 3 MCT or grade 2 MCT with negative prognostic factors). The goal is tumour control or delay/prevention of metastasis. Adjuvant chemotherapy may also be useful in incompletely resected MCT if another resection or radiation therapy are not possible.

Corticosteroids are often used as part of the chemotherapy regime because of their direct inhibitory effect on MC. However, response rates of prednisolone used as a signle agent are generally modest (20-40% remission) and short lived. A variety of other chemotherapeutic drugs are used including vinblastine, cyclophosphamide, lomustine, or hydroxyurea. Vinblastine and/or lomustine (CeCeNu) combined with prednisolone are the most potent agents. Response rates vary between 20-60%. Even with chemotherapy patients with grade 3 MCT generally only achieve median survival times of 7-12 months. An alternative to conventional chemotherapy are tyrosine kinase inhibitors (TKI).

Tyrosine Kinase Inhibitors inhibit cellular signaling and lead to growth arrest and apoptosis. In veterinary medicine 2 tyrosine kinase inhibitors (TKI) *masitinib and toceranib* have been licensed. They act as intracellular competitive inhibitors at the ATP binding site of the tyrosine kinase resulting in inhibition of tumour cell proliferation or angiogenesis and eventually apoptosis (programmed cell death). Masitinib (Masivet®, AB Science) is the TKI most commonly used nowadays. It is a TKI licensed by the European Medicines Agency (EMEA) to treat non-resectable canine mast cell tumours (grade 2 or 3) *with confirmed mutations* of c-Kit. In a clinical trial dogs with mast cell tumours (grade 2 or 3) treated with Masivet® did not achieve a higher rate of clinical remission than placebo-treated animals. Also, the survival rate of dogs with inoperable mast cell tumours was significantly higher in Masivet-treated dogs than in placebo-treated dogs at 12 months (62.1% vs 36.0%) and at 24 months (39.8% vs 15.0%). Although it is not licensed in dogs without c-kit mutations, clinical success has been documented in some dogs without confirmed mutations suggesting additional mechanisms of actions may be involved.

Prognosis

Clinical aspects with prognostic relevance are mainly tumour grade and stage. Dogs with grade 1 or grade 2 MCT that were completely excised or irradiated after incomplete surgical resection achieve local control in >90 % of cases: they are essentially "cured". Median survival of dogs with grade 3 tumours treated with multimodality therapy is ~7-12 months. Treatment with TK-inhibitors may be useful and achieve long-term survival in a subset of those patients.

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Canine urinary tract tumours

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Renal tumours:

Metastatic tumours to the kidney are common in end-stage patients. Primary renal tumors are rare (less than 2% of all tumors in dogs). Bilateral tumours are reported in a minority of cases. Etiology is unknown. Malignant epithelial tumours predominate (>75% of canine renal tumors) esp. renal carcinoma, less commonly other carcinomas. Mesenchymal tumors (esp. haemangiosarcoma) are rare (10%). Nephroblastoma is a congenital tumor with both multiple components. Lymphoma is rare in dogs, but common in cats. In the German Shepherd breed (rarely others) a familial cancer syndrome has been described with bilateral renal, cystadenocarcinomas (age 5-9), multifocal nodular dermatofibrosis (age 3-12) and uterine leiomyomas.

Diagnostic workup typically includes ultrasound, radiographs or CT and potentially cytology. It is imperative to rule out lymphoma, bilateral disease or metastatic disease prior to surgery. Lymphoma is potentially bilateral and should be treated with chemotherapy! Renal carcinomas should be treated with nephrectomy potentially followed by carboplatin chemotherapy. Median survival time is 12 months. For haemangiosarcomas without hemascos nephrectomy followed by doxorubicin chemotherapy is advisable. Ruptured haemangiosarcomas have a grave prognosis.

Bladder tumours:

Bladder tumours are uncommon and occur mostly in older pets (8-12 years). Transitional cell carcinoma is the most common type: 0.5-2% of all canine tumours, 70-90% of all canine bladder tumours, rare in cats.

Risk factors:

- Older types of flea control products; lawn chemicals; obesity; female sex; a very strong breed-associated risk (e.g. Scottish Terriers);
- A history of urinary tract infections is common in TCC cases in both species
- Vegetable consumption reduces risk of TCC in Scottish T.

Biologic behavior:

- typically high grade invasive cancer (in contrast to humans)
- Dogs: 90% at bladder neck/trigonum
- Cats: variable. Only ~15% at bladder neck
- Obstructing ureteral openings and urethra à hydronephrosis à renal failure
- Metastases (late): regional LNN, lungs, other (liver, vertebrae, etc)

History and clinical signs: dysuria, pollakisuria, stranguria, haematuria

Differentials

Differentials for non-specific urinary signs:

- Cystitis
- Urolithiasis
- Bladder tumour/urethral tumour
- Pyometra

Differentials for bladder mass(es):

- Transitional cell carcinoma (>95% in dogs)
- Other tumours (e.g. squamous cell carcinoma, adenocarcinoma, undifferentiated carcinoma, leiomyoma, leiomyosarcoma, and rhabdomyosarcoma, lymphoma…)
- Bladder Polyps
- Granulation tissue following trauma
- (Granulomatous urethritis)

Diagnostic options:

- *Ultrasound alone:* Not specific for TCC
- *VBTA-Test (Urine tumour marker:* Quick latex-agglutination test; Detects tumor-related glycoproteins in the urine; High sensitivity (<15% false negatives) but poor specificity (=cannot distinguish TCC from cystitis or other urinary tract disorders).Therefore only useful as a screening test in healthy individuals of high-risk breeds!
- *Cystoscopy:* diagnostic: 65% in males, 96% in females; Requires expensive equipment & expertise; only small biopsies can be taken; Potentially traumatic to the urethra

- *Cystotomy:* Invasive, higher risk of tumour seeding
- *Transcutaneous FNA-biopsy: c*onvenient, fast, often diagnostic, minimally invasive; but, potential risk of tumour seeding!
- *Cather-suction biopsy*: Useful, esp. if combined with ultrasound; often only small samples (cytology).

Staging:

- X-rays or CT (chest), ultrasound abdomen
- Cystoscopy

Systemic chemotherapy:

Systemic chemotherapy is the treatment of choice for non-resectable tumours. 75% respond favorably with a better quality of life and better life expectancy. First-line treatments are:

1. Cisplatin & NSAID
2. Carboplatin & NSAID
3. Mitoxantrone & NSAID

E.g. Mitoxantrone: 5-5.5mg/m^2 IV q 3 weeks; 4-6 x in total, slow infusion, diluted with 25-50ml saline and Meloxicam (daily treatment at standard dose). COX-inhibitors are useful as TCC are COX-2 positive. Median survival time: 10-13 months.

Rescue options:

1. Vinblastine 2.2-3.0 mg/m^2 IV every 2 weeks or
2. Chlorambucil (4 mg/m^2, PO, q 24 h)

Radiation therapy:

This is theoretically possible, but technically demanding. Possible collateral damage (intestine)! Unclear, if better than chemo alone.

Laser ablation:

Transurethral endoscopic near-infrared diode laser ablation can be used to bebulk obstructive TCC lesions. Well tolerated! Survival not better than with chemo alone, but there is better resolution of clinical signs! Median survival time 380 days.

Surgery:

Standard of care in humans with invasive tumours. In dogs reconstruction and urinary diversion can be problematic. Very useful if tumours are not at the trigone, but one should still give adjuvant chemo.

Prostate cancer:

The most common prostate tumours are carcinomas. Other tumours are rare. In a castrated male a large prostate is highly suggestive of cancer.

Biological behavior:

Locally invasive, obstruction of urination, regional metastases and distant metastases (incl. bone).

Clinical signs:

Lower urinary tract signs, tenesmus, pelvic limb weakness or lumbar pain or non-specific systemic signs

Treatment:

Surgery, chemo or radiation therapy are not effective! Potentially palliative urethral stenting! Sometimes there is palliative success with NSAIDs. Potentially Palladia (Toceranib) could be tried as it has shown some success with other carcinomas. Castration may resolve concurrent BPH and provide some palliation for a few months. Prognosis varies from weeks to up to year (median 3-6 months).

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Anal sac and perianal tumours

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Differential diagnoses for perianal masses

A wide variety of perianal tumours occur in dogs. Differentials include anal sacculitis (abscess), anal sac carcinoma, perineal (hepatoid) adenoma/carcinoma, mast cell tumour, soft-tissue sarcoma, lymphoma, squamous cell carcinoma, melanoma, transmissible venereal tumour, lipoma and other.

Adenocarcinoma of the anal sac

Introduction

Adenocarcinoma of the anal sac (ASAC) is the most common tumour type. Adenomas or squamous cell carcinomas are very rare. It is typically a disease of older dogs (median age: 10 years). According to recent studies, there is no sex predilection. Etiology is uncertain. A history of anal sacculitis is reported in some dogs. Sex hormones are not involved in the pathogenesis. The biological behavior of ASAC is characterised by locally invasive growth and early (pelvic, sublumbar) lymph node metastases (40-90% at initial presentation). Distant metastases (lungs, spleen, vertebrae, etc) occur typically only late in the course of disease. 25-80% have paraneoplastic hypercalcaemia due to production of a parathyroid substance (PTHrp). In 20-40% the tumour is an incidental finding on rectal examination. Bilateral tumours occur occasionally.

Therefore, the clinical signs vary from subclinical to tenesmus, licking the anus and signs of hypercalcaemia (polyuria/polydipsia, inappetence, lethargy...).

Diagnosis and staging

Diagnosis is made following clinical suspicion after rectal exam by cytology or histology. Finding a mass in the anal sac in a hypercalcaemic patient is highly suggestive. On cytology monomorphic cells with few criteria of malignancy and indistinct cell borders can be found. "Naked nuclei" are common, distinct epithelial clusters are less common. In contrast to hepatoid adenomas/carcinomas (see below) there is only one cell population.

Staging should include rectal palpation of pelvic or sublumbar lymph nodes, ultrasound or CT of the abdomen and chest radiographs or CT. Also, do a complete blood count and biochemistry including total or ionised calcium.

Clinical signs of hypercalcaemia

Generally hypercalcaemia "paralyses" cell function. Organs commonly affected are CNS, GI tract, heart, muscles and kidneys manifesting as lethargy, weakness, stupor, anorexia, vomiting, constipation, arrhythmia, polyuria/polydipsia and many other symptoms.

The kidneys are particularly vulnerable to the effects of hypercalcaemia:

a. Polyuria/polydipsia is the result of a lowered responsiveness of the cells in the distal tubules to antidiuretic hormone.
b. Reduced renal (blood) perfusion
c. Soft-tissue mineralisations
→ risk of renal insufficiency

Clinical signs are more severe if hypercalcaemia develops rapidly. Also, clinical signs are worse if acidosis is present than in alkalosis, as acidosis increases ionised calcium levels. Phosphate levels also determine the risk of soft-tissue mineralization: if the product of calcium (in mg/dl) and phosphate (in mg/dl) is higher than 60-80, the risk of soft-tissue mineralization increases (incl. heart and kidneys) with potential loss of function.

Therapy

If there is no evidence of distant metastases and the mass is smaller than 50% of the circumference of the anal sphincter, treatment of choice is resection of the anal mass and any enlarged regional lymph node. As only marginal resection is possible in most cases, adjuvant radiation therapy and/or chemotherapy is indicated (e.g. carboplatin 220-300mg/m^2 IV every 3 weeks for a total of 4-6 treatments; alternatives are: toceranib (Palladia, Zoeits) or metronomic chemotherapy). Median survival times with such a multimodal treatment is 15-18 months. If the mass is larger than 50% of the circumference of the anal sphincter, the risk of post-operative faecal incontinence increases. Therefore, primary radiation or chemotherapy (see above) can be suggested to reduce the mass to a resectable size or as palliation. The median survival

time is considerably less (3-9 months), but some cases live way more than a year.

Treatment of hypercalcaemia

Hypercalcaemia should normalize after resection of all macroscopic tumour. Recurrence of hypercalcaemia can be an early sign of tumour recurrence. If tumour resection is not possible, hypercalcaemia can be managed symptomatically:

Prednisolone and other glucocorticoids increase calcium excretion by the kidneys and are typically very effective to limit hypercalcaemia. However, you must not use these drugs prior to finishing your diagnostics as diagnosis (esp. of lymphoma) may not be possible afterwards. For mild to moderate hypercalaemia rehydration (IV 0.9% NaCl infusion) is typically enough. Only after rehydration you may potentially add frusemide (1-4 mg/kg, SID-TID PO or OV).

The following procedures may alleviate signs:

1. Prednisolone 0,5-1mg/kg/day PO
2. Pamidronate (1 mg/kg IV as slow infusion over 1-2 hours dilut. In 0.9% NaCl q 3-4 weeks)
3. Calcitonin/Salcatonin (4-10 units/kg, SC, SID-BID)
4. Mithramycin (25µg/kg IV over 2-4 hours q 7-14 days)

Recurrence of hypercalcaemia can be an early sign of tumour recurrence.

Perineal (hepatoid) adenoma and carcinoma

Perineal adenoma

50-96% of perineal tumours are perineal (hepatoid) adenomas. They are benign and occur as solitary or multiple lesions. They are typically slowly growing (months to years) and often asymptomatic. Tumour growth is sex hormone responsive: testosterone is growth stimulating, while estrogen is growth suppressing. Therefore, typically older, intact male dogs are affected by hepatoid adenoma. Concurrent testicular tumours are common (esp. (testosterone-producing?) interstitial cell tumours) auf. In female dogs this tumours almost exclusively occurs in spayed dogs. There are anecdotal reports of testosterone-producing adrenal tumours though. Cocker spaniels, beagle, bulldogs, Samoyed dogs appear to be predisposed. Diagnosis is made by signalement, appearance and cytology (or histology). On cytology epithelial cell clusters with 2 epitheliale populations can be found: large, polygonal hepatoid cells and small basal cells.

Treatment of choice is a marginal resection and castration. In some dogs castration alone achieves a partial or even complete remission. Alternatives are cryotherapy, electrochemotherapy and laser therapy. Estrogen treatment is not advisable due to its potential for (fatal) myelotoxicity. Prognosis is excellent. Recurrence are common if the clients opt for resection without castration.

Perineal carcinoma

Perineal carcinoma constitute 3-21% of all perineal tumours. They are malignant, locally invasive and metastatic (sublumbar and pelvic lymph nodes, lungs, etc). They are often fixed and ulcerated. They grow independent of sex hormones and thus occur in any sex and neuter status. Castrated male dogs or intact female dogs are more likely to have carcinoma than adenoma. Metastasis at initial presentation is rare (20%). Diagnosis is commonly made by histology (cytologically it is most often impossible to distinguish between adenoma and carcinoma).

Therapy is as for anal sac carcinomas: wide resection, followed by radiation and/or chemotherapy (same drugs as for ASAC). The tumours are not castration-responsive. Prognosis depends on the clinical stage: good (>70% live >2 years) for dogs with non-metastatic, low-grade carcinomas less than 5cm diameter. Higher tumour stages have a worse prognosis.

Recommended reading

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Ophthalmology

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Is this case an ophthalmological emergency? – part I-II

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In eye emergencies it is always an after and the owner will tell you the same story: my dog was hit by a car and after eye "popped", my dogs were playing very nice and after one of them closed the eye, we went in the park and after my dog closed the eye, my cats had a fight and after one of them closed the eye, we went to grooming center and after my dog closed the eye. Eye emergency means pain and the ocular examination revealed: closed eye, blepharospasm and miosis.

To make a proper diagnosis in eye emergencies you need: proper light, local anaesthetic, mydriatic, fluorescein test, loupe, ophthalmoscope and tonometer.

We can write a huge list with ocular emergency: ocular proptosis (figure 1 and 2), palpebral wounds (figure 5), third eyelid wounds (figure 6), ocular foreign bodies (figure 7 and 8), chemical corneal burns, melting corneal ulcer (figure 11 and 12), penetrating corneal wounds (figure 9), descemetocel (figure 10), acute vision loss (glaucoma – figure 13 and retinal detachment – figure 14, 15 and 16).

A proper diagnosis and a quick treatment can save the eye.

The most important challenges in ocular proptosis are complications: vision loss, strabismus and exposure of the cornea (figure 3), "dry eye" due to trauma of the lacrimal gland and phthisis of the globe (figure 4).

Figure 1 – Ocular proptosis in dog

Figure 2 – Ocular proptosis in cat

Figure 3 – Strabismus and exposure of the cornea secondary to ocular proptosis

Figure 4 – Phthisis of the globe secondary to ocular proptosis

In palpebral wounds and third eyelid wounds bleeding can be important. In palpebral wounds a proper suture using 4/0, non-absorbable will make a perfect edge of the eyelid and can avoid coloboma, ectropion or entropion.

Figure 5 – Palpebral wound in dog

Figure 6 – Third eyelid wound in cat

Figure 7 – Conjunctival foreign body (grass awn)

Figure 8 – Orbital foreign body (piece of wood)

In penetrating corneal wound and descemetocel surgical approach is the option. Corneal suture, coneo-conjunctival flap or using a Vetshield® collagen lens and third eyelid flap are used depend on the case and surgeon's experience.

In melting corneal ulcer, systemic (using Doxycycline®) and local treatment (using acetylcysteine, EDTA, antibiotic, hyaluronic acid or serum) can be an option in moderate cases. In severe cases, systemic treatment (using Doxycycline®) and surgery (simple tarsorrhaphy or VetShield and tarsorrhaphy) can save the eye. The sutures are removed after 2-3 or 4 weeks.

Figure 9 – Penetrating corneal wound in cat

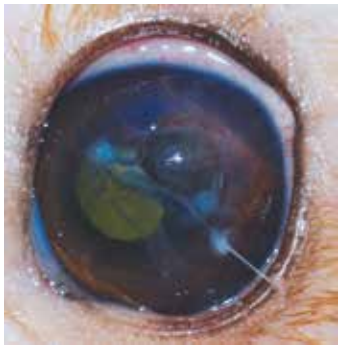

Figure 10 – Descemetocel in dog

Figure 11 – Melting corneal ulcer

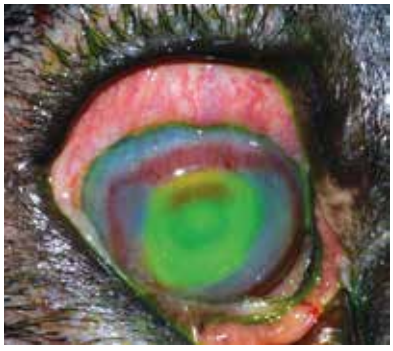

Figure 12 – Melting corneal ulcer

Acute vision loss secondary to glaucoma is common in dog and cat in lens luxation. The retinal detachment's etiology is diverse, that is why further investigations are necessary: measuring of the arterial blood pressure (arterial hypertension >180 mmHg can cause retinal detachment), biochemistry (urea, creatinine, T4, glucose), infectious diseases (Snap 4Dx, FIP, Distemper), hematology (thrombocytopenia, severe anemia), neoplasia (cilliary body tumor, lymphoma, fibrosarcoma). Even the dog or cat remain blind the cause must be treated.

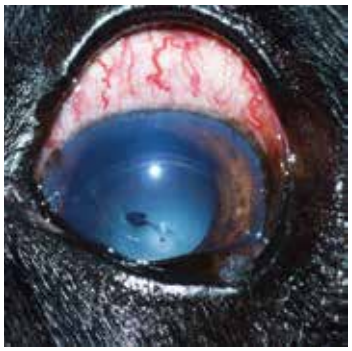

Figure 13 – Glaucoma in dog

Figure 14 – Retinal detachment in cat

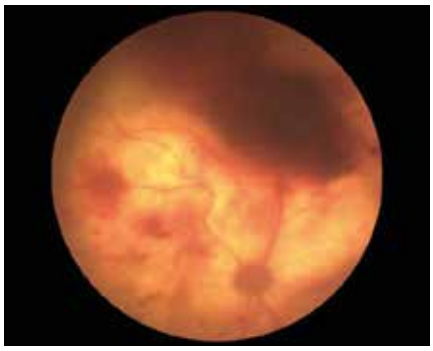

Figure 15 – Ocular fundus aspect in retinal detachment in cat

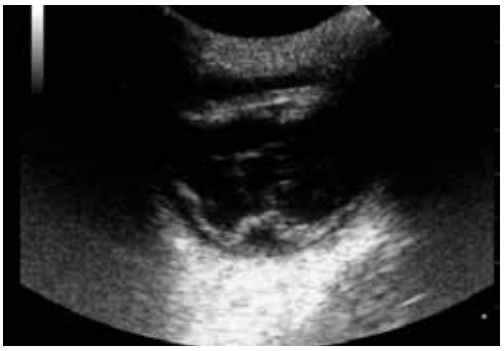

Figure 16 – Ultrasound aspect in retinal detachment

1. David J Maggs, Paul E. Miller, Ron Ofri – Slatter's Fundamentals of Veterinary Ophthalmology – fifth edition – Elsevier Saunders – 2013
2. Iuliana Ionascu Atlas de oftalmologie veterinara – Editura Curtea Veche – 2013
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"Dry eye" – always a challange!

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Keratoconjuctivitis *sicca* is common disease in dogs and cats and it has a large variety of clinical signs. It affects both females and males of any breed and any age.

There are genetic predisposition for keratoconjunctivitis *sicca* in some breeds (English Bulldog, Pug, Beagle, Samoyed, Shih Tzu, Cocker, West Highland White Terrier, Yorkshire Terrier). Can be senile, symptom in infectious diseases (Distemper) or in endocrine diseases (diabetes, Cushing), secondary to NSAID's therapy, secondary to periorbital trauma and frequent, secondary to third eyelid gland ablation.

It is often misdiagnosed as allergic blepharo-conjunctivitis, bacterial or viral conjunctivitis, anterior keratitis, vascularized corneal ulcer or pigmentary keratitis.

The Schirmer Tear Test (STT) is mandatory for all patients. The correlation between the value of the STT and the corneal aspect and neovascularization is very important. Cornea can be clear (figure 1), with mucous secretion (figure 3), secretions can stains fluoresceine (figure 4), neovascularization can cover the cornea (figure 5, 6) or pigmentation (figure 7,8).

Depending on the stage of the disease, the corneal neovascularization has different aspects: unique blood vessels, branched towards the center of the cornea; numerous blood vessels starting from the limbus on whole corneal circumference; blood vessels with limitrophe pigmentation, or covered by it. In any of this situations, the STT can have values between 0 and 10 mm/min (figure 2).

The most important part in managing the disease is the owner. Pay special attention how you instruct the owner to perform the cleaning of the eye before every treatment. In patients with dehydrated lacrimal secretion and adherent to the conjunctival fornix and corneal surface, the treatment's efficiency depends on the owner. He needs to be instructed that before administering any treatment, all secretions must be removed by sprinkling saline in a stream, using a 10 ml syringe put in the superior conjunctival fornix.

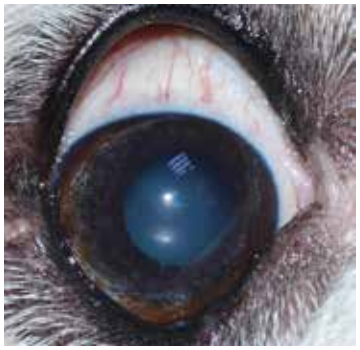

Figure 1 – The cornea is transparent

Figure 2 – STT is 5 mm/min

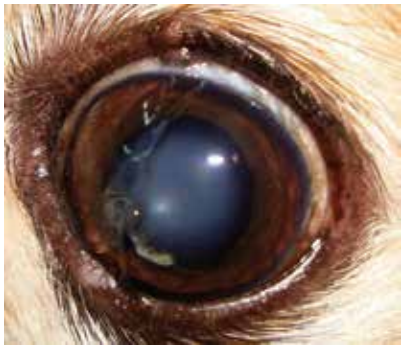

Figure 3 – Mucous lacrimal secretion

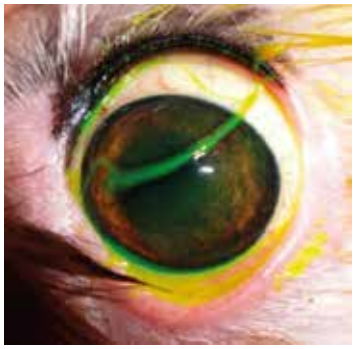

Figure 4 – The lacrimal secretion stains fluorescein

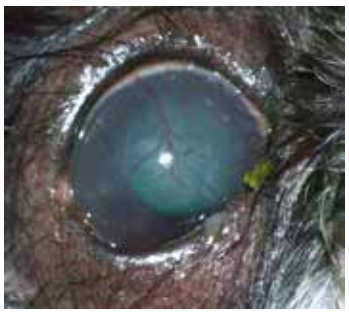

Figure 5 – OD Opaque cornea with branched blood vessels

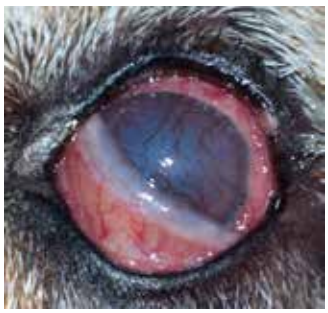

Figure 6 – Branched blood vessels on the entire corneal surface, "rain-like aspect"

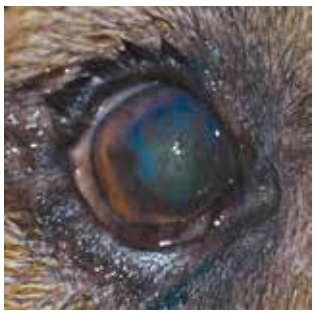

Figure 7 – OD when Tacrolimus therapy was started

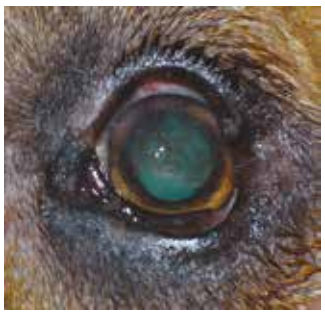

Figure 8 – OS when Tacrolimus therapy was started

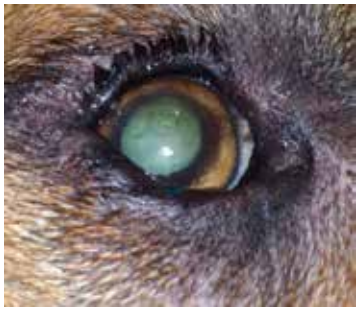

Figure 9 – OD after 6 months of Tacrolimus treatment

Figure 10 – OS after 6 months of Tacrolimus treatment. Transparent, shiny cornea

Don't forget important rules when you are dealing with "dry eye":

- instruct the owner to remove the dehydrated lacrimal discharge from the conjunctival fornix and cornea because all remaining discharge will absorb the medication like a sponge, leading to treatment failure.
- perform the STT for the correct evaluation of the prescribed treatment
- the owner must be informed that the disease will not be cured, it can be kept under control with proper medication, the entire life of the animal
- when the cornea is clear but STT under 10, prescribe artifial tears gel or drops
- to removal the pigmentation use Ciclosporine 2% or Tacrolimus (figure 9, 10)
- take pictures of the lesions in order to evaluate the treatment's efficiency
- if the local treatment is not efficient, investigate the patient, think that the "dry eye" can be a symptom of a systemic disorder
- if you carefully look at an eye and you discover on the surface of the cornea images that can be compared with the roots of a tree or with the branches of a tree without leaves, think first of keratoconjunctivitis sicca

1. David J Maggs, Paul E. Miller, Ron Ofri – Slatter's Fundamentals of Veterinary Ophthalmology – fifth edition – Elsevier Saunders – 2013
2. Iuliana Ionascu–Ghid Terapeutic Oftalmologic Veterinar – Editura Curtea Veche – 2015
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How can we save this cornea?

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Cornea is the structure of the eye constantly exposed to injuries. Histological, cornea has five layers: anterior epithelium, basal membrane, stroma, Descemet's membrane and posterior epithelium. Depending on the structure affected, corneal wounds can be: superficial (anterior epithelium), deep (anterior epithelium, stroma and basal membrane) and penetrating (the whole structure of the cornea, with or without loss of aqueous humor).

The superficial corneal wounds can be secondary to trauma, to eyelid abnormalities (entropion, ectopic cilia, distichiasis, trichiazis, chalazion, papillomas) or foreign bodies (awn grass – figure 1). The patient has closed eye, edema and erosions on the eyelids, blepharospasm, epiphora and miosis. Fluorescein test is positive (figure 2). Removing the foreign body under local anesthesia) or treating the underlying cause (surgery of the eyelids). Local treatment, 3-4 times a day, until fluorescein test is negative with: antibiotic, acetylcysteine 1% and hyaluronic acid.

Figure 1 – OS Superficial corneal wound secondary to awn grass

Figure 2 – Positive fluorescein test secondary to foreign body

In deep corneal wounds corneal epithelium and stroma are affected, until the Descemet membrane. The causes can be exogenous (trauma, foreign bodies) or endogenous (eyelids abnormalities or tear film deficiencies). The cornea can be transparent, with perilesional edema (figure 3), or totally opacified for lesions older than 14 days. Clinical signs are epiphora, blepharospasm, photophobia, miosis. The fluorescein test is positive and the lesion looks like a *"crater"* (figure 4).

Figure 3 – OD Deep corneal wound

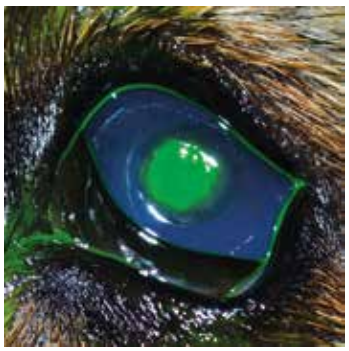

Figure 4 – OS Deep corneal wound

Local treatment, 3-4 times a day, until fluorescein test is negative with: antibiotic, acetylcysteine 1% and hyaluronic acid. In some cases general antibiotic (doxycycline) is indicated.Penetrating corneal wounds, secondary to cat claws, are quite often seen and have a poor prognosis, most of the time leading to loss of the eye. The chance to save the eye is always early diagnosis and aggressive treatment.

Sudden onset with deep ocular pain (closed eye, blepharospasm and aqueous humor loss which can be mistaken for an excessive lacrimation). In early cases miosis is the unique clinical sign (figure 5) and the fluoresceine test is negative. Untreated, after 2-3 days, the penetrated corneal wound is complicated with uveitis (the cornea is opaque and perilimbal blood vessels are *"short, straight, brush-like aspect"*, present on the entire corneal circumference – figure 6)

The penetrating corneal wound has different localization (central–figure 7 or perilimbal–figure 8) and shapes (round–figure 9 or linear–figure 10).

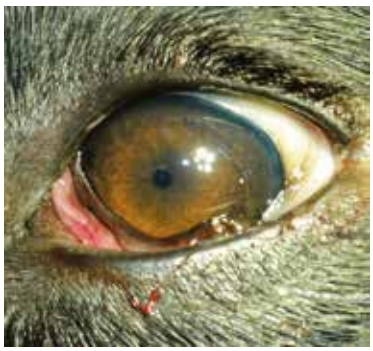

Figure 5 – OS Miosis

Figure 6 – OS Uveitis

Figure 7 – OD Central penetrated corneal wound

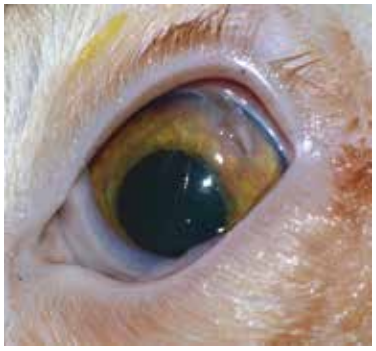

Figure 8 – OS Perilimbal penetrated corneal wound

Figure 9 – OD Round penetrated corneal wound

Figure 10 – OS Linear penetrated corneal wound

Surgical treatment is always mandatory: keratoraphy and tarsorrhaphy. Corneal wound suture using simple interrupted non-absorbable 7/0 (figure 11). Tarrsorrhaphy with 3 weeks maintenance of the suture (figure 12). Systemic antibiotic (amoxicilline or doxycycline) for 14 days, eye rinse with saline and kanamycin ointment application, 2-3 times a day, until suture removal. Elizabethan collar is mandatory for 21 days.

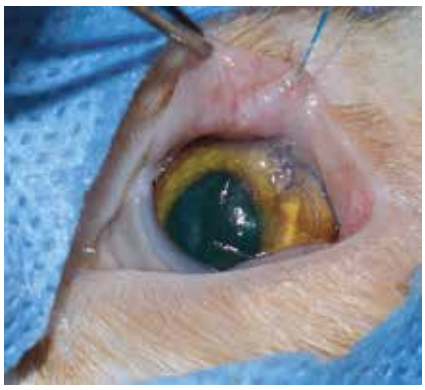

Figure 11 – OS Suture of the corneal penetrated wound

Figure 12 – Tarsorrhaphy

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Orthopaedics

Alessandro Piras (IT)

DVM, MRCVS, Specialist in Veterinary Surgery

Alessandro graduated from the Turin University College of Veterinary Medicine in 1989. He completed his surgical training in the College of Veterinary Medicine of the University of Parma in 1993 gaining the diploma of Specialist in Veterinary Surgery. In 1994 he spent an externship at the Canine Sport Medicine Center of the University of Florida and at the Hollywood Animal Hospital in Hollywood, Miami. Head Surgeon of Surgical Referral and Canine Sport Medicine Center of San Benedetto Veterinary Hospital, Forli, Italy. Mostly dedicated to Sport Medicine, traumatology, orthopedics and spinal. Alessandro is member of several scientific organizations, being involved in the boards and in the their didactic activities. Among these organizations, Alessandro is particularly active with AOVET. Member of the AO Veterinary Expert Group from 2004 to 2010, International Chair of the Community Development Commission from 2009 to 2015. Alessandro has been elected as Trustee of the AO Foundation in 2011. Presently Alessandro is International Chair of the AOVET education Commission. Alessandro is very active as AO Faculty member and International Speaker in several courses and conferences in Europe, Asia, Australia, USA and Latin and Central America.initiator and teacher of the Canine Sport Medicine Post Graduate Certificate at the University College of Dublin (UCD). Scientific Director and Co Founder of TRAKR, a continuing education provider in Canine Sport Medicine. Author of several chapter in 7 orthopedic books and Textbooks, Co Author in several scientific papers. Main areas of interest are: fractures treatment (particularly of distal extremities), musculo tendinous injuries and conditions, limb deformities correction, cruciate disease, circular and Hybrid External Skeletal Fixation, feline and toy breeds orthopedics. An important area of particular interest is canine sport medicine and surgery. A relevant part of Alessandro's clinical activity is dedicated to the treatment of injuries in sporting and high performance dogs.

Shoulder problems in the growing dog

Alessandro Piras – DVM – Specialist in Veterinary Surgery

Learning objectives

- Discuss the anatomical characteristic of the feline musculoskeletal system.
- List the most common fractures of the feline fore and hind limb.
- Discuss the optimal methods of fracture repair according to the different anatomical locations.
- Recognize the most common orthopedic pathologies affecting the feline patient.

Introduction

Among the developmental conditions of the canine and feline shoulder, joint dysplasia, congenital subluxation, growth deformities following trauma to the growth plates, ununited accessory caudal glenoid ossification centre and osteochondral lesions as a consequence to osteochondrosis are the most important. In the following only the last two conditions will be covered.

Accessory caudal glenoid ossification centre (ACGOC)

Small lesion like areas caudal to the caudal margin of the articular surface of the glena are most of the time incidental findings that are visible on lateral radiographs of the shoulder. This lesions, often bilateral, are only on rare occasions cause of pain and lameness and therefore all other causes of forelimb lameness must to be ruled out before attempting a diagnosis of a symptomatic accessory caudal glenoid ossification centre (ACGOC) (1).

During the orthopedic examination is possible to elicit a pain response by extending the affected shoulder joint. Flexion maneuvers are less sensitive and rarely painful. The confirmation of the diagnosis comes from arthroscopic visualization and palpation of the fragment. Only those fragments that are loose on palpation during arthroscopy are thought to cause pain via instability, inflammation of the subchondral bone and reactive synovitis (2). The latter indicates local inflammation and strengthens the diagnosis of a symptomatic ACGOC. Even though fragmentation of a formally normal caudal glenoid rim may occur in some very rare cases, destabilization of an ununited ACGOC is the most probable etiology in the majority of the cases. Ciliberto et al. (3) attempted to determine the frequency of ACGOC and the age of radiographic ossification of the ACGOC in the Rottweiler, one of the breeds most commonly affected by this condition. Overall, seven out of forty-five dogs (15.5%) had an ACGOC, and all were affected bilaterally. ACGOC was visible radiographically as early as 13 weeks of age and fused to the glenoid in six of seven dogs by 24 weeks of age. In case the ACGOC is the true cause of forelimb lameness, arthroscopic fragment removal is associated with good prognosis. (1,2). In most cases of radiographically diagnosed ACGOC a complete orthopedic work up including radiographs and CT of the elbows, ultrasonography of the biceps and supraspinatus tendons and measurement of abduction angles of the shoulder joint is necessary in order to identify other possible concomitant pathologies.

In case of simultaneous fragment coronoid process (PCP) and ACGOC and inconclusive clinical findings treatment of FCP alone is probably the best choice.

Osteochondrosis dissecans (OCD)

Osteochondral fragments of the humeral head almost exclusively develop in consequence to osteochondrosis (OC). Mechanical disruption of local blood vessels at the junction of subchondral bone plate and vascularized hyaline growth cartilage in immature dogs leads to focal disturbance of endochondral ossification and subsequently the development of osteochondrosis dissecans (OCD) (4). The most commonly cited etiologic factors are heredity, rapid growth, anatomic conformation, trauma, and dietary imbalances; however, only heredity and anatomic conformation are well supported by the scientific literature (4). Because the term OC has been used for a wide variety of conditions Ytrehus et al. (4) proposed to refine the terminology by introducing the terms “osteochondrosis latens” for lesions confined to the epiphyseal cartilage, “osteochondrosis manifesta” for lesions accompanied by delay in endochondral ossification, and “osteochondrosis dissecans” for cleft formation through the articular cartilage. The humeral head is the most common localization of OCD in dogs, being bilateral in about 25 to 65% (5, 6). Large and giant breed dogs are commonly affected and males are more often affected than females. Clinical signs often develop between 4 and 8 months of age; however, some dogs may not be presented for veterinary evaluation until they are mature. Affected animals are usually presented for examination because of unilateral forelimb lameness. Owners usually report a gradual onset of lameness that improves after rest and worsens after exercise.

Physical examination: The shoulder should be palpated and moved through a complete range of motion. Crepitation or palpable swelling of the joint is seldom evident, but affected animals usually exhibit pain when the shoulder is moved into hyper-extension or extreme flexion. Often the examiner can detect muscle atrophy of the forelimb by loss of muscle mass adjacent to the spine of the scapula.

Radiographs: Despite apparent lameness in only one limb, both shoulders should be radiographed because this condition is often bilateral. Sedation may be required for quality radiographs particularly in large hyperactive dogs. The earliest radiographic sign of OCD is flattening of the caudal humeral head. This is due to thickening of the articular cartilage and deviation of the subchondral bone line. As the disease progresses, a saucer shaped radiolucent area in the caudal humeral head may be visualized. Calcification of the flap may allow visualization of the flap either in situ or within the joint if it has detached from the underlying bone. In chronic cases, large calcified joint mice are often observed in the caudoventral joint pouch or cranially within the bicipital groove.

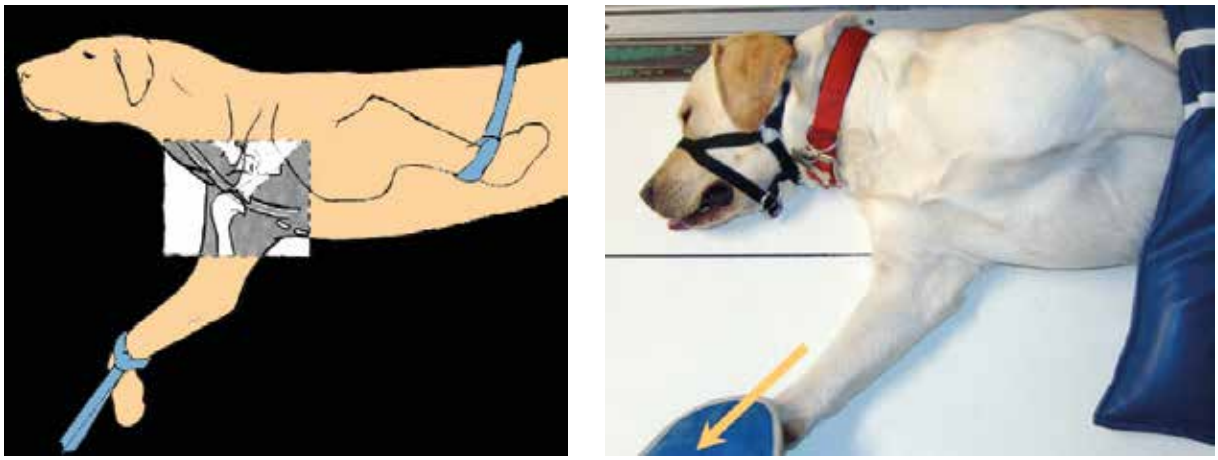

Figure 1 – Radiographic positioning: affected limb extended and contralateral limb flexed avoid superimposition of the two shoulder joints. (Courtesy of D. A. Vezzoni).

Figure 2 – Three radiographic appearances of OCD lesions.

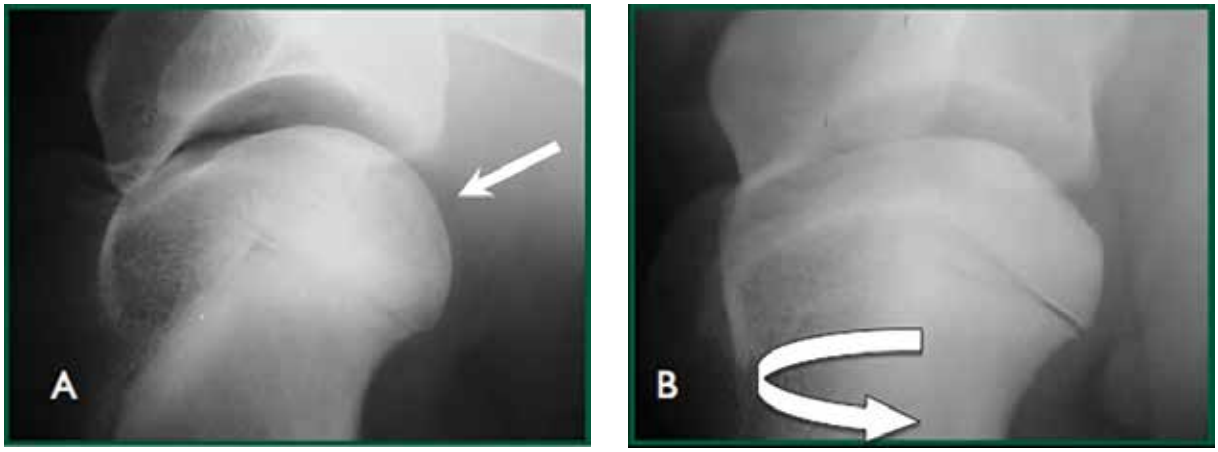

Figure 3 – External rotation of the limb helps in detecting lesions localized caudo medially. A: the lesion is not clearly visible in the standard ML view. B: external rotation evidence the OCD lesion.

Treatment: Even though conservative management, hoping for spontaneous debridement of the lesions and subsequent healing by formation of fibrocartilage may be an option, the spontaneous repair of osteochondral defects is of limited quality only (7). Therefore surgical debridement and removal of any loose cartilage and subsequently curettage of the lesion site until bleeding from the subchondral bone occurs is considered the treatment of choice in symptomatic OCD (8). In addition thorough examination of the entire joint for dislodged cartilage fragments is advised, as entrapped fragments may cause chronic synovitis and pain. Nevertheless bone marrow stimulating techniques can only promote fibrocartilage healing, which is of inferior mechanical quality than hyaline-like and for sure hyaline cartilage (9). Because OCD lesions mostly develop at sites exposed to high mechanical stress such as the caudal aspect of the humeral head (10, 11), the mechanical effect of the fibrocartilage scar is negligible (9). One might better think of fibrocartilage as a biologic wound dressing of an OCD lesion rather than a biomechanically functional repair, able to withstand the impacting forces. The only thing fibrocartilage is effective at is to reduce the coefficient of friction and to protect the nerve endings within the underlying, formerly eburnated bone from the inflamed synovial fluid and rubbing from the opposing joint surface. Fortunately, the prognosis following standard OCD treatment either by arthrotomy or even better by arthroscopy is considered good to excellent in up to 95% of the shoulders (12).

This leaves some shoulders with OCD with a guarded prognosis, which might be related to the exact localization of the osteochondral defect within the caudal region of the humeral head. Most of the lesions are located centro-medially making arthroscopic treatment challenging in some cases, but at the same time this localization is associated with a better prognosis (12), probably because the highest intra-articular forces are exerted at the caudo-central region of the humeral head (10, 11). Large lesions at this caudo-central region may fail to allow good postoperative function as the persistent loss of subchondral bony joint congruency and insufficient mechanical quality of the repair tissue cause persistent pain and promote accelerated degenerative joint disease. Osteochondral resurfacing either using autologous transplants or allografts has the potential to overcome these limitations, especially in (very) large caudo-centrally located lesions.

Now commercially available, synthetic plugs may be used successfully too, but information on long-term performance and the potential risk of aseptic loosening of such implants is lacking. Our limited experiences using auto- as well as fresh allografts, including prospective follow-up of

these cases using radiographs, CT and second-look arthroscopy are very encouraging. However, due to significant donor site morbidity at the stifle (patellar luxation) in those cases treated with autografts let us switch to fresh allografts. Another significant advantage of allografts is the bigger diameter of the grafts, and the orthotopic type of transplantation, which means that the damaged joint surface is reconstructed using osteochondral transplant matching cartilage and subchondral bone morphology and most importantly having the same surface curvature than the recipient site. Only matching for morphologic and topographic characteristics will allow for anatomical joint resurfacing, probably impacting on long-term performance of the reconstructed joint surface (13-15).

Surgical technique

Anatomic considerations

The shoulder joint is easily located by palpating the acromial process of the scapula and the greater tubercle of the humerus. The acromial head of the deltoideus is bordered cranially by the omobrachial vein and caudally by the axillobrachial vein. Muscular branches of the axillary nerve and caudal circumflex vessels are located deep in the caudal aspect of the acromial head of the deltoideus muscle, superficial to the triceps muscle.

Equipment

Standard surgical pack, two medium or large Gelpi retractors (depending on the size of the dog), blunt Hohmann retractor, bone curettes, pin chuck or high-speed wire driver, Kirschner wires or small Steinmann pin for forage

Preparation and positioning

Prepare the leg circumferentially from dorsal midline to the carpus. Use a hanging leg preparation with the dog in lateral recumbency to allow for maximal manipulation of the shoulder joint during surgery.

Procedure

Craniolateral Approach: Incise the skin and subcutaneous tissues in a curvilinear fashion from mid-scapula to midhumerus. Incise the deep fascia between the acromial and spinous portions of the deltoideus muscle (Fig 4 A). Further delineate this separation by blunt dissection with Mayo scissors, allowing for cranial retraction of the acromial head and caudal retraction of the spinous portion of the deltoideus muscle. The muscle branch of the axillary nerve is visualized at this point and preserved. Place the Gelpi retractors at 90 degrees to each other to facilitate visualization. Incise the joint capsule parallel to the rim of the glenoid cavity and replace the Gelpi retractors within the joint space to facilitate visualization. Internally rotate and adduct the humerus to maximize exposure to the caudal aspect of the humeral head. Place a blunt Hohmann retractor caudomedial to the humeral head to exteriorize the humeral head and further facilitate lesion visualization (Fig 4 C).

Caudal Approach: Incise the skin and subcutaneous tissues in a curvilinear fashion from mid-scapula to mid humerus.

Incise between the caudal border of the spinous head of the deltoideus and the long and lateral heads of the triceps muscle (Fig 4 B). Bluntly dissect under the deltoideus muscle to visualize the axillary nerve and caudal circumflex humeral artery and vein. Use Gelpi retractors to craniodorsally retract the teres minor muscle located deep to the spinous head of the deltoideus muscle. Elevate and gently retract the axillary nerve off of the joint capsule. Incise the joint capsule parallel to the rim of the glenoid cavity, and replace the Gelpi retractors within the joint space to facilitate visualization. Internally rotate and adduct the humerus to maximize exposure to the caudal aspect of the femoral head (Fig 4 D).

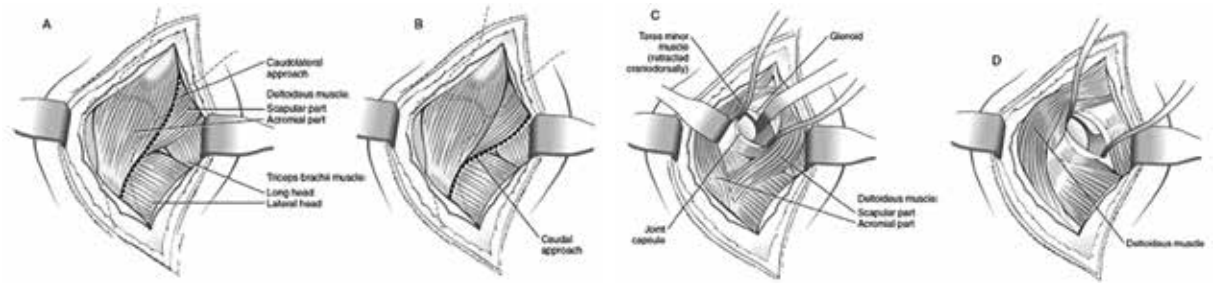

Figure 4 – A: Craniolateral approach to the shoulder joint. B: Caudolateral approach to the shoulder joint.

Curettage: Remove the cartilage flap with thumb or Halstead forceps. Probe the remaining cartilage surrounding the defect with a curette, and remove any abnormal cartilage not adherent to the subchondral bone (Fig 5 A).

Forage: Using a small Kirschner wire or small Steinmann pin, penetrate the sclerotic subchondral bone in multiple sites until it bleeds (Fig 5 B). Explore the caudal cul-de-sac of the joint for loose or free fragments of cartilage. Lavage the joint, and close the joint capsule and wound in a routine fashion.

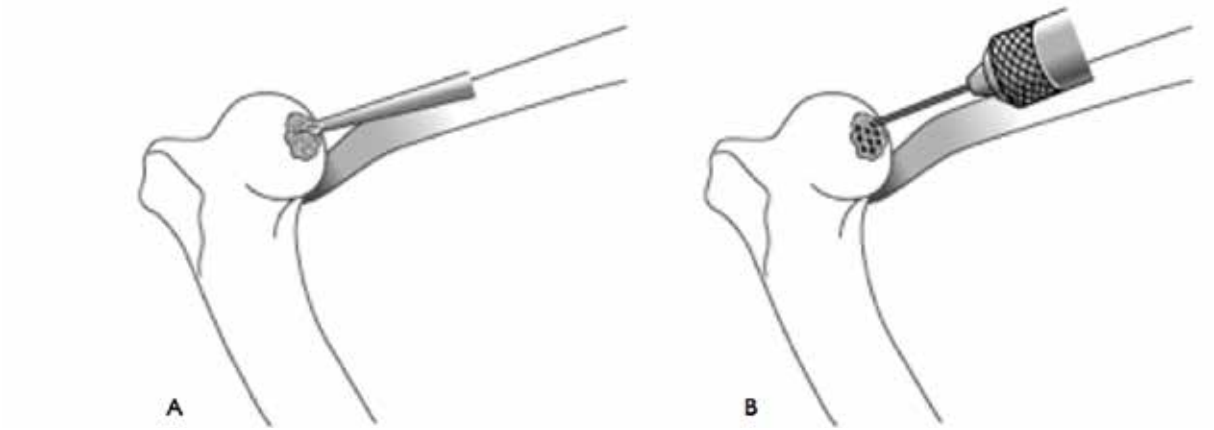

Figure 5 – A: curettage of the osteochondral defect. B: Forage of the lesion bed with a small diameter Kirshner wire.

Cautions

Osteochondrosis is often bilateral (42% to 65%)3; both shoulders should be evaluated, even if the animal exhibits a unilateral lameness.

Accurate hemostasis should be used when approaching the shoulder, as hemorrhage will greatly impede joint visualization.

Postoperative evaluation

No specific postoperative evaluation is required.

Postoperative care

Exercise should be restricted for 3 to 4 weeks to allow soft tissue healing and cartilage resurfacing, and then normal activity should be reintroduced slowly.

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How to make your life easier in treating successfully complex shaft fractures

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Comminuted fractures can be especially challenging due to the complexity of the fracture fragments and concomitant soft tissue injury. Careful consideration should be given to decision-making prior to onset of fracture repair. Factors that should be considered include mechanical, biological and postoperative compliance. Complex fractures that are treated with a mechanically sound repair often leave the surgeon pondering what could have possibly gone wrong when a "perfect" repair fails. Often times, the answer lies in the neglect of the biological or postoperative compliance factors. Neurologic function should always be assessed because complex fractures are often associated with high-energy trauma that also can injure the brachial plexus or peripheral nerves of the forelimb. This lecture will focus on presentation of clinical cases involving complex fractures of the forelimb and hindlimb, with an emphasis on the decision-making process. A variety of fracture repair techniques will be discussed including interlocking nails, plate-rod construct and linear external fixators.

Minimally-invasive surgical approaches reduce pain and minimize trauma to the soft tissues. Biological factors important for fracture healing are preserved, enhancing the body's ability for indirect bone healing. The technique can be used with all fracture types, but is particularly useful for stabilization of comminuted fractures. This type of bone healing is also referred to as secondary bone healing, spontaneous bone healing and callus healing. Stabilization of fractures using the principles of biologic fracture management is performed with the same type of implant systems used with traditional fracture repair, including externally and internally applied devices.

Fracture management

Comminuted fractures of the extremities can be challenging. It is always a race between a fracture healing and an implant failing. Steps can be taken to tip the scale in the direction of early fracture healing. These steps include:

1. Minimally invasive surgical approach
2. Preservation of soft tissue attachments to bone fragments
3. Use of cancellous bone grafts
4. Rigid method of fracture stabilization
5. Early return to function

It is always important to obtain an accurate history prior to stabilizing fractures. A complete physical exam and appropriate diagnostic tests should performed. Pathologic fractures are more likely to be seen in the geriatric dog and cat and should be identified preoperatively to ensure proper client education and communication

Indirect bone healing

Biological fracture management utilizes indirect fracture reduction to preserve the soft tissue envelope at the expense of anatomic reduction. Indirect bone healing occurs as a result. Indirect bone healing consists of three elements: first the formation of granulation tissue at the fracture site, second is fracture gap widening due to resorption of the bone ends and third is the new bone formation involving generation of a bone callus. Less disruption of the vascular supply to bone fragments is achieved through minimal handling of the fragments, promoting early callus formation.[2,3,6,7] Indirect bone healing is first associated with the formation of fibrous connective tissue and cartilage callus between the fragments.[4] Indirect bone healing occurs due to instability at the fracture site and is partially regulated by fragment gap strain.[4] Interfragmentary strain is a ratio of change in the gap width to the total width prior to physiological loading.[1,5] A study of the "interfragmentary strain hypothesis" using ovine osteotomy models demonstrated that the initial stages of indirect bone healing occur earlier and more extensively between gaps with lower shear strain.[1] Management of a non-reducible diaphyseal fracture with an implant system that does not utilize anatomical reconstruction and creation of subsequent small fracture gaps avoids high interfragmentary strain, favouring bone healing.

Implant systems

External and internal implant systems can be used to achieve bone healing using biological fracture management. Examples of external devices when used in an appropriate manner include casts, splints, linear external fixators and circular fixators. Internal devices commonly used for this application include the plate-rod system, interlocking nail and bone plates. Other implant systems can also be used for biologic fracture management as long as the soft tissue envelope is preserved at the fracture site. Whatever implant system is used, its application must be possible with minimal or no handling of the comminuted fracture fragments.

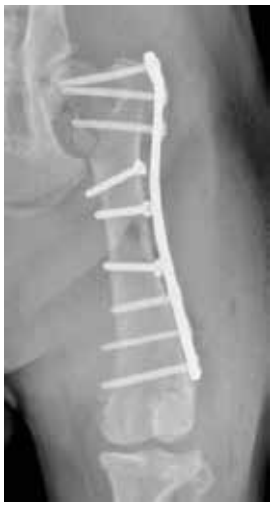
External fixator

External fixators provide rigid stabilization and can be used with minimally-invasive technique. Many fractures of the radius and tibia can be reduced closed and stabilized with an external fixator. The main disadvantage is the potential for complications with premature pin loosening and the added care needed in the postoperative period. The use of external fixators for fracture repair is not optimal if the patient or owner is likely to have poor compliance in the postoperative period. External fixators frames can be applied in one of 3 configurations-linear, circular or as a hybrid of linear and circular.

Plate-rod construct: The plate rod system has been found to be an ideal implant system for biological fracture management. Management of a non-reducible diaphyseal fracture with a combination of an IM Steinmann pin and bone plate can be applied without anatomical reconstruction and thus, avoids the development of small fracture gaps with high interfragmentary strain. The addition of the IM pin to the plate also significantly increases the construct stiffness and estimated number of cycles to fatigue failure when compared to a plate only construct. An IM pin serves to replace any trans-cortical defect in the bone column and acts in concert with the eccentrically positioned plate to resist bending.[2] Mathematical analysis of the plate-rod construct in the canine femur demonstrated that the pin and plate act most like a dual-beam structure, assuming slight motion of the pin in the canal.[2] Addition of an IM pin to a bone plate has been shown by Hulse et al. to decrease strain on the plate two-fold and subsequently increase the fatigue life of the plate-rod construct ten-fold compared to that of the plate alone.[1] In the canine femur, plate strain is reduced by approximately 19%, 44%, and 61% with the addition of an IM pin occupying 30%, 40% and 50% of the marrow cavity, respectively.3 Stiffness of plate-rod repairs may be as much as 40% and 78% greater when the pin occupies 40% and 50% of the marrow cavity, respectively.2Ideal diameter of the IM pin should be between 30 and 40% of the medullary canal diameter measured at the istmus. Increasing diameter up to 50 dramatically challenge the ability to insert screws trough the plate holes.

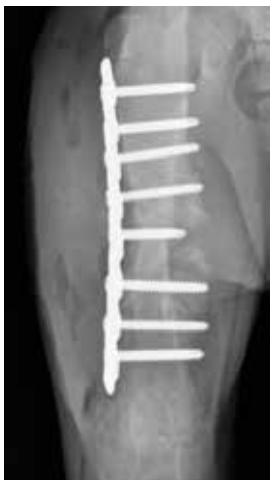
Locking plates

Locking plates have become very popular for minimally-invasive fracture repair. Many locking plate systems are available including the Synthes, FIXIN, SOP and ALPS. Locking plates have the ability to lock the screw into the hole of the plate. The mechanism for locking varies amongst manufactures. The Italian design FIXIN locking plate system has a conical locking mechanism while the Synthes system has a threaded locking mechanism. The FIXIN plate hole is tapered to match the conical nature of the head of the screw. This type of fitting is similar to the Morse taper of the head and neck fitting of the Total Hip Replacement implant. The stability of this design is extremely secure. The Synthes locking plate has threaded holes in the hole of the plate. Corresponding threads in the head of the screw engage the threads of the hole, locking the screw to the plate. The ability to lock the screw to the plate increases pull-out strength of the screw and construct stability. Traditional plates do not have threaded holes. Screws placed in ordinary plates apply pressure to the plate, pressing it onto the bone surface. The friction between the plate and the bone provides the stability to the bone-implant construct. In contrast, the locking plate achieves stability through the concept of a fixed-angle construct. The locking plate is not pressed firmly against the bone as the screws are tightened. The locking screws and plate function more like an external fixator. Locking plates are essential "internal fixators". The plate functions as a connecting bar and the screw functions as a threaded fixator pin. The tapered or threaded head of the locking screw engages the hole of the plate, similar to the clamp of an external fixator. The Synthes locking plate also has combi-holes which allow use of traditional or locking screws when desired. Traditional screws should be place prior to locking screw when using locking plates.



The FIXIN locking plate uses a conical head to lock into a matching conical hole in the plate creating fixed-angle stabilization.

Locking plates are ideal for minimally-invasive fracture repair for several reasons. Blood supply to the bone is preserved because the plate is not pressed tightly against the bone. The plate does not require perfect anatomic contouring because the displacement of the plate will not occur as the screw is tightened into the hole of the plate. Accurate contouring is difficult with a minimally-invasive approach due to the minimal exposure to the shaft of the bone. Lastly, locking screws give fixed angle support to the non-reduced fracture, increasing stability and less chance of collapse and instability at the fracture gap.



A Synthes locking plate and locking screws were used to revise the fracture. The fracture healed quickly without complication. Locking screws have increased pull-out strength compared to traditional screws.

Interlocking nail

The Deuland interlocking nail system presently available in the U.S. (Innovative Animal Products, Inc., Rochester, MN) is a modified Steinmann pin modified by drilling one or two holes proximally and distally in the pin, which allows the placement of transverse bolts or screws through the bone and nail. The nail, bolts and screws can be applied in closed or open fashion due to the incorporation of a specific guide system that attaches to the nail. The equipment needed to place the nail includes a hand chuck, extension device, aiming device, drill sleeve, drill guide, tap guide, drill bit, tap, depth gauge, and screwdriver. Cost of the system is reasonable and each nail is approximately half the cost of a comparative bone plate. The nails are available in diameters of 4.0, 4.7, 6, 8 and 10 mm and varying lengths and hole configurations. The 4.0 and 4.7 mm nails use 2.0 mm screws or bolts. The 6 mm nail is available in two models and will accommodate either 2.7 or 3.5 mm screws or bolts. The 8 mm nail is also available in two models and will accommodate either 3.5 or 4.5 mm screws or bolts. The 10 mm nail uses 4.5mm screws or bolts. The solid cross locking bolts have a larger diameter compared to a similar diameter screw, thus are less likely to break. Bolts also provide superior mechanical behavior compared to screws.

The interlocking nail is placed along the mechanical axis of the bone. The interlocking nail neutralizes bending, rotational and axial compressive forces due to incorporation of transfixation bolts or screws which pass through the pin and lock into the bone. This is in contrast to a single intramedullary Steinmann pin which is only effective in neutralization of bending forces. The interlocking nail has a similar bending strength compared to bone plates, but is slightly weaker in neutralization of torsional forces. The screws also prevent pin migration, a common complication seen with Steinmann pins.

When using an interlocking nail, the largest diameter nail should be selected that can be accommodated by the medullary cavity at the fracture site. In most large dogs, an 8 mm nail and either 3.5 or 4.5mm screws or bolts can be used in the femur and humerus. In medium-sized dogs, the 6 mm nail and either 2.7 or 3.5 mm screws or bolts are typically used. In small dogs and cats, the 4.7 mm nail and 2.0 mm screws are typically used. The tibia of medium and large–sized dogs will usually accommodate a 6 mm nail, but some large dogs will accept an 8 mm nail. Small dogs and some cats will accept a 4.0 mm nail for repair of tibial fractures. Dejardin et. al. have developed a novel interlocking nail that provides an angle stable locking mechanism. The advantage of angle stable locking is the elimination of torsional and bending slack, resulting in reduced interfragmentary motion. This interlocking nail system provided comparable mechanical performance to a plate system. Dejardin's nail is currently unavailable, but release of the nail is expected in the near future.

Interlocking nails provide axial, bending and rotational stability due to the ability of the screw to lock the IM pin to the bone.

Surgical approach

Closed reduction and stabilization is the optimal method of treatment when possible. Unfortunately, this method is rarely possible in the senior patient due to the severity of fractures seen, long time until bony union, and the tendency for patients to develop bandage sores. Open surgical approaches can be either traditional or minimally invasive. The minimally invasive approach has also been described as an "open but don't touch" approach. The acronym, OBDT, is used to describe this technique. The advantages to using an OBDT technique is preservation of vascular supply to the fracture site and thus quicker healing, shorter intraoperative time, less postoperative pain and early return to function. Methods of stabilization that work well with an OBDT approach include the interlocking nail, plate-rod hybrid and external fixation. The key feature of a minimally-invasive approach is the preservation of the soft tissue envelope at the fracture site. Small comminuted fragments will become quickly incorporated into the bony callus if left with a vascular pedicle. Anatomic reduction of small fragments is difficult if vascular supply to the fragment is to remain uncompromised.

Bone grafts

Numerous sites for harvest of cancellous bone graft have been described in the dog, but the most practical are the greater tubercle of the humerus, wing of the ilium and the medial, proximal tibia. The humerus provides the greatest amount of cancellous bone, but the ilium and tibia provide sufficient amounts for most applications. All of these sites are readily accessible, have easily recognizable landmarks, have little soft tissue covering, and provide relatively large amounts of cancellous bone. The greater trochanter can also be used if other sites are not available; however, the yield of cancellous bone is markedly less. Occasionally multiple sites are required to harvest sufficient quantities of bone to fill large bone defects or during arthrodesis.

Minimal instrumentation is required for harvest of cancellous bone graft. Basic surgical instruments are used to approach the site selected for harvest. A hole is drilled through the near cortex using either a drill bit, trephine or trocar-pointed pin. A curette is used to scoop the graft out of the metaphyseal cancellous bone. The cancellous bone should be scooped out in large clumps if possible. Use a curette that can be comfortably manipulated in the medullary cavity; I prefer to use a relatively large curette as this speeds harvest and reduces

trauma to the graft. Closure is performed routinely in 2-3 layers. Recently, a technique was described using an acetabular reamer to harvest large amounts of corticocancellous bone graft from the lateral surface of the wing of the ilium.

The graft collected should be handled gently. It is desirable to collect the graft immediately prior to usage. This increases the osteogenic properties of the graft. As graft is harvested, it should be placed on a blood-soaked gauze until transfer to the recipient site. Extreme care should be taken to store the graft properly; do not accidentally discard the graft due to misidentification of the gauze as being used. The graft should be

atraumatically packed into the recipient site. Lavage of the site should be avoided after the graft is placed.

Acknowledgements to Dr B. Beale

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Feline fractures and orthopedic conditions

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Learning objectives

- Discuss the anatomical characteristic of the feline musculoskeletal system.
- List the most common fractures of the feline fore and hind limb.
- Discuss the optimal methods of fracture repair according to the different anatomical locations.
- Recognize the most common orthopedic pathologies affecting the feline patient.

Introduction

Over the last twenty years, we assisted to a dramatic improvement in the care of companion animals and nowhere has this been as evident as in peoples' attitudes towards cats. Today cats are living longer, healthier lives. With this, owners have begun to demand more advanced care for their feline pet.

Although the broad principles of orthopedics are the same irrespective of species, cats are not small dogs, and important differences exist with respect to anatomical features and function as well as management of fractures and orthopedic conditions.

Sadly, the common misconception that repairs of feline long bones is simple to perform and not prone to the same range of complications seen in dogs, is still strongly radicated in many members of our profession. It has been facetiously remarked that two cat bone fragments will heal if placed together in the same room. Unfortunately, this casual attitude is not borne out by clinical experience and studies, which have shown the cat to be susceptible to the same range of complications as the dog. The notion that feline fracture repair is straightforward may have arisen because of the cat's ability to compensate for impaired function. This lecture provides an overview of the most common orthopedic conditions and methods of fracture repair in the cats with the emphasis on features that are pertinent to this species.

Diagnostics

As with canine orthopedics, a good history and orthopedic exam are the cornerstone to making the correct diagnosis. Unfortunately, due to the indoor/outdoor "style of life" of the majority of domestic cats, it is often impossible to obtain a reliable history from the owners.

Major differences include also basic feline anatomy and the cat's non cooperative nature. Most cats are reluctant participants when it comes to gait evaluation and it is very difficult to get a gait analysis in the exam room. They frequently are hesitant to walk and prefer to huddle in a corner or under a chair. Cats may walk if approached form behind or encouraged by their owner. It may be necessary to observe the cat though a window or from behind a door, or by having the owners bring a video taken in the cat's environment. For these reasons, historical information may provide valuable details about the affected limb. Additional dilemmas arise when trying to examine a cat that is painful or fractious.

Sedation may be required after a complete physical exam, especially in trauma patients, and an orthopedic exam is performed. If possible, always attempt to localize which region of a particular limb is affected prior to sedation, including neurologic status. A complete orthopedic exam consists of careful, systemic palpation of all four limbs, generally examining the affected limb last. Each joint should be checked through a full range of motion, paying attention for limited movement, effusion, pain response or crepitus. It may be necessary to repeat any palpation that elicits an abnormal reaction, as it can be difficult to differentiate uncooperativeness from true discomfort. The neurologic status of the patient should always be assessed.

Although two orthogonal views represent the core of the radiographic examination, occasionally special views will also be needed (oblique, skyline etc.). Computer Tomography (CT) and Magnetic Resonance Imaging (MRI) are valuable modalities for specific indications; however, they are relatively expensive and generally limited to large referral practices or veterinary teaching hospitals at this time. Ultrasonography has been occasionally used in cats to image tendons and soft tissue masses. Arthroscopy of the feline joint is also an excellent method of evaluating the feline shoulder, elbow and stifle.

Orthopedic implants systems

The successful management of a fracture depends on proper diagnosis and planning. Several variables must be considered prior to selecting a method of repair. Size and age of the cat, as well as fracture type and concomitant soft tissue damage play a large role in selection of repair. Other factors that should be weighed are disease status of the cat, owner's willingness to comply with postoperative management, and the cat's temperament and environment. Cats that are allowed to return outside shortly after fracture repair will require different fixation than strictly indoor cats. The surgeon must also consider equipment availability, cost, and his/

her skills level. Successful fracture repair is a race between healing and implant failure. Therefore, the selection of an appropriately placed implant can be the single most important factor in outcome. Several implants systems have been used successfully in feline orthopedics.

Intramedullary pinning (IM) has been successfully used for years to effectively stabilize fractures of long bones in cats. Pin type includes Steinmann pins, Kirschner wires (K-wires), and Rush pins. Steinmann pins are available in diameters from 1.5 mm to 6 mm, and may be threaded or smooth. Even large cats will rarely require a pin larger than 3.5/4.5 mm. K-wires are similar to Steinmann pins, with diameters of 08 mm with 2 mm increments till 2.8 mm. Rush pins are introduced at an angle such that they flex, and are seated with two to three points of contact within the medullary canal. Since cats have straight bones, relative to dogs, it is possible to fill the entire medullary canal, and still maintain anatomic reduction. However, this may lead to disruption of medullary blood supply, and increase risk of delayed union. It is generally best to use a pin that is 60 to 75 % of the medullary canal. IM IM pins must not be used as a single form of fixation as they do not provide sufficient stability. IM pins counteract only bending and shearing forces but are not effective to provide rotational and axial stability. Generally IM pinning is associated to application of cerclage wires where the bony column can be anatomically reconstructed or with external skeletal fixators (ESF) particularly in cases of unreconstructable fractures.

Recently, studies have shown good success in the repair of some fractures in cats with interlocking intramedullary nails.

The interlocking nail (IN) allows a more limited approach than bone plates, and provides adequate rotational and axial stability. This technique had been reserved for dogs, due to the diameter of available interlocking nails in veterinary medicine until the release of the Small Interlocking Nail System. This system contains nails of 4.0 and 4.7mm diameter, which are available in lengths of 68, 79, 91, 101, and 112mm. Each nail accepts 2.0mm screws in either three or four interlocking holes. Nails which contain only three holes will have only a single hole proximal or distal. This system also contains a jig and extension device to allow placement without fluoroscopy.

For years external skeletal fixation has been used to repair several types of fractures in cats. Many different types of external skeletal fixators (ESF) have been used, depending on the type and location of the fracture. Advantages of the external fixator include ease of application, minimal approach to the fracture, allowance for management of associated open wounds, compatibility with other fixation devices, it is well tolerated by cats, ease of removal, and lower cost to the surgeon and owner. Many miniaturized ESF system are currently available in the market proposing small clamps and connecting bars that are adequate to lodge small threaded pin sizes. Acrylic frames are an excellent alternative due to their great versatility and low cost.

Selection of pin size is important, as pins too large can weaken the cortex. A good rule of thumb is to select a pin 20-30 % the bone diameter. The most common type of fixator pin used in cats is a positive profile threaded pin, usually end threaded. A recent development in circular ring fixators may make placement of Ilizarov-type ring fixators, for congenital abnormalities, as well as fractures, in cats possible. The Miniature Circular External Skeletal Fixator provides 35mm rings with 0.8/1.0/1.2 mm K-wires.

Bone plates: When plate osteosynthesis is chosen as the method of internal fixation, implants size becomes the major dilemma. Short bones with a narrow diameter prove to be a challenge when selecting the appropriate sized bone plate. For many years, the most frequently used implant size was the 2.7 mm DCP for femoral, tibial and humeral fractures and the 2.0 mm DCP for radial fractures.

Unfortunately both implants have shown to be suboptimal and frequent complications related to implant size and shape where stress protection, screws loosening, bending and breakage.

Fortunately, in recent times many “cat friendly” implants have become commercially available Most of Mini Fragment Sets include 2.0/1.5 mm LC-DCP type (Low Contact Dynamic Compression Plate) that ranges from 4 holes (approx. 25mm length) to 14 holes (approx. 85mm length), and the 2.4/2.0 mm that ranges from 4 holes (approx.33 mm length) to 14 holes (approx. 113 mm length).

In particular, the 2.4 LC-DCP stainless steel plate, has proven very successful in treatment of the cat’s long bones fractures (K. Zahn, U. Matis-2002).

The limited bone-implant interface, the screws and plate size together with the interesting mechanical characteristics, make the 2.4mm a very adequate implant for the average cat size.

Another excellent implant is the veterinary Cuttable plate (VCP). The VCP it is cuttable to the desired length, its stiffness is adjustable, it has a large number of holes per unit of plate length, it can be used with different size screws which can be adapted to the different size bones, and is also economical to use and stock. Two sizes of VCP are currently available for use, the 1.5/2.0mm and the 2.0/2.4/2.7mm. These two plates differ in thickness and plate hole diameter but they have the same length (300 mm.), width (7 mm.) and distance between the plate holes centers. The Veterinary Cuttable Plate can be cut to the desired length with standard pin cutters (usually pin cutters for 2.5 diameter pins are sufficient).

One of the great advantages of the VCP is the possibility of increasing the degree of stiffness of the fixation by stacking (sandwiching) two plates of the same or different length and thickness. Stacking plates allows the surgeon to vary the plate rigidity according to the different fracture situation. Staking can be full length or partial with the top plate overlapping the bottom plate for three quarters or one half of its length. This change in the plate stiffness allows a more gradual stress distribution and decreases the stress risers at the end of the plate.

When stacking the veterinary cuttable plates, certain considerations should be kept in mind.

When using plates of different thickness, the screw diameter is determined by the largest screw that will fit the smaller plate and the thicker and longer plate is always placed adjacent to the bone.

The VCP are easy to contour making them adaptable to a wide variety of irregular shaped bones. If plates are to be stacked, they should be contoured simultaneously. Overlapping the two plates and inserting the largest size screw at each end of the plates minimizes slippage and eases the contouring procedure.

Among the advantages of the VCP is the large number of holes per unit of plate length which allows not only secure fixation of short bone segments but also permits the inclusion of small fracture fragments in the reconstruction that otherwise would be difficult to capture beneath the plate.

VCPs can be placed in buttress fashion in cases of non reconstructable comminuted fractures. It is recommended to stack the plates to increase plate stiffness in order to avoid implant failure. A combined rod and VCP plate construct offers another excellent option.

Last but not the list, in this overview of "cat friendly implants" are the Locking Plates.

Recent advances in the treatment of fracture repair in human and veterinary orthopedic surgery have led to the development of locking plates. Locking plates are fixed-screw angle implants where the screw locks into the plate either by a threaded or conical coupling mechanism and the standard cortical threads, which engage the bone. The locking plate systems can be likened in their function and behavior to an internally placed external skeletal fixator and offer certain advantages in fracture and osteotomy fixation over conventional plating methods. Locking plate systems increase the stability of the construct compared to traditional plates minimizing the amount of screws needed for adequate stabilization, and do not require anatomic contouring of the plate simplifying plate application.

There are currently several different types of locking plate mini systems available including the Kyon's Alps System, Synthes LCP, Fixin and Securos just to mention some, which may be an excellent method of fixation for the treatment of cat's fractures.

The LCP mini system (2.0/1.5, 2.4, 2.7) is available in a wide range of standard straight plates as well as a variety of shaped plates.

The most interesting and differentiating feature of this system is the screw hole design, which is a combined locking and compression hole or "Combi" hole. The "Combi" hole allows the plate to be applied with either fixed angle locking screws in the threaded part of the combined hole or standard cortical screws that are placed in the dynamic compression unit (DCU) of the same hole. Application of the LCP with entirely locking self-tapping screws results in fixed-angle construct for superior fragment stabilization. Using standard cortical screws, in case of a reducible fracture, is possible to apply dynamic compression across the fracture line. A specially designed drill guide is used for insertion of the locked screws to ensure appropriate screw placement of the bone to allow for the screws to adequately engage and lock into the threads of the plate. The standard AO drill guides are used for screws placed in dynamic compression fashion.

They "Combi" hole concept has been reproduced by several manufacturers and somehow even implemented with the possibility of compression in both directions together with the locking capability. As well some manufacturers improved the plates design by proposing specially shaped plates for specific anatomical locations.

Common fractures in cats

Antebrachial fractures: Feline antebrachial fractures were reviewed in a retrospective study of cases presented to two university teaching hospitals (Wallace AM et al, 2008). A high incidence of complications was noted, with 9/46 (19.6%) of cases requiring revision surgery. The most problematic fracture types were the combined diaphyseal and proximal ulnar fractures (Monteggia and olecranon fractures) with 23.1% and 30% of these cases requiring revision respectively. Open fractures and those with major comminution were significantly more likely to require revision surgery. Final limb function following recovery was assessed as good to excellent in 90.3% of cases. For the combined diaphyseal fractures, stabilisation of both bones proved an effective repair strategy with only 1/8 cases (12.5%) requiring revision versus 5/18 cases (27.8%) where only one bone was stabilised. For the combined diaphyseal fractures the two main repair methods were external skeletal fixation (ESF) or radial plating. The success rate was greater for radial plating with only 1/10 (10%) cases requiring revision versus 4/14 (28.6%) for ESF. However ESF tended to be applied to the more complicated fractures.

Synostoses and radiohumeral luxation were noted as complications associated with the fractures stabilised by ESF.

Femoral fractures: Femoral fractures are common in cats accounting for 38% of fractures in one survey.

In adult cats the medullary canal is fairly uniformly filled by a Steinmann pin (3.0 mm-4.5mm). The pin should be placed in a normograde fashion from the intertrochanteric fossa to avoid risk of sciatic nerve damage this results in the pin being placed more laterally. The pin should then be driven into the distal fragment until the tip of the pin is located level with the proximal half of the patella when the stifle is in a neutral position. The proximal pin is cut flush with the greater trochanter.

Rarely is the fracture suitable for IM pinning alone, unless the fracture interdigitates after reduction. An oblique fracture, where the length of the oblique part is at least twice the diameter of the bone, is suitable for cerclage wiring in combination with IM pinning.

Short oblique or transverse fractures may remain rotationally unstable when a single IM pin is used for fixation. Methods to reduce rotation include the addition of an external skeletal fixator (ESF), use of multiple pins (stack pins), a triangular nail, an interlocking nail or plate and screw fixation.

Plate and screw fixation

The veterinary cuttable plate is an ideal implant for use in both the feline tibia and femur. The classic 2.0mm DCP mini T plate can be used for simple distal or proximal fractures. For severely comminuted fractures which are commonly seen in the cat the plate can be applied in the 'Open But Do No Touch' method (OBDNT) or in Mini Invasive fashion. The fracture is stabilized enough to gain correct rotational and angular alignment and to maintain bone length but no attempt is made to reduce the small fracture fragments. Addition of an intramedullary pin should be considered to assist reduction and increase stability (Plate-rod technique). The aim of this technique is to leave the soft tissue envelope intact, thus minimally interfering with the blood supply and fracture healing.

Interlocking nail

The interlocking nail is useful for midshaft transverse or comminuted femoral fractures when there is enough bone distally and proximally for two (or one) screws. There are two sizes available for cats–4.7mm and 4.0mm. These are used with 2.0mm screws. The advantage of this method of fracture stabilisation over plate and screw fixation is that there is less disruption to soft tissue and the pin is placed in the mechanical axis of bone – a biomechanically advantageous site.

Complications of femoral fracture repair

In a review of 26 cats, 6 of them (23%) had sciatic nerve entrapment. The cause of sciatic nerve damage was either due to direct trauma at the time of pin insertion or related to the fibrous tissue that formed around the pin tip. In all cases pins were inserted in retrograde fashion, left longer at the trochanteric fossa and placed more medially, compared to those without sciatic problems.

In a series of 22 cats with femoral fractures repaired by internal fixation, four cases of quadriceps contracture (18%) were observed.

Tibial fractures

Tibial fractures account for approximately 10% of long bone fractures in cats. In a survey of 73 feline tibial fractures by Richardson and Thacher they concluded that classifying fractures into mild moderate or severe can help establish a prognosis. Severe fractures (open and comminuted) took longer to heal and had a higher incidence of complications including infection, delayed union, malunion and non-union.

Species differences

The tibia is a long tapered bone in the cat with a slight S bend in it. Management techniques for fracture repair include external coaptation, intra-medullary pinning, plating and external skeletal fixation.

Small bone plates can be used effectively to treat diaphyseal fractures of the feline tibia. Veterinary cuttable plates are particularly useful because of their size, versatility and ability to accommodate 1.5-2.7 mm screws. Intramedullary pins can be combined with plate and screws when using a biologic approach or to supplement stability. The addition of the intramedullary pin protects the plate from bending forces and decreases the chance of plate failure. Bone plates are applied to the medial surface of the tibia.

Simple, non-displaced fractures may be suitable for external coaptation, particularly if the fibula is still intact. The advantage of this technique is that the blood supply is not disrupted by an open surgical procedure. However cats do not always tolerate casts well, the cast may slip, sores can develop and tendon laxity can result from the joint immobilization especially in the kitten. A simple 4 pin unilateral ESF will often prove to be a suitable and perhaps more satisfactory alternative.

The tapered nature and S shape of the tibia limits the diameter of pin to a fairly narrow one that can be used in this bone. The pin should be introduced in a normograde fashion. In transverse fractures rotation must be prevented with addition of an ESF or cerclage wires when appropriate.

Distal physeal fractures – (salter harris type I or II). The distal fragment is very usually very small and thin. If there is minimal displacement external coaptation may be sufficient. Usually ORIF is preferable using two crossed K wires. External coaptation (cast or TESF) should then be provided until evidence of healing has been documented radiographically.

Complications: in Richardson and Thachers study the overall rate for osteomyelitis for 66 tibial fractures was 15%. The distal aspect of the tibia has relatively little surrounding soft tissue and risk of open fractures, displacement of fragments and avulsion of periosteum is higher than with other long bone fractures. These risk factors are associated with delayed fracture fragment revascularisation, inadequate callus formation, osteomyelitis and increased likelihood of non-union or delayed union. Placing autogenous cancellous bone graft around these distal tibial fractures is to be recommended.

Common orthopedic conditions in cats

Hip dysplasia: Hip dysplasia in cats may be detected as an incidental finding when the pelvis or abdomen is radiographed for other reasons. The lower incidence, or detection rate, is related to the smaller size and varied genetic background of cats. In addition different clinical signs are exhibited. Pure bred cats may be predisposed. In one study the incidence was reported to be 6.6%(Keller et al 1999). Radiographic signs in cats included more acetabular remodelling with minimal femoral neck changes. A study performed at the University of Pennsylvania confirmed that cats have high hip joint laxity and there is a relationship between DJD and laxity in the hip joint of cats (Langenbach et al 1998).

Slipped capital epiphysis: Slipped femoral epiphysis (metaphyseal osteopathy)

This condition is seen mainly in young male neutered cats, aged 2 years or less. Affected cats present with a unilateral hind limb lameness often of insidious onset. Radiographs show a slipped femoral epiphysis, there may be 'apple coring' of the femoral neck (Queen et al 1998). This is a hypervascular response associated with attempts to repair the fracture. Biopsies of the affected femoral neck showed evidence of fracture healing. In some cases the fracture has healed but a malunion is present. One review of 26 adult cats with spontaneous femoral capital physeal fractures suggested that they were most likely to be heavier, neutered males with delayed physeal closure (McNicholas et al 2002). Treatment is femoral head and neck excision. The other femoral head may fracture at a later date.

Hip luxation (dislocation): Hip dislocation is a common traumatic injury in cats; it is the most commonly dislocated joint in the cat. The luxation usually occurs in a dorsocranial direction, mainly due to the pull of the gluteal muscles. Lameness may vary from non-weight bearing to mild with some external rotation of the foot. Manipulation, palpation and comparison of leg length can aid in diagnosis, however fractures in this area can have similar clinical findings. Definitive diagnosis is by radiography – lateral and ventro-dorsal extended. It is best to radiograph the hip joint prior to attempting closed reduction, if fracture fragments are present or the cat has hip dysplasia / DJD or another traumatic injury then closed reduction is unlikely to be successful.

Treatment options include closed reduction, conservative, transarticular pin, ilio-femoral suture and femoral head and neck excision amongst others. The transarticular pin is a useful method of hip stabilisation in the cat, and the commonest technique we employ at CUVS. 1.6mm K wires are used, and left in for 2-3 weeks, the duration mainly dependant on the presence of other injuries. The prognosis is good for maintenance of reduction, except in bilateral cases where reluxation or of one hip is likely. Conservative treatment is an option in cats where cost is an implication, however stiffness is likely.

Cranial cruciate ligament disease

Cats do suffer cranial cruciate ligament disease. There are two main forms, traumatic and degenerative. In the traumatic form there is usually damage to other structures such as the collateral ligaments and menisci. Management of this latter condition will be discussed under the deranged stifle.

Cats with degenerative cranial cruciate ligament ruptures (or the occasional isolated traumatic rupture) will have hind-limb lameness, stifle joint swelling and the cranial drawer test will be positive. Radiographs of affected stifles will show compression of the infra patella fat pad associated with a joint effusion. Meniscal calcification can be seen especially in older animals (Reinke & Mughannam 1994, Whiting & Pool 1985). In Reinke & Mughannams (1994) paper they report on six spayed female cats, five of which had a cruciate rupture. The lameness resolved after cruciate surgery and meniscal calcification resection. Calcification may also be present in the normal stifle.

Treatment of cranial cruciate ligament rupture in cats is either conservative or surgical. Surgery may have the advantage of offering a quicker return to function and a decreased chance of osteoarthritis and meniscal damage. Generally extracapsular stabilisation techniques are suitable and the prognosis is good. TPLO and TTA has been reported to be effective in treatment of cats with cranial cruciate ligament tears as well. Meniscal tears have also been observed in uncomplicated cranial cruciate ligament tears. Partial meniscectomy results in a good outcome in these patients.

Patella luxation: Patella luxation is not common in cats, when it occurs it is generally medial and can be uni or bilateral. Both traumatic and developmental (congenital) forms are seen. The condition has been reported in the Devon and Cornish Rex, Persian and Abyssinians as well as domestic short-haired breeds (Engvall 1990). Houlton and Meynard (1989) report on 8 cats with patella luxation, six of which had bilateral disease. Conservative treatment was unsuccessful but there was a ninety percent improvement with surgery. One patella fracture occurred 6 months post operatively.

Patella fracture: Patella fractures in cats, if displaced, need surgical stabilization usually with a pin and figure of eight tension band wire. If the fracture fragments are small then these can be resected and the injury treated similar to a patella ligament rupture (Harari et al 1990, Carb 1975, Brunnberg et al 1993). The latter can be repaired with a Bunnell type suture pattern threaded through a hole in the patella. Both repairs need protection with a wire suture placed through a hole in the tibial tuberosity and around or through a hole in the patella. TESF can be used but complications can occur if the cat is inadequately immobilised (Bruce 1999).

Cats do have the radiographic appearance of bipartite and tripartite patella and these must be differentiated from acute patella fractures, although they may be chronic undisplaced fractures. If in doubt stressed radiographs are taken, with fractures the fragments should distract when the stifle is flexed .

Tarsal collateral ligament injuries

Traumatic hock injuries are common and usually associated with fractures. Occasionally cats will present with hind limb lameness associated with closed collateral ligament injury, often just the short collateral ligament may be ruptured. Prosthetic ligament reconstruction is recommended as primary repair is often difficult and ineffective. Anchorage of prosthetics is possible using small suture anchors.

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External skeletal fixation (ESF)

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External fixation systems

There are three basic types of external fixation frames – linear, free-form, and circular frames. Linear and circular frames can be combined together to make a hybrid frame. Some examples of linear systems include the Kirschner-Ehmer (K-E) system, the IMEX SKTM system, and the SECUROS system, among others. The SK and SECUROS system components may be easier to apply, however the K-E system's smaller and lighter components make them attractive for very small dogs and cats. Alternatively, a 'free-form' external fixator can be fashioned out of threaded or straight pins and polymethylmethacrylate molded to make the connecting bars.

The most widely used circular fixator systems are the IMEX SKTM circular system and the Small Bone Fixator (Hofmann S.a.S., Italy). Application of these fixators is based on the Ilizarov method and can be used to lengthen or straighten bones when combined with an osteotomy, as well as static stabilization for fractures and corrective osteotomies, and luxations.

Their use is extremely versatile, especially when combined with linear components to create a hybrid frame. Clinical advantages of external fixation External skeletal fixation (ESF) is an extremely versatile form of bone and joint stabilization in cats, dogs, and exotic species, allowing for early use of the operated limb. ESF can be used to stabilize fractures and osteotomies, as well as provide joint immobilization for the treatment of tendon repairs, luxation or arthrodesis. When applied with linear or angular motors ESF can correct angular and length discrepancies. It can be used as the sole form of fixation or as a secondary stabilization for internal bony or soft tissue repair. ESF allows for additional and often flexible options for challenging fracture configurations, particularly those adjacent joints. As the fracture heals, portions of the frame and pins can be removed to allow for increased micromotion and enhanced fracture healing, so called staged disassembly or sometimes dynamization. When applied in a minimally invasive or closed method, there is preservation of blood supply to the fracture site and adjacent soft tissues. Wound debridement, lavage, drainage, bone grafting, and skin grafting can all easily take place while external fixation is intact. When ESF is the primary form of stabilization, all implants are removed when fracture healing is complete, thus it is ideal for use in infected tissues, where internal implants may become a nidus for infection.

Limiting factors for ESF

While external fixators are extremely versatile, the individual fracture configuration, location, affected bone, and available equipment will play a role in the usefulness of a particular form of external fixation. Additionally, one must consider the available soft-tissue corridors for pin and connecting bar placement. The most common limiting factors are joint interference and the body wall for humeral and femoral fractures. One must not underestimate the importance of patient and client selection and consultation. Owners must be informed and educated on the postop appearance, home care, and recheck requirements for external fixators. Temperament and lifestyle of the patient must also be considered. If the animal is feral, aggressive, or unable to be kept confined and monitored while wearing the frame, the use of external fixation should be carefully considered.

Frame selection and application

The injury type (soft tissue injury versus open, closed, comminuted, simple, or transverse fractures), location (bone, metaphyseal, diaphyseal, intrarticular), patient characteristics (age, weight, activity level) all play a role in planning the frame application. The biomechanics of ESF have been reviewed previously, and variables which may be altered to tailor the frame to the specific case include pins (type, number, configuration, the shank and thread diameter, the thread shape and pitch), fixator type (Ia, Ib, II, III), and connecting rods (material, number, configuration). Pins should be placed through 'safe corridors' in order to avoid important structures including neurovascular bundles, large muscle masses, and the body wall. The ideal placement of unilateral frames is medial for the tibia, medial or craniomedial for the radius, craniolateral for the humerus and lateral for the femur. The body wall prevents bilateral frames in the humerus and femur, except in very leggy dogs. ESF allows for a completely closed approach to the fracture site which enhances healing due to preservation of the hematoma and blood supply. When applied in a closed or minimal approach, alignment of the limb is of utmost importance. Placing the proximal and distal pins parallel to their respective joints and then aligning those two pins parallel to each other can achieve alignment. Assistance with alignment and limb length restoration can be had by using the hanging-leg technique and through the use of fluoroscopy.

Pin placement

To ensure maximal stability, pins should be placed in the center of bone. Threaded pins are preferred to smooth pins, as they have a stiffer bone-implant interface and resist loosening. Smooth pins must be at angled to the

surface of the bone in order to reduce pull-out, maximize stiffness, and decrease loosening. Adequate placement of smooth pins can be challenging in small dogs or with small fracture segments. Pins must be placed no less than 2-3x's the pin diameter or greater than 1/2 diameter of bone from fracture planes. At least 2-4 pins should be placed in each fracture segment. The pin clamp connection should be placed close to the skin but with enough room to allow for soft tissue swelling. Shorter pins are stiffer. Stiffness can also be increased by adding connecting bars. Prior to pin insertion, a 1cm longitudinal skin incision should be made and blunt dissection performed to the level of the periosteum. With the aid of a drill guide and a stickette drill bit, the hole should be pre-drilled to approximately 80% of the pin diameter (typically 0.1mm smaller) under low speed (<300 rpm). The pin can then be inserted with power, under lowspeed. For linear frames, it is often easiest to insert the most proximal and then most distal pins first, then attach the connecting bar and finally place the subsequent pins by drilling through clamps attached to bar. Specialized aiming devices are available for Type II frames.

Circular fixators

The minimal fixation for each segment is either four wires in each segment or two wires and one to two 1/2 pins in each segment of bone. Wires may be either smooth or with a stopper (olive wire). Wire size is predominantly chosen based on patient weight: 1.0mm if < 10 kg, 1.2mm if 10-20 kg, 1.5mm or 1.6mm if > 20 kg, as is the tension which should be applied to the wires: none if < 5 kg, 20-30 kg if 5-10 kg, 30-60 kg if 10-20 kg, 60-90 kg if >20kg. There is a 30kg maximum tension on partial rings and posts. Circular or partial rings may be attached to linear bars with the aid of hemispheric washers and bolts for added versatility.

Post-operative care

After frame application, leave pins long prior to initial post-op radiographs, so that adjustments can be made if needed. The bolts should then be retightened and pins cropped. A bandage should be placed on the limb with sponges placed between the skin and frame to provide light compression, decrease swelling and oedema. Pain management typically consists of NSAIDs and opioids and should be tailored to the specific patient. Animals should be confined for the duration of fixation and care taken to protect the frame. Pin tracts may be gently cleaned with a dilute 0.05% chlorhexidine solution. The frame should be assessed weekly and radiographs performed every 3-5 weeks until the fracture or osteotomy has healed. If a limb is being distracted, radiographs should be taken more f adjustments, and then every 3-4 weeks. Linear frames can undergo staged disassembly to enhance bone healing. Such planned removal of fixator bars and pins decreases the rigidity of the frame and increases micromotion at the fracture site, which in turn may accelerate healing. Pins with morbidity should be removed first. Typically, frames may be down-staged at 4-6 weeks post-operative, but timing should be based on the degree of bone healing present on radiographs. Frame removal can typically be performed under heavy sedation with the use of a pin cutter and hand chuck. A light wrap may be placed on the limb for 8-12 hours if bleeding occurs from the pin tract sites. Exercise should be restricted for an additional 3-4 weeks after frame removal to allow bone to fill in the pin tracts.

Complications

Complications of ESF are common, however most are minor and can be managed conservatively. Major complications of ESF (4%) less than ORIF with bone plate (18%). (Dudley et al JAVMA 1997) Complications can be either mechanical (pin, wire, clamp breakage) or biological in nature. Biologic complications can include wire/pin tract drainage, pin loosening, ring sequestrum, osteomyelitis, delayed union, nonunion, or malunion, fracture, hemorrhage, neuropraxia, and loss of range of motion. Fortunately, most of these complications can be avoided with adherence to the principles of ESF application, good post-operative fixator care and physiotherapy for the patient. The most common complications include pin tract discharge, infection, and subsequent loosening. Pin tract infections can be treated by bacterial culture and sensitivity, appropriate antibiotic therapy and pin removal or replacement. Pin or wire breakage requires removal or replacement of the implant. Malunion, non-union, or delayed union require a critical assessment possible causes and subsequent frame adjustments and often autogenous cancellous bone grafting.

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Acknowledgments Dr. M. S. Bergh

How to diagnose carpal instability

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Learning objectives

- Discuss the anatomical characteristic of the feline musculoskeletal system.
- List the most common fractures of the feline fore and hind limb.
- Discuss the optimal methods of fracture repair according to the different anatomical locations.
- Recognize the most common orthopedic pathologies affecting the feline patient.

The carpus is classified as a hinged (ginglymus) joint, consisting of three joint levels and four rows of bones.

The most proximal joint is the antebrachio-carpal (ABC) or radiocarpal joint, where the radial carpal and ulnar carpal bones articulate with the distal articular surfaces of the radius and ulna, respectively; it contributes for the 70% of the carpal ROM. The middle or intercarpal (MC) joint consists of the distal articular surfaces of the radial carpal and ulnar carpal bones and the proximal articular surfaces of C1 through C4; it contributes to the 25% of the total carpal ROM.

The carpo-metacarpal (CM) joint is the most distal and is made up of the distal articular surfaces of C1 through C4 and the proximal articular surfaces of metacarpals one through five and contributes only for a 5 % of the motion.

The ABC joint communicates with the Accessory Carpal Bone joint space on the palmar aspect of the carpus and is separated from the MC joint. The MC and CM joints communicate between them.

Soft tissues structures responsible for supporting the carpus include ligaments, tendons, the flexor retinaculum and the palmar fibrocartilage. The medial and lateral collateral ligaments consist of the short radial collateral ligament and short ulnar collateral ligament respectively. The short radial collateral ligament originates on the tubercle of the radius just above the styloid process and inserts on the most medial aspect of the radial carpal bone. The short ulnar ligament originates on the styloid process of the ulna and inserts on the ulnar carpal bone. Short and thin ligaments connect the dorsal aspect of each carpal bone and the distal carpal bones with the dorsal aspect of the base of the metacarpal bones. A distinct ligament connects the dorsal articular margin of the radius with the dorsal most proximal aspect of the Ulnar carpal bone.

Palmar support is offered by the thin palmar ligaments, the palmar fibrocartilage distally (it includes only the MC and CM joints) and the flexor retinaculum. The accessory carpal bone articulates with the ulnar carpal bone and two strong accessorio-metacarpal ligaments connect the distal free end of the bone to the palmar aspect of the metacarpals Iv and V. This two ligaments act as a stay apparatus that is a system where tendons and ligaments act in synergy to stabilize a joint in a physiological weight bearing position minimizing muscular energy expenditure (Fig.1 – 2 – 3).

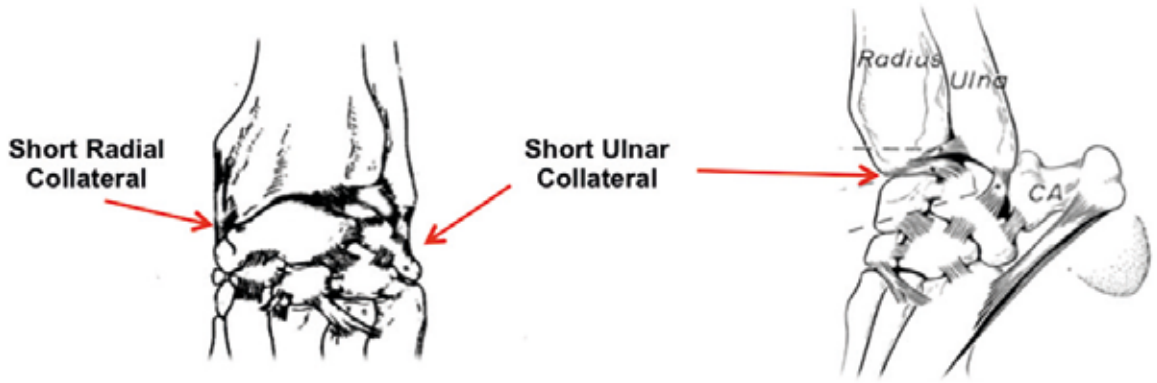
The flexor carpi ulnaris is the only tendon that significantly contributes to the carpal stability in extension originating from the caudo-medial surface of the olecranon and from the medial epicondyle of the humerus it inserts on the dorsal proximal aspect of the accessory carpal bone.

In dogs and cats, each forelimb carries approximately 30% of the weight during a normal stride. This weight bearing, combined with high-impact forces from running, jumping, or trauma, can predispose the carpus to hyperextension injuries. The normal range of flexion is approximately 100° for the ABC joint, 40° for the MC joint, and 10° for the CM joint. The normal standing angle of the carpus is approximately 140° to 180° in dogs and 160° to 180° in cats.

A detailed knowledge of the radiographic anatomy of the distal extremities is necessary to demonstrate a suspected condition or injury affecting these specific anatomic locations.

Clinical examination consists in a series of maneuvers that start with palpation of the bony prominences and individual joint spaces, palpation of the surrounding musculo tendinous structures followed by an accurate evaluation of the range of motion and stability of the individual articular levels. Any abnormality or pain response is recorded and compared to the normal contralateral carpus.

Damage to ligaments most commonly occurs following substantial trauma. Ligament damage may occur in combination with substantial adjacent soft tissue (shear wound) or from excessive force placed across a joint. Less commonly, ligaments may be disrupted by sharp laceration. Whatever the cause, if external forces exceed the tensile strength of the ligament, disruption of the collagen bundles results, leading to various degrees of instability. Ligaments are very inelastic and collagen fiber bundles become permanently deranged after 10% elongation.



LIGAMENT	ORIGIN	INSERTION	FUNCTION
Short RCL Straight portion	Radial Styloid	Medial aspect RCB	Prevents valgus in extension
Short RCL Oblique portion	Radial Styloid	Palmaro-medial aspect of RCB	Prevents valgus in flexion
Short UCL	Ulnar Styloid	UCB	Prevents varus along full ROM

Figure 1 – Carpal collateral ligaments.

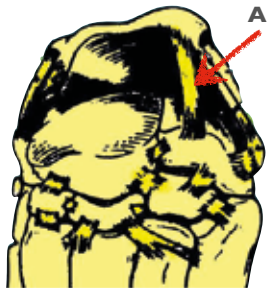
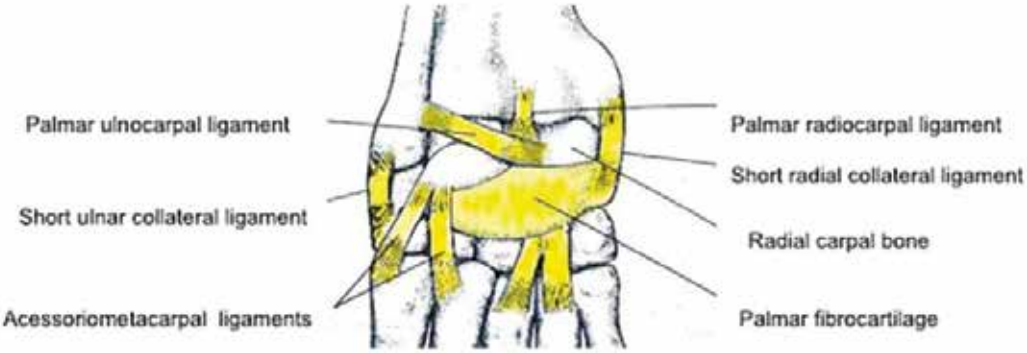


Figure 2 – Dorsal Inter-carpal and carpo-metacarpal ligaments. The red arrow (A) points the dorsal radio-carpal ligament.



LIGAMENT	ORIGIN	INSERTION	FUNCTION
Palmar RCL	Palmar border of radius	Palmar aspect of RCB	Prevents hyperextension of ABC Joint
Palmar UCL	Palmar aspect of ulnar styloid	Palmar aspect of RCB	Prevents hyperextension of ABC Joint
Palmar fibrocartilage	All carpal bones	Bases of Metacarpals II to IV	Prevents hyperextension of middlecarpal and carpo metacarpal joints

Figure 3 – Carpal palmar ligaments.

Ligamentous injuries are classified as grade 1, grade 2 or grade 3.

A grade 1 injury is associated with disruption of relatively few collagen fibrils and little functional compromise. This type of injury is commonly seen with mild collateral ligament injuries of the carpus and tarsus. The ligament is grossly intact. Palpable or radiographic instability is likely not apparent. Diagnosis of a grade 1 injury is made based on history, pain on palpation and stressing of the joint, and evidence of mild soft tissue swelling at the site of injury.

A grade 2 injury has greater amount of collagen damage, resulting in functional deficits and joint instability. Increased swelling and hematoma formation is seen. Collateral ligament injuries are often associated with grade 2 injury. Documentation of instability can generally be accomplished by stress radiographic views. It is often helpful to compare radiographs of the abnormal to the normal joint. A significant amount of the ligament has been disrupted with grade 2 injury, but a portion of the ligament remains intact. Grossly, a portion of the ligament may appear torn or simply stretched.

A grade 3 injury is associated with complete tearing of the ligament, resulting in total loss of ligament function. Palpable and radiographic instability is readily evident. This type of injury is commonly associated with subluxation or dislocation of the joint.

Radiographic examination of the carpus starts with routine views. Due its anatomical complexity, visibility of these regions in routine radiographic views is obscured by the superimposition of the different bony structures. For this reason, in order to make or confirm a suspect diagnosis, supplemental or additional radiographic views are often required.

Additional radiographic views may consist of special projections as flexed or extended oblique views and stress views.

Stress projections are, by definition, views taken during "the application of controlled force to a joint to demonstrate abnormal spatial relationship between two or more of its components" (Farrow 1982).

Stress views can be obtained by application of shear, traction, wedge and rotatory maneuvers. When ligamentous injuries are suspected, stress views are invaluable to demonstrate the presence of subluxation, joint instability or to evaluate the location and size of avulsed fragments.

An interesting variation of stress views are, whenever possible, the weight bearing views, taken with the dog in standing position and the x-ray beam in horizontal position in respect to the weight bearing limb.

Whenever doubts about the radiographic anatomy exist, it is extremely useful to x-ray the contralateral normal limb.

- Routine Projections
  - Medio-Lateral (ML)
  - Dorso-Palmar (DP)

- Additional Projections
  - Dorso-Lateral Palmaro-Medial Oblique 45 degrees (DLPMO)
  - Dorso-Medial Palmaro-Lateral Oblique 45 degrees (DMPLO)
  - Medio-Lateral Flex (ML-Flex): this view is helpful to evaluate the dorsal articular margin of the radius, the dorsal surface of the radial carpal bone and the articular surface of the accessory carpal bone.

- Stress Views
  - ML hyper extended view: useful to evaluate palmar instability, subluxation of the radial carpal bone and proximal carpal joint, damage of the palmar fibrocartilage, subluxation and luxation of the carpo-metacarpal joints, fractures affecting the accessory carpal bone. (Fig.4).

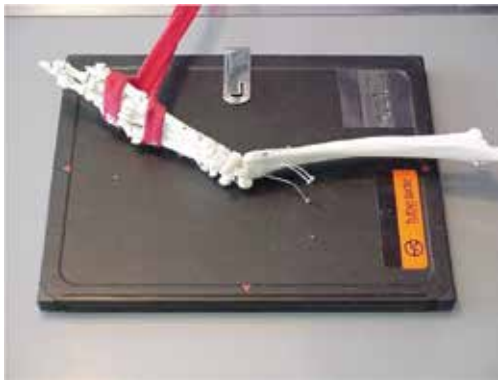


Figure 4 – With the patient in lateral recumbence and the affected limb directly over x-ray cassette, push the palmar surface of the foot with a paddle, forcing the foot into a hyper extended position. This view has some limitations and may not show the level of carpal instability.

– DP views with application of lateral or medial stress (varus and valgus stress): very helpful views in case of suspected collateral instability, damage to the collateral ligaments with avulsed fragments from the styloid process, subluxation of the second carpal bone, carpo metacarpal collateral instability (Fig.5).

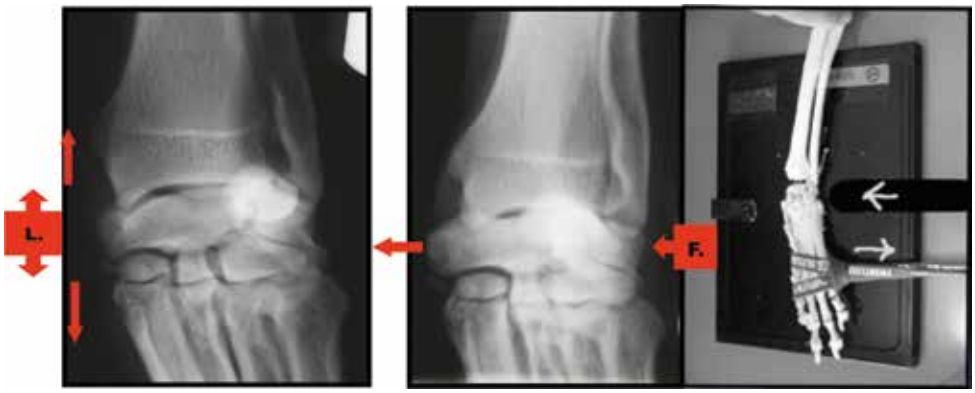


Figure 5 – Stress views with application of fulcrum and counter traction. Placing the dog in sternal recumbence, extend the affected limb cranially; after securing the metacarpal area with a tie, position a radio transparent stick that will act as a fulcrum. Pulling the tie and holding firmly the fulcrum the opposite joint space will be distracted.

– Standing ML view is in this Author opinion the most sensitive and useful technique to evaluate palmar instability. With the patient in standing position the affected limb is allowed full weight bearing by lifting the opposite unaffected limb. The radiographic beam is placed perpendicular to the x-ray cassette by lowering the X-ray machine head (Fig. 6–7).

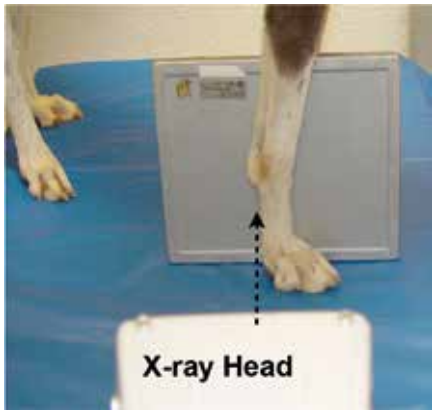


Figure 6 – With the patient in standing position the affected limb is allowed full weight bearing by lifting the opposite unaffected limb. The radiographic beam is placed perpendicular to the x-ray cassette by lowering the x-ray machine head.

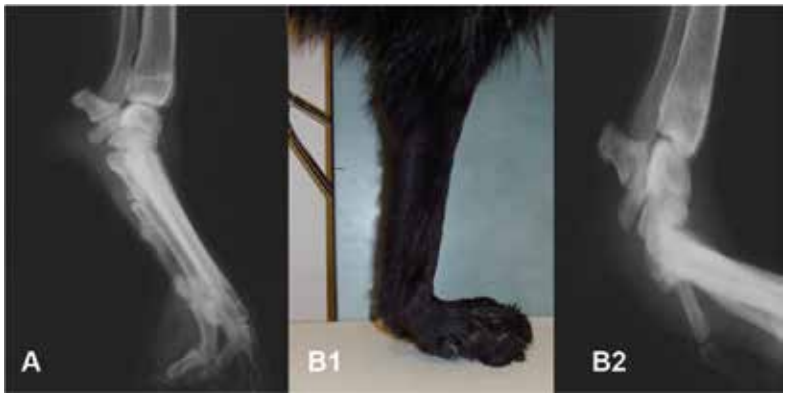


Figure 7 – Stress view in weightbearing position.

A: ML hyperextended view. The palmar subluxation and hyperextension is obvious but is not possible to exactly identify the affected joint level.

B1-B2: The same leg is placed in weightbearing position and the x-ray shows the exact location (carpo-metacarpal) of the subluxation.

Dedicated views

Specific radiographic views are sometime necessary to deeply evaluate some of the carpal bones or the dorsal articular margin of the radius.

Dorsal articular margin of the radius

This region can be affected by fracture avulsion of the origin of the dorsal radio-carpal ligament hairline fracture of the dorsal margin or incomplete fracture of the radial or ulnar styloid.

Palmaro-Medial Dorso-Lateral hyper flexed oblique view (PMDL-Flex)

Palmaro-Lateral Dorso-Medial hyper flexed oblique view (PLDM- Flex)

With the carpus in direct contact with the x-ray cassette position the limb at an angle that vary from 30 to 45 degrees (Fig. 8).

Figure 8 – Palmaro-Medial Dorso-Lateral hyper flexed oblique view (PMDL-Flex) and Palmaro-Lateral Dorso-Medial hyper flexed oblique view (PLDM- Flex).

Sky line or tangent views are invaluable for a complete evaluation of the dorsal aspect of the radius. These views can be associated with other views to confirm the suspect of a fracture or chip affecting this region. Incomplete fractures and small chips can sometimes be detected only with the aid of these views (A. Piras. Free communication, Montecatini, Italy, 1998)

Sky line views: lock the wrist at the maximum degree of flexion that is possible to achieve and secure it with a strip of tape, with the dorsal aspect of the distal row of carpal bones in direct contact with the x-ray cassette, position the beam tangent to the dorsal articular margin of the distal radius with an angle that can vary between 20 to 45 degrees. Two or three views are obtained at different angles to allow a complete evaluation of the region.

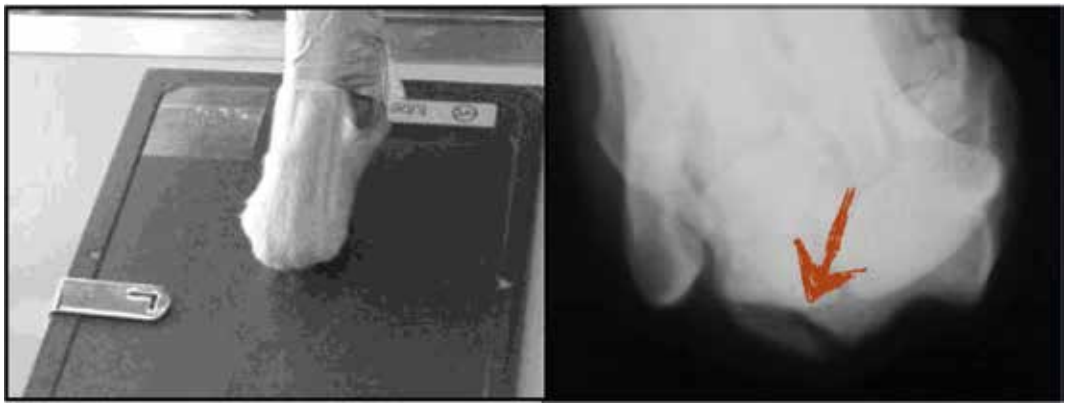


Figure 9 – Left: positioning of the limb for the sky line view. Right: red arrow shows a fracture of the dorsal articular margin of the radius in a sky line view.

Classification of Palmar Instability.

In 1982 Slocum classified Carpal Hyperextension in 3 categories defining the characteristics of the instability and relative indications for pancarpal versus partial carpal arthrodesis (Partial carpal fusion in the dog–B. Slocum, T. Devine–JAVMA, Vol.180, No.10 – 1982).

The 3 categories were defined by clinical examination and radiographic evaluation with valgus and varus stress and weightbearing.

Slocum Category 1: rupture of the oblique short radial collateral lig.–rupture of the palmar radiocarpal lig.–rupture of the palmar ulnocarpal lig. (Fig. 10-11).

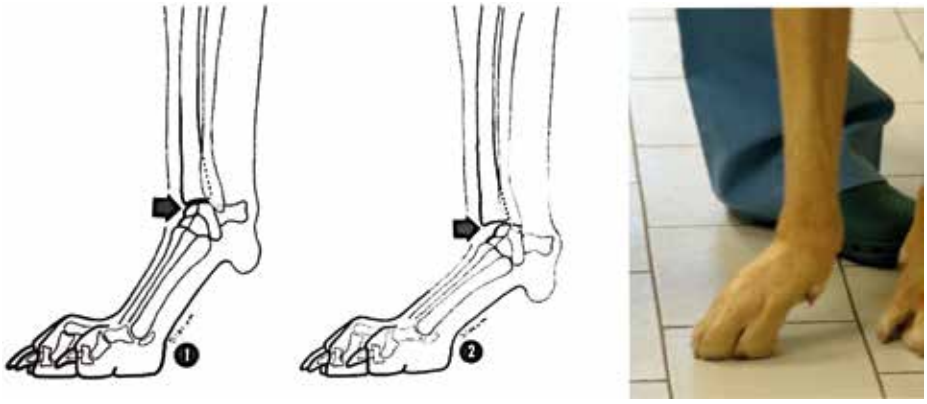


Figure 10 – Category 1 Slocum: Palmar subluxation and collateral instability. Detectable at clinical examination. Confirmed radiographically.

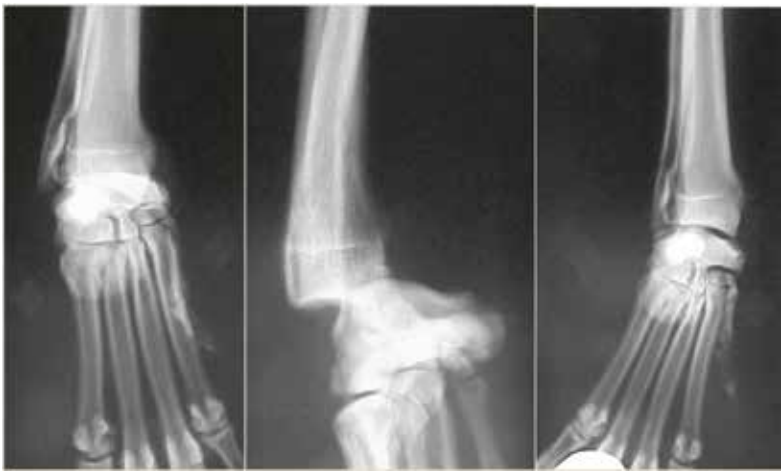


Figure 11 – Category 1 Slocum: Axial rotation instability due to palmar subluxation of the radial carpal bone if only the radial collateral is involved stress view shows valgus deviation.

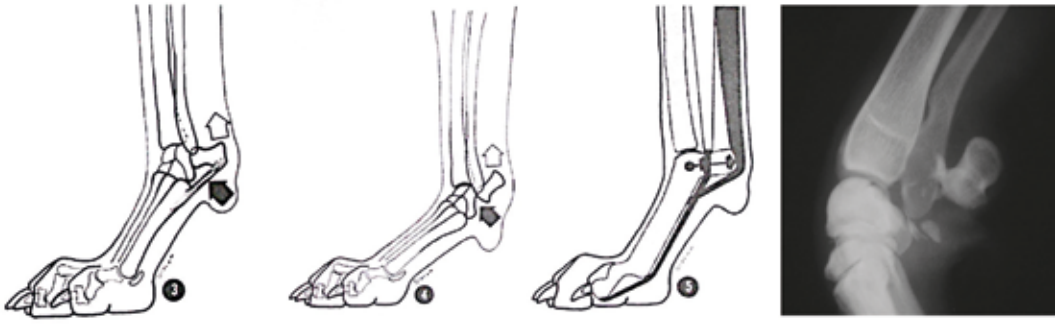


Figure 12 – Category 2 Slocum: Rupture of the accessory-metacarpal lig.–rupture of the accessory -ulnocarpal lig. Unrestricted hyperextension of the ABC joint, detectable at clinical examination, palmigrade stance and unrestricted instability of the Accessory Carpal Bone.

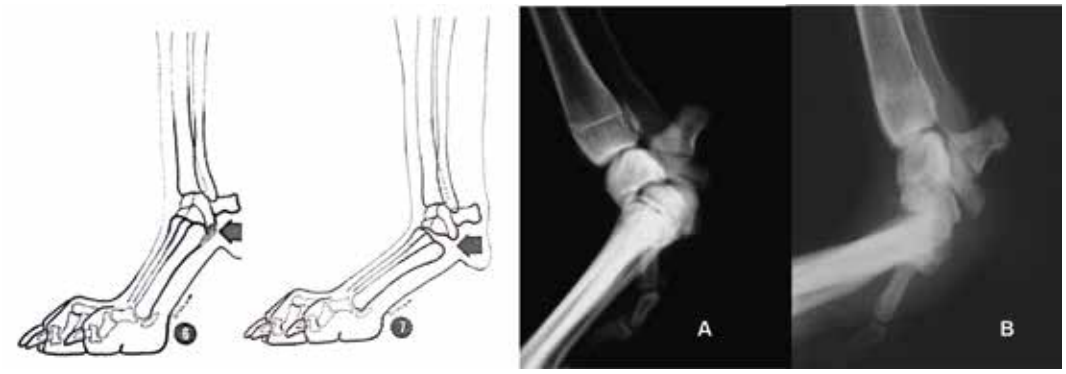


Figure 13 – Category 3 Slocum: Rupture of the middle carpal joint ligaments (A), or/and rupture of the carpometacarpal ligaments (B), rupture of the palmar fibrocartilage (A & B).

Clinically evident hyperextension and palmigrade stance but very difficult to assess the location of the instability. Weightbearing x-ray stress views are 100% diagnostic.

Slocum indications for pancarpal versus partial carpal vary slightly from the recommendations by Piermattei (1981) and Willer (1990). According to Slocum pancarpal arthrodesis is indicated for Categories 1 and 2 and partial carpal arthrodesis only for category 3. While maintaining the same indications for pancarpal arthrodesis for category 1 and partial carpal for category 3, the above mentioned Authors extend the indications for partial carpal arthrodesis also for category 2.

Pancarpal 	**Partial carpal** 	
Category 1 Category 2	Category 3	Slocum 1982
Category 1	Category 2 Category 3	Piermattei 1981 Willer 1990

The accessorio metacarpal joint is a very important stabilizer of the carpus and is very unlikely that damage at this level do not involve other important palmar stabilizers like the palmar radiocarpal and ulnocarpal ligaments. Treatment of category 2 instability by accessorio-carpal arthrodesis has been described either in the current literature and anecdotally reported. This Author experience overlaps Slocum recommendations of pancarpal arthrodesis for category 2 instability.

References available upon request.

Surgery

Jackie Demetriou (UK)

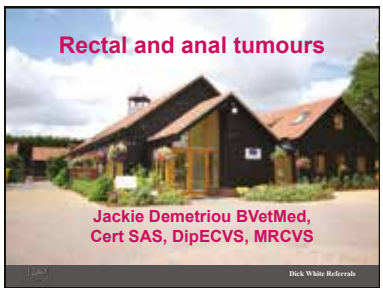
BVetMed, CertSAS, DipECVS, MRCVS

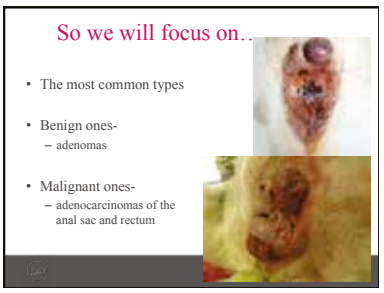
European and RCVS-Recognised Specialist in Small Animal Surgery

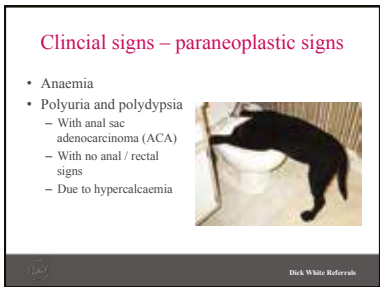
Honorary Associate Professor, University of Nottingham. Jackie qualified from the Royal Veterinary College London, in 1996 and spent one year in mixed practice before joining Edinburgh Veterinary School as an intern. She completed a Residency in Small Animal Surgery in 2001 and after a year in a private referral practice, was appointed Lecturer in Small Animal Surgery at the University of Cambridge. She joined DWR in 2012. Jackie passed her RCVS Certificate in Small Animal Surgery in 2000 and the ECVS Diploma in 2002. She has served on the European College of Veterinary Surgery examining board for 5 years. She enjoys all aspects of soft tissue surgery, from clinical work and teaching to participating in clinical research. She publishes widely in peer reviewed journals, has published a textbook on Small Animal Oncology with Rob Foale and is co-editor and contributor to the only dedicated book on feline soft tissue surgery. She contributes regularly to national and international conferences and surgery courses. She has undertaken advanced training in Interventional Radiology (IR) and laparoscopy.

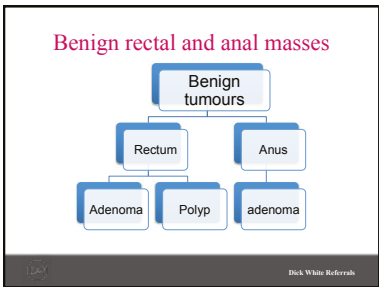
Rectal and anal tumours

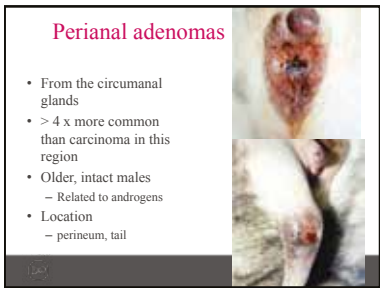
Jackie Demetriou BVetMed, CertSAS, DipECVS, MRCVS

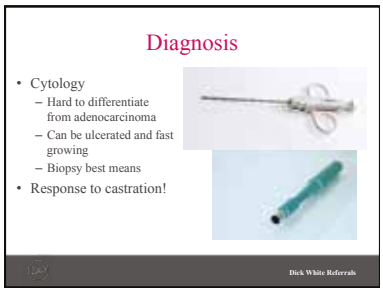


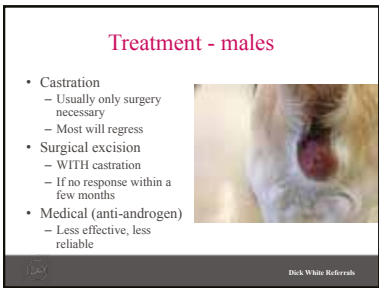


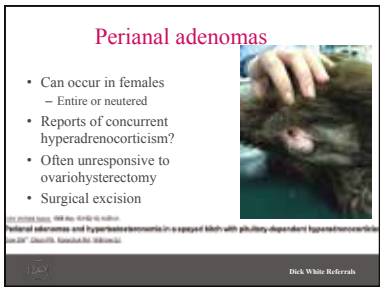


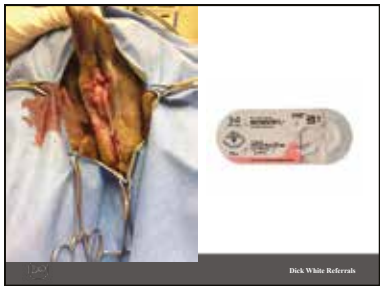


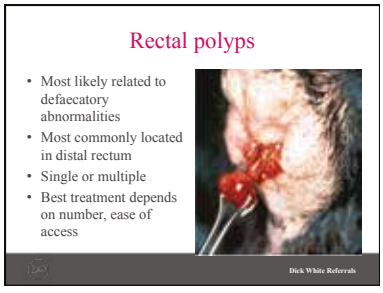


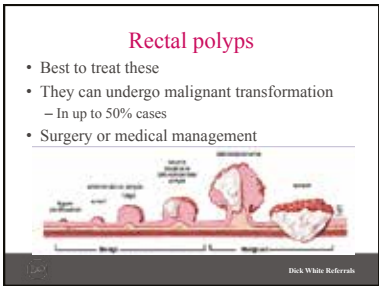












What surgery can we do?

Single distal polyp

- Pull-out rectal procedure

Multiple polyps throughout rectum

- Cauterise via endoscopy
- Local or systemic piroxicam therapy

Dick White Referrals

Multiple polyps

- Piroxicam capsules
  - NSAID
- Either systemically (0.3mg/kg/every other day) or
- intra-rectally (suppositories)
  - 0.3mg/kg/day
  - Every second or third day with smaller patients

Dick White Referrals

Multiple polyps

- Endoscopic cauterisation
  - For extensive lesions
  - Recommend concurrent anti-inflammatory
  - Monitor for complications
  - May need repeat treatments

Dick White Referrals

Single polyp

- Fortunately most are distal rectum
- Pull out procedure recommended
- Usually resection is not full thickness
- Complications
  - Stricture formation

Dick White Referrals

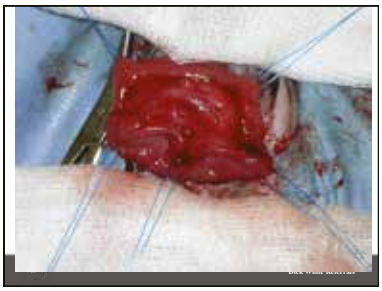
Pull-out procedure

- Simple equipment
  - Suture material
  - Haemostat forceps
  - suture kit
  - a helper!
- Progressive placement of stay sutures to evert the rectum

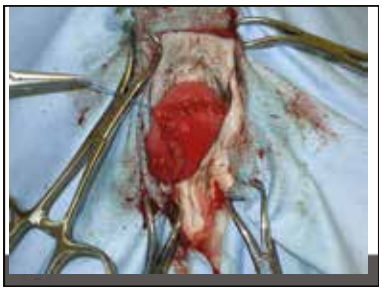
Dick White Referrals

Dick White Referrals

Dick White Referrals



Dick White Referrals



Dick White Referrals

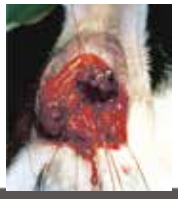
Adenocarcinoma

- Diagnosis
  - Clinical signs
  - Endoscopy
  - Biopsy is best
  - Stage
- Types
  - Pudunculated (distal rectum)
  - Infiltrative (middle rectum)
    - These often cause stricture

Dick White Referrals

Surgery

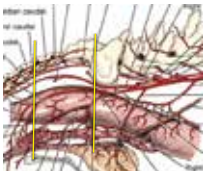
- Depends on type (single or diffuse?)
- Depends on location
- Distal rectum
  - Can often access with a pull-out procedure
  - Often palliative (margins not achieved)



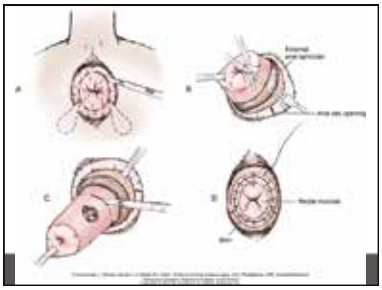
Dick White Referrals

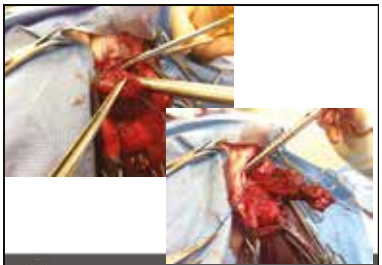
For more cranial tumours

- Pull-through procedure
- OR
- Ventral abdominal celiotomy and pubic symphysiotomy



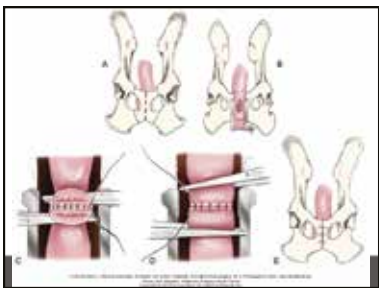
Dick White Referrals





Dick White Referrals

Dick White Referrals



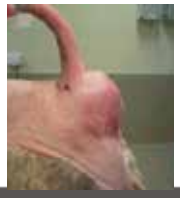
Prognosis for adenocarcinoma

- Depends on type
  - Annular mass : worse prognosis (1.6 months)
  - Nodular mass : 12 months
  - Single mass : best prognosis (32 months)
- Depends on treatment
  - Surgery results in greater survival times
  - No proven benefit of additional medical therapies

Dick White Referrals

Anal sac adenocarcinoma

- Malignant tumour
- Frequent metastasis
- Often associated with hypercalcaemia



Dick White Referrals

Diagnosis

- Breed type
  - Cocker Spaniels
- Gender
  - Males and females (equal)
- Age
  - Older dogs but variable

Dick White Referrals

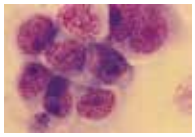
Clinical signs

- Local signs
  - Recurrence anal sac problems
  - Perineal mass obvious
  - Rectal palpation
  - Tenesmus / constipation
- Systemic signs
  - Polyuria/polydypsia
  - Hypercalcaemia
  - Pseudohyperparathyroidism
  - PT like hormone

Dick White Referrals

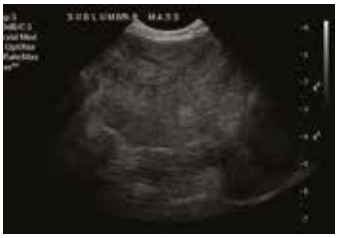
Investigations

- Local palpation
- Aspiration – cytology
- Extent of disease
  - Radiography
  - Ultrasonography
  - CT



Dick White Referrals

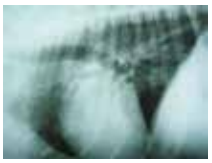
Dick White Referrals



Dick White Referrals

Metastasis

- 50% will have at the time of diagnosis
- Rate of metastasis is very variable
  - Local lymph nodes (medial iliac)
  - Liver
  - lungs



Dick White Referrals

Treatment

- Correct hypercalcaemia
- IV fluid therapy for diuresis
- + / - fruseminde

Dick White Referrals

Surgery

- With just local disease, small tumour
- Simple anal sacculectomy
- Monitor thereafter for progression / metastasis

Dick White Referrals

Surgery – what do we do with metastatic disease?

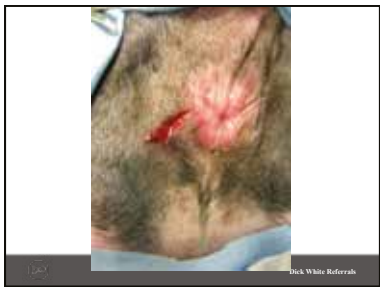
- Surgery is still the best treatment
- Local surgery
- Excision of sublumbar lymph nodes

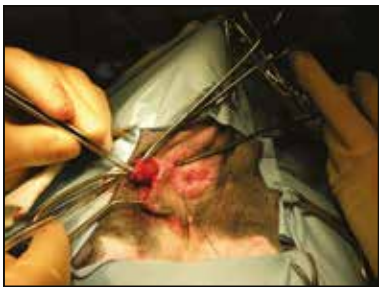
Dick White Referrals

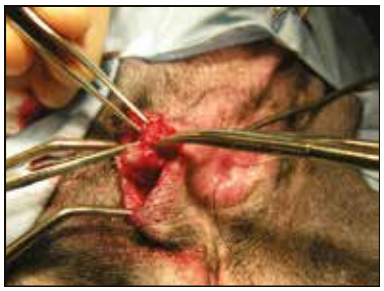
Anal sacculectomy

- Closed technique
  - Do not open anal sac
- Difficult to get a good margin
- Local recurrence is low despite close margins

Dick White Referrals







Complications of anal sacculectomy

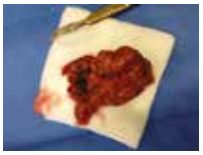
- Incontinence
  - Something often vets worry about
  - Rare especially if unilateral procedure
- Infection
  - Careful wound monitoring and hydrotherapy

Surgery – lymph node excision

- Aims
  - to stage ie. has metastasis occurred?
  - to improve clinical signs of tenesmus
  - to reduce the tumour volume for additional chemotherapy / radiotherapy

Surgery – lymph node excision

- Will increase the morbidity
  - Duration of surgery
  - Risks – haemorrhage
- Some are easy to remove and some are really fixed



Additional therapies

- Chemotherapy
  - Cisplatin
  - Carboplatin
  - Mitoxantrone
  - Palladia / Masivet
  - Some respond
- Radiotherapy
  - Local and / or lymph nodes

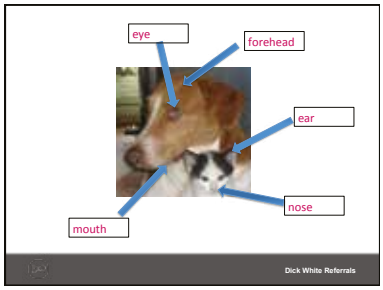
Prognosis for anal sac adenocarcinoma

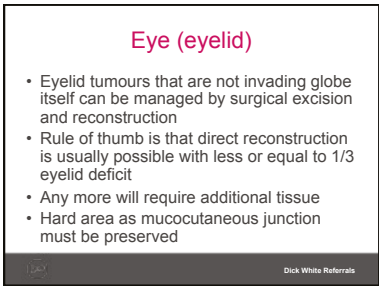
- Around average 550 days
- Worse if
  - Large tumour size
  - Metastasis
  - No surgery
- So some form of surgery is indicated

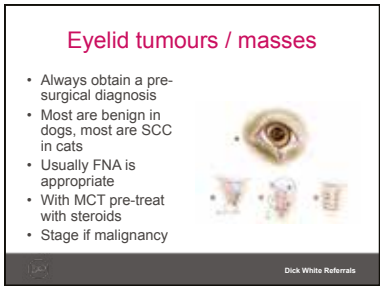
Any Questions?

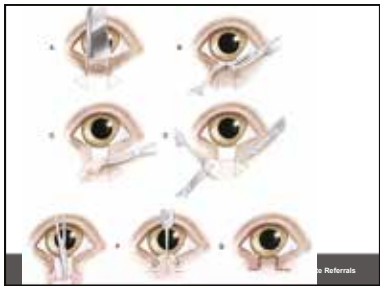
Tumours around the head and neck – what are the surgical options?

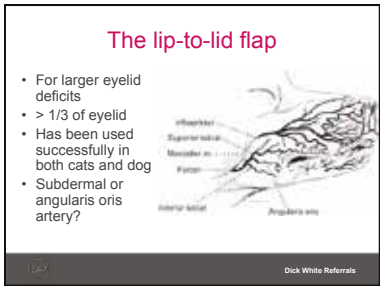
Jackie Demetriou BVetMed, CertSAS, DipECVS, MRCVS

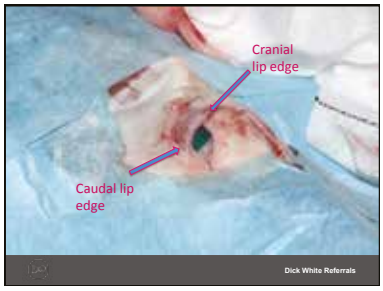




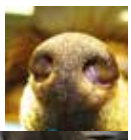




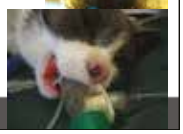




The nose



- External nares
  - Majority are surgical
- Nasal cavity tumours
  - Majority or not surgical
  - Radiotherapy treatment of choice
  - Will not talk about in this lecture!
- DOGS ≠ CATS!



Clinical signs

- Deep lesions
  - Crusting and erythema
  - Superficial erosions
  - Ulcers
  - Deeply invasive and erosive lesions
- Can confuse all forms with eosinophilic granulomas

Dick White Referrals

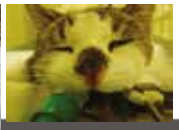
Diagnosis

- Visual exam, rhinoscopy, MRI/CT
- Biopsy!
  - Incisional
  - Deep enough to avoid surface inflammation
- Assess and aspirate regional lymph nodes
  - Submandibular
- Radiograph thorax
  - Distant metastasis rare (especially in the cat)
  - MRI/CT to determine extent

Dick White Referrals

Treatment

- Superficial minimally invasive SCC
- Deep infiltrating disease



Dick White Referrals

Superficial minimally invasive

- Surgery
- Radiotherapy
- Photodynamic therapy
- Intralesional carboplatin

Dick White Referrals

Deeply invasive SCC

- Surgery is the treatment of choice
- Complete excision can be hard
- Local recurrence may occur
- Nasal planum resection
  - In dogs may need to combine with a pre-maxillectomy

Dick White Referrals

Nasal planum resection

- Fully stage first
  - Lymph nodes
  - Thoracic met check
  - **Extent of local disease**
- Discuss with owners
  - Cosmetic appearance
- Partial or complete resection

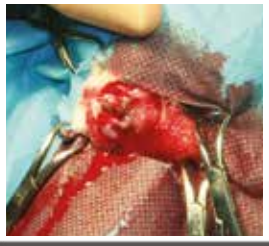
Dick White Referrals

Dick White Referrals

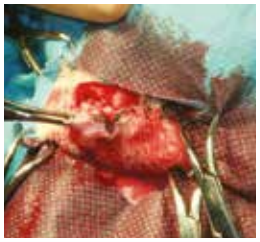
Case - Archie

- 13 year old DSH
- Lesion left sided
- Extending laterally
- Crossing midline
- SCC
- > 4mm deep
- Plan
  - Bilateral nasal planectomy

Dick White Referrals



Dick White Referrals



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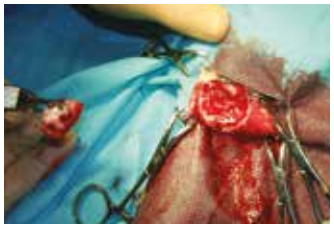


Dick White Referrals

Dick White Referrals

Dick White Referrals

Dick White Referrals

Nasal planum tumours - dogs

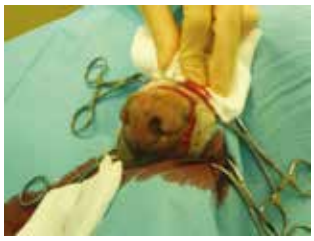
- Tend to originate caudal to planum
- Further imaging / extent assessment
- Often combine with pre-maxillectomy to obtain margins

Dick White Referrals

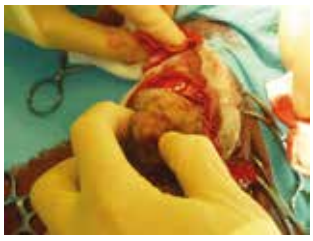
Case - Max

- 6 year old labrador
- Mainly R sided but involving left septum
- Traditional approach
  - Similar to cats
- Without pre-maxillectomy for superficial lesions

Dick White Referrals



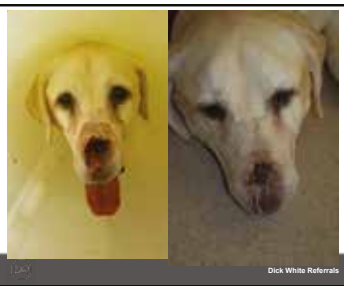
Dick White Referrals



Dick White Referrals

Dick White Referrals

Dick White Referrals



Dick White Referrals

Combined with pre-maxillectomy

- Standard method
- Or
- Nasal reconstruction
  - Island labial flap
  - Better cosmesis
  - May reduce stenosis, dehiscence as direct mucosal apposition

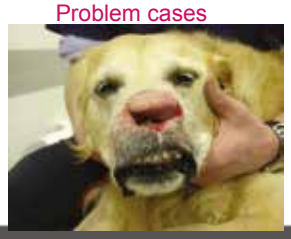
Dick White Referrals

Nosectomy/premaxillectomy

Dick White Referrals

Dick White Referrals

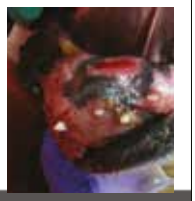
Problem cases



Dick White Referrals

Oral tumours

- Common area for a wide variety of malignant and benign cancers
- Most common type is the oral epulis in dogs
- Most common malignant tumours
  - Melanoma, SCC, fibrosarcoma in dogs
  - SCC in cats
- Bone often involved
- Surgery often treatment of choice



Dick White Referrals

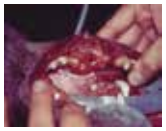
Clinical signs

- Halitosis
- Difficulty in mastication
- Oral haemorrhage
- Ptyalism
- Swelling
- Pain, weight loss
- Mass often not noticed

Dick White Referrals

Aims of treatment

- Determine type
- Determine location
- Determine local invasion
- Determine metastatic spread

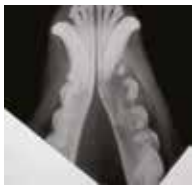


Dick White Referrals

Imaging

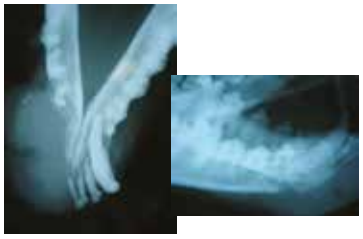
- Local invasion
- Distant metastases
- GA required
  - Intraoral views
- Conventional radiographs not very sensitive
- CT and MRI

Dick White Referrals



Dick White Referrals

Dick White Referrals



Dick White Referrals

Surgery

- Aim:
  - Local control, preservation of function
- Simple excision/debulking
  - Benign epulis (except acanthomatous)
  - Debulk prior to radiotherapy
- Radical excision
  - Mandibulectomy/maxillectomy
  - Wide margins (2cm)

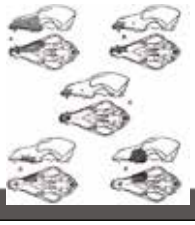
Dick White Referrals

Mandibulectomy

- Rostral
  - Symphysis to PM1
- Central
  - PM1 to M1
- Caudal
  - Caudal to M1
- Hemi-mandibulectomy
- Three-quarter

Dick White Referrals

Maxillectomy



Dick White Referrals

Mandibulectomy

Dick White Referrals

Dick White Referrals

Dick White Referrals

Dick White Referrals

Dick White Referrals

Dick White Referrals

Maxillary osteosarcoma

Dick White Referrals

After excision

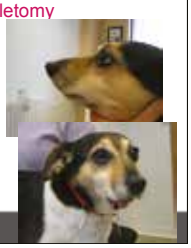
Dick White Referrals

Cosmetic appearance

Dick White Referrals

Cosmetic appearance - mandibuletomy

- Premaxillextomy with canine tooth removal
  - Worst appearance
  - Drooping nose
- Retention of canines
  - Excellent cosmesis
- Mandibulectomy
  - Tongue
  - cheiloplasty



Dick White Referrals

Aural tumours

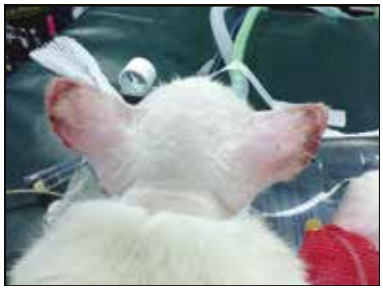
- External pinna
- External ear canal
- Middle ear

Dick White Referrals

Pinna

- Most commonly SCC in cats
- Similar pathogenesis to nose
- Very unlikely to metastasise
- Surgery is curative

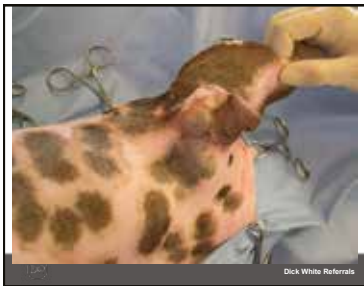
Dick White Referrals



Tumours in external ear canal

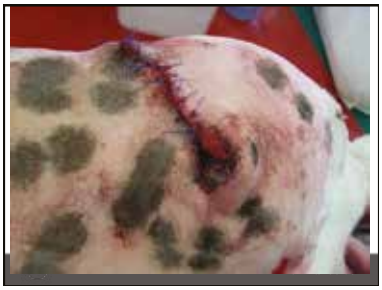
- If located proximal pinna
  - Pinnectomy or secondary intention healing
- If located distal pinna close to cartilage of vertical canal
  - Vertical canal ablation is usually warranted

Dick White Referrals



Dick White Referrals

Dick White Referrals



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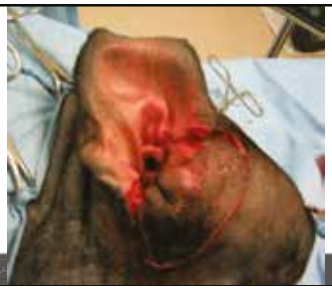
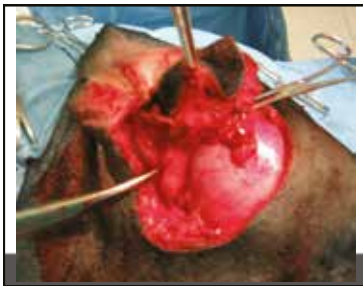
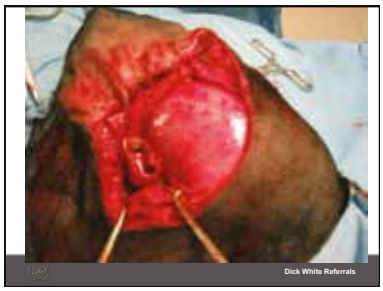




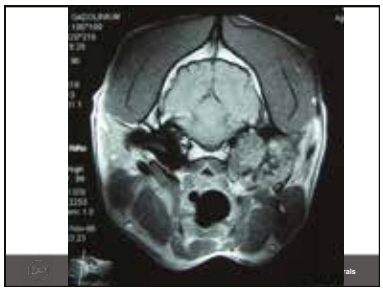
Dick White Referrals

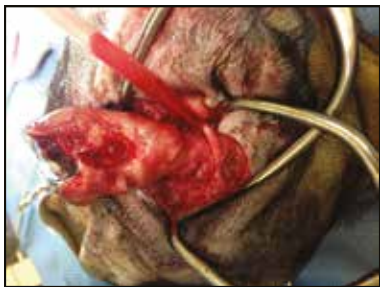

Dick White Referrals

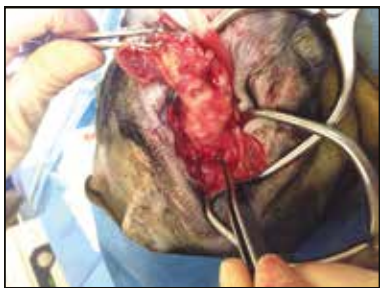

Dick White Referrals

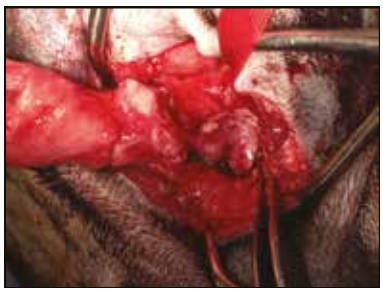

Dick White Referrals

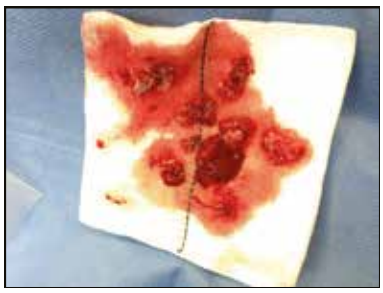

Tumours in horizontal ear cannal or middle ear
• Total ear canal ablation is required
• Imaging helpful guide for degree of local extension
Dick White Referrals












Tumours around the face and forehead – see next lecture!
Dick White Referrals

Tumours around the head and neck – case studies

Jackie Demetriou BVetMed, CertSAS, DipECVS, MRCVS

Head and Neck Cases

Case – bonnie, 1 year old FN DSH

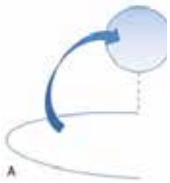
- Cat from local rescue centre
- Congenital eyelid dyspasia
- Too short – entropion and repeated ulceration

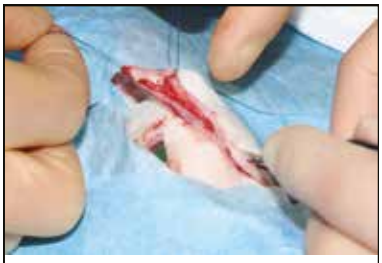
Case - bonnie

- What can we use to extend the lid margins?
- A flap that can also be used to replace lid margins following tumour excision (most common use)

The lip to lid

- Uses the junction of the caudal upper lip skin and labila mucosa as an interpolation flap
- Subdermal plexus or ? Axial pattern by contribution from angularis oris artery



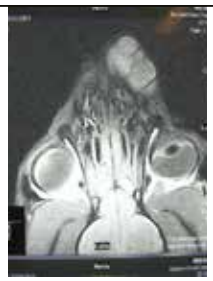


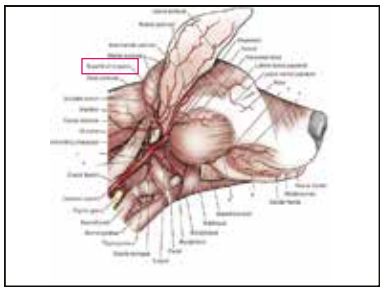
Wensley – 19 year old M GSH

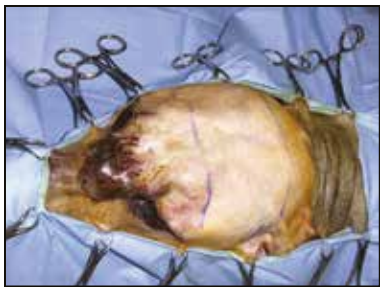
- Presented with a slowly growing mass between eyes
- Recent increase in growth rate
- Biopsies – low grade sarcoma
- Staging clear

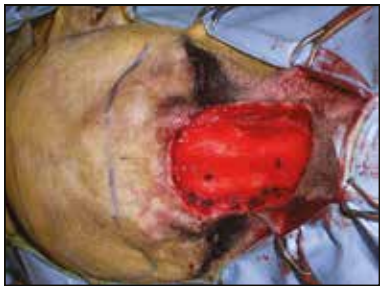
What to do next

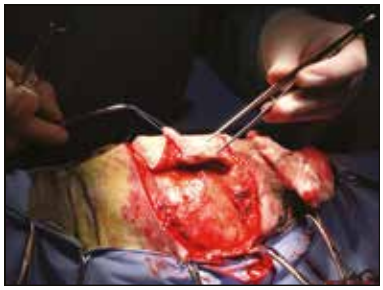
- Distant staging negative
- Local extent
- CT / MRI scan

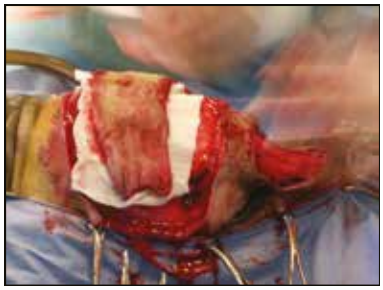


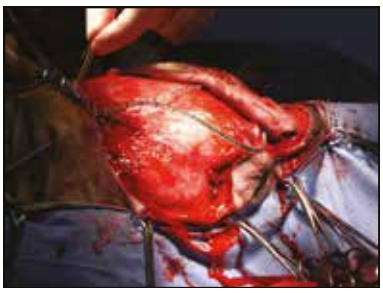






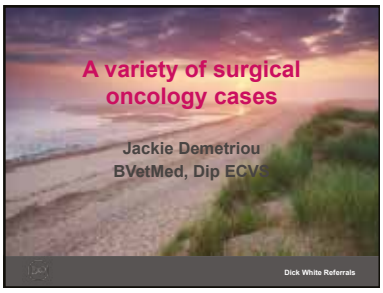


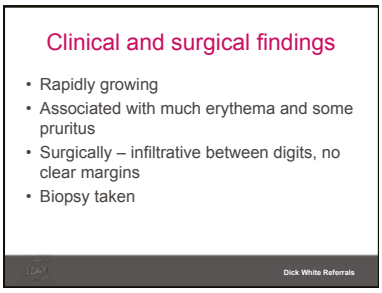


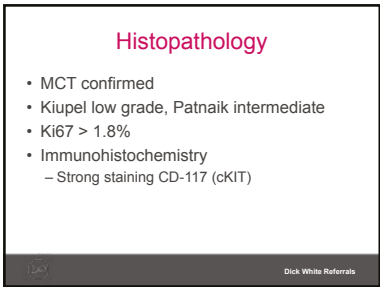


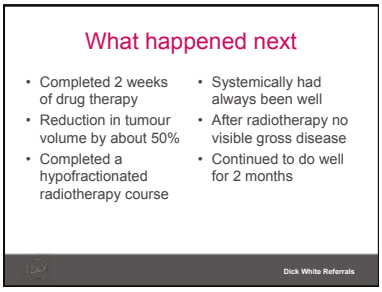
A variety of surgical oncology cases

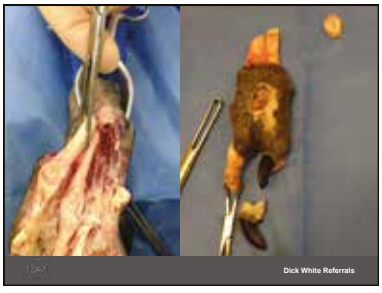
Jackie Demetriou BVetMed, CertSAS, DipECVS, MRCVS

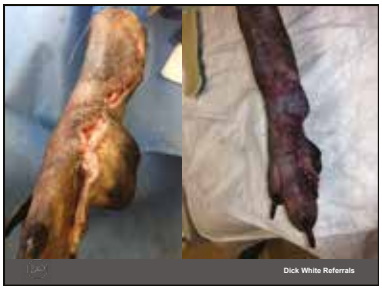


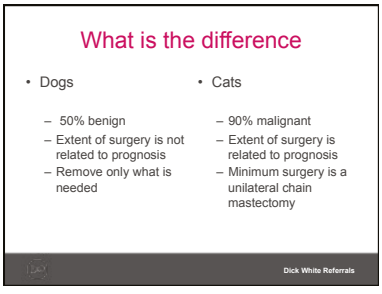












Dissect down to body wall

Dick White Referrals

Dick White Referrals

Dick White Referrals

Dick White Referrals

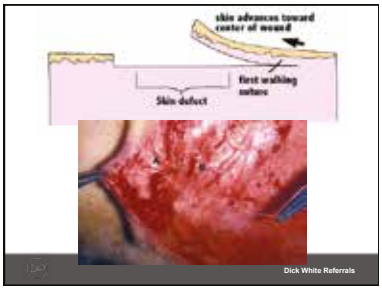
Dick White Referrals

Dick White Referrals

My top tips to reduce tension

- Limited undermining
- Walking sutures ++
- Effective haemostasis
- Multiple layer closures

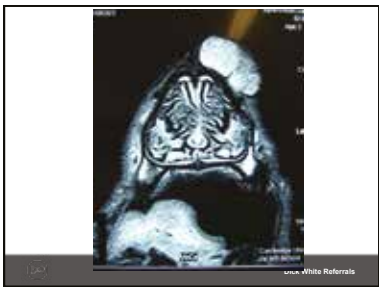
Dick White Referrals



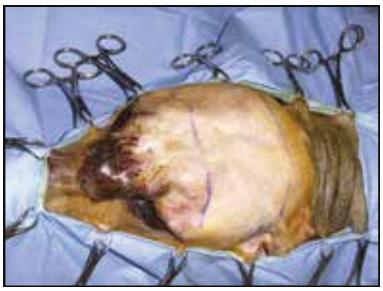
Dick White Referrals

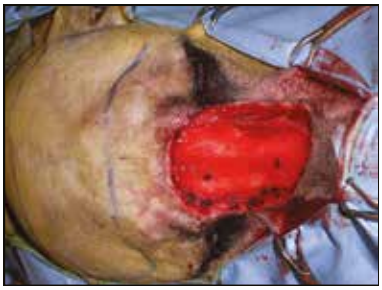
"Wensley" a 7 year old M GSD

Dick White Referrals



Dick White Referrals





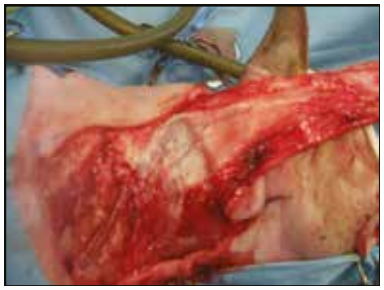
Superficial temporal

Dick White Referrals

"Wilf" 5 year old Terrier cross

- Fast growing STS on forehead
- In exactly location of superficial temporal flap!
- What other option?
  - Advancement
  - Caudal auricular

Dick White Referrals



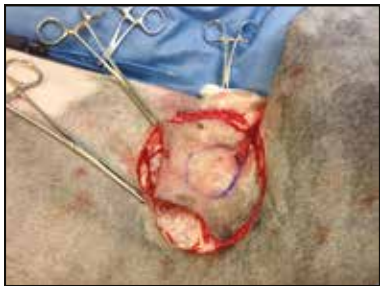
The caudal auricular

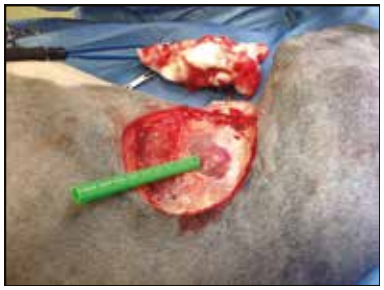
Dick White Referrals

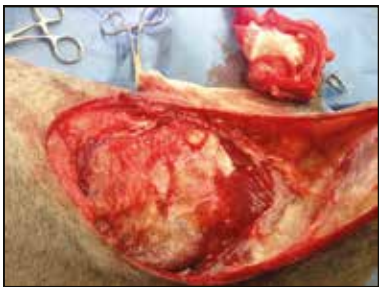
Ralph, 5 year M Border Collie

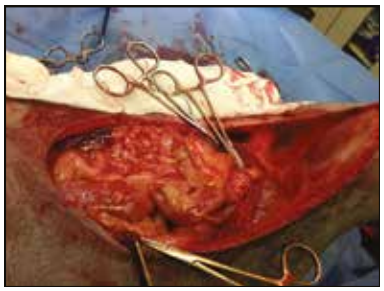
- Mass noticed on lateral flank area
- FNA diagnosis, strongly suggestive of STS
- Surgery planned following staging

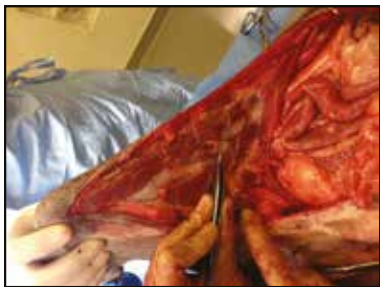
Dick White Referrals

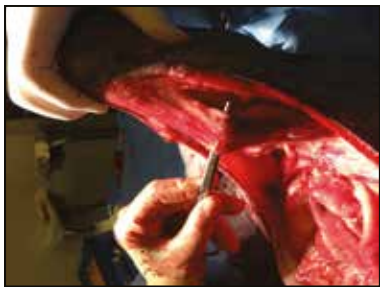


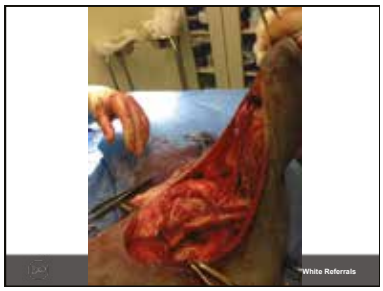


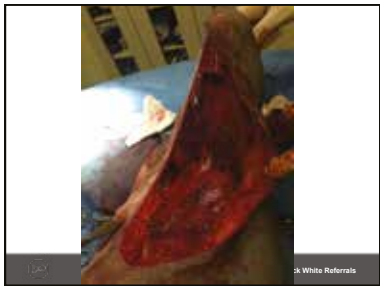












2 year follow-up

- Normal mobility 1 month post-operatively
- High grade STS
- Complete excision
- Monitored and re-staged every 3, then 6 months

Dick White Referrals

Thoracic wall masses

- Chondrosarcoma
  - Good news
  - Do well with surgery
  - DFI > 1000 days
- Osteosarcoma
  - Bad news
  - Do better with surgery than without
  - DFI about 200 days with chemo

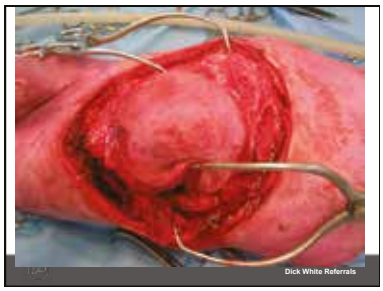
Dick White Referrals

Oakey, 2 year Cocker Spaniel

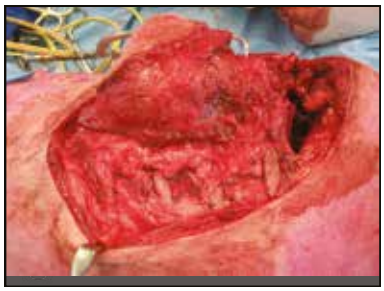
- Large osteosarcoma, caudal ribs
- Over 4 rib spaces
- Rib resection and reconstruction planned after CT staging

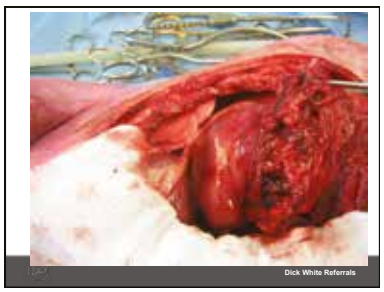
Dick White Referrals

Dick White Referrals



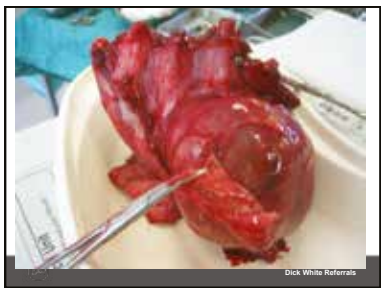
Dick White Referrals





Dick White Referrals

Dick White Referrals

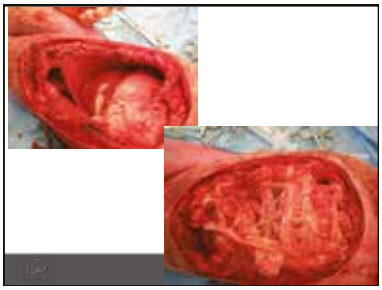


Dick White Referrals

Thoracic wall reconstruction

- Advance diaphragm
- Lasttissimus dorsi flap
- Mesh + omentalisation
- Other flaps (rectus abdominis)
- Useful for ribs 9+
- Always try to preserve
- Polypropylene
- For more caudal defects

Dick White Referrals



Thoracic wall reconstruction

- Probably up to 8 ribs possible
- 2 or less do not need reconstruction
- Prgnosis dependent only on
  - Type of tumour
  - Whether surgery has occured

Dick White Referrals

Henry, 8 year M old black lab

- 3 week history of a black mass in metatarsal pad
- Painful and lame
- FNA suggestive of melanoma
- ?benign or malignant

Dick White Referrals

Henry

- Occupied almost entire metatarsal pad
- Essential for walking
- Margins required removal of majority
- Decision to biopsy to obtain more accurate information

Dick White Referrals

biopsy

- Either tru-cut or a small skin punch biopsy
- Consider seeding
- No need to obtain "normal" tissue
- Confirmed malignant melanoma

Dick White Referrals

Henry

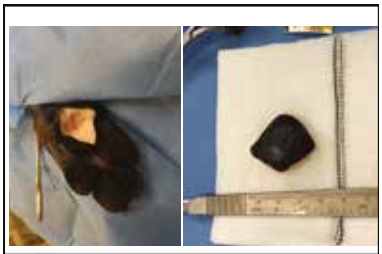
- Staging
- Abdominal ultrasound
- FNA (if indicated abdominal lymph nodes)
- Aspirate popliteal LN
- Thoracic radiographs or consider whole body CT

Dick White Referrals

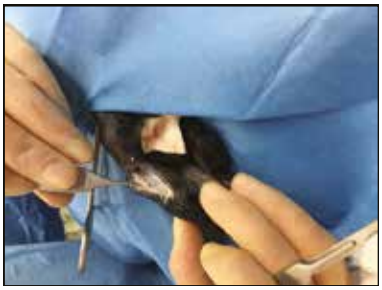
Surgery planned

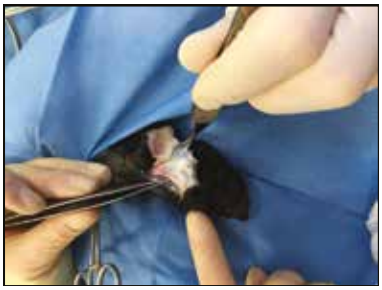
- Considerations
  - Margins for melanoma?
  - How to reconstruct
  - Options
    - Graft
    - Flap
  - Flap preferable due to consistent vascular supply

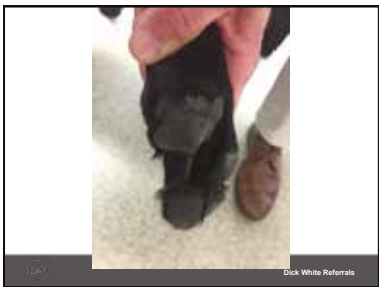
Dick White Referrals



Dick White Referrals







Dick White Referrals

Outcome for Henry

- We managed him postoperatively with metronomic chemotherapy
  - Piroxicam and chlorambucil
- Did well for 4 months
- Polpliteal involvement
- Surgical excision confirmed metastasis
- 8 months still alive

Summary

- Oncological surgery needs planning
- Find out what you are dealing with
- Best outcomes are associated with first surgery
- Surgery is the best option for most tumours but think about combined approaches
- Consider proven good outcomes before deciding it is not a good idea
- Careful discussion with owners
- Consider referring
- Remember- tumours do not always read the text books!

Advanced skin flaps and grafts – parts 1 and 2

Jackie Demetriou BVetMed, CertSAS, DipECVS, MRCVS

Advanced skin flaps and grafts

Jackie Demetriou BVetMed, Cert SAS, DipECVS

Dick White Referrals

Summary

- Skin flaps
  - Subdermal plexus, axial pattern, local, distant, composite
- Skin grafts
  - Meshed, non-meshed, split / full thickness
  - Postoperative care
- Specific skin diseases
  - Cancer surgery, axillary wounds, dermoid sinus, skin folds, ulcers

Dick White Referrals

Subdermal plexus flaps and axial pattern flaps

Dick White Referrals

Skin flaps

- Portion of skin elevated and moved from one area to another
- Relies on an intact circulation
- Classification
  - Circulation
  - Location
  - composition

Dick White Referrals

Skin flaps

- Composition
  - + muscle = myocutaneous flaps
  - E.g. latissimus dorsi or cutaneous trunci flaps to close thoracic or abdominal defects
  - Limited use in small animals

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Skin flaps

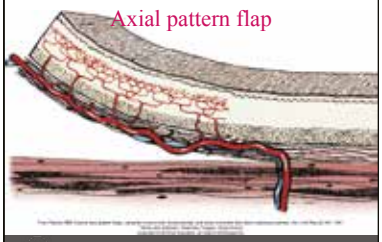
- Circulation
  - ➢+ direct cutaneous artery and vein = axial pattern flap
  - ➢- direct cutaneous artery and vein = subdermal plexus flaps

Dick White Referrals

Subdermal plexus flap

Dick White Referrals

Axial pattern flap



Dick White Referrals

Axial pattern flaps

- Flaps that specifically include a direct cutaneous artery and vein
- Several described
- Much larger area can therefore be elevated
  - 50% more than subdermal plexus flaps

Dick White Referrals

Axial pattern flaps

- What does this mean for the patient?
- Larger (> 50%) larger area can be taken
- A more reliable vascular supply
- Limited locations

Dick White Referrals

Axial pattern flaps

- Omocervical
- Thoracodorsal
- Deep circumflex iliac
  - Dorsal and ventral branches
- Cranial and caudal superficial epigastric
- Genicular
- Humeral
- Reverse saphenous

Dick White Referrals

Thoracodorsal flap

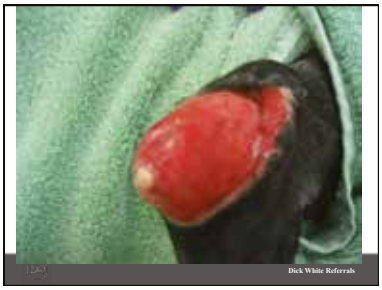
- Landmarks
  - Spine of scapula
  - Dorsal spinous processes
  - Humeral process
- Wounds of
  - Axilla
  - Shoulder
  - Forelimb
  - thorax

Dick White Referrals

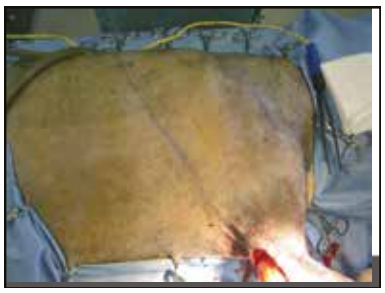
Thoracodorsal flap

Dick White Referrals

Dick White Referrals

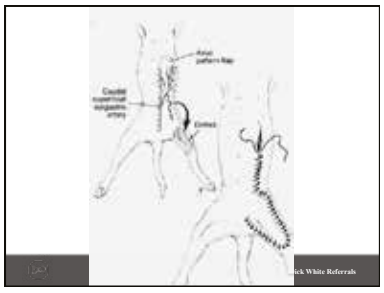


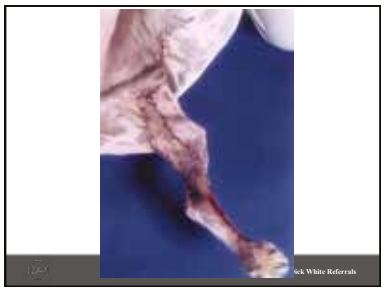
Dick White Referrals

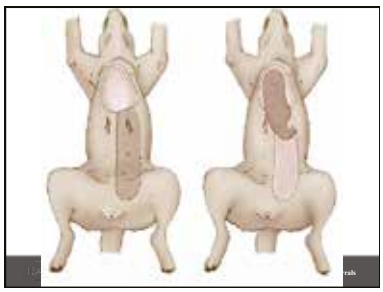


Caudal superficial epigastric

- Landmarks
  - Abdominal midline
  - Mammary glands
  - Prepuce in males
- Wounds of:
  - Inguinal region
  - Flank
  - Stifle
  - Perineum
  - prepuce







The omocervical

- Useful for neck areas
- Almost the symetrical version of the thoracodorsal
- Small dogs / cats can extent to forehead

Deep circumflex iliac (dorsal brance)

- Upper thigh, perineal regions
- Will not extend as distally as the epigastric

Dick White Referrals

Surgical considerations

- Clip wide
- Patient positioning
- Atraumatic technique
  - Avoid electrocautery
  - Use skin hooks, stay sutures
  - Avoid tension, pointed corners
- Use drains
- ? Bandaging

Dick White Referrals

Drains

Dick White Referrals

Flap considerations

- Simplest method
- Assess skin tension and pliability
- Better survival
  - Base of flap wider than width
  - Limit length to that required to cover defect
- Undermine beneath cut trunci or deep fascia (where cut trunci not present)
- Include direct a and v with large flaps
- Two or more flaps often preferable to one larger one

Dick White Referrals

Flap considerations

- Recipient bed
  - Free of debris, necrotic tissue, infection
  - Excise chronic granulation bed
  - Remove epithelialised borders

e Referrals

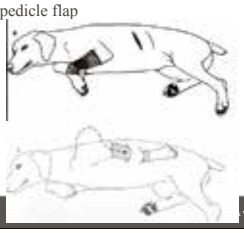
Distant direct flaps

- Single pedicle

Referrals

Distant direct flaps

- Bipedicle flap



White Referrals

Distant direct flaps

- Single pedicle or bipedicle
- Donor area
  - Lateral thorax / abdomen
  - Width of flap = width of defect
  - Limb secured under flap
    - Easiest with lateral limb defects
  - Survival depends on establishment of circulation from flap onto defect
  - Division in stages (1/3 every 2 days)

Dick White Referrals

Distant direct flaps

- Bandaging and limb security essential
  - Patient and owner cooperation
  - Movement minimised
  - Protect area
  - Tape and stay sutures
  - Change every 3 days
- Generally successful
  - Time and ££

Dick White Referrals

Skin grafts

- Differ from flaps
  - Rely on a process of revascularisation
- Vascularised recipient bed is required
- Unsuitable areas to graft onto
  - Tendon, fat, bone, chronic granulation tissue, contaminated wounds
- Suitable surfaces
  - Fresh granulation tissue
  - Fresh surgical wounds

Dick White Referrals

Suitable surface

Dick White Referrals

Unsuitable surface

Dick White Referrals

Process of graft taking

- Plasmatic imbibition
  - Tissue fluid that accumulates between graft and bed is absorbed by capillary action
  - First 48 hours
- Innosculation (“kiss”)
  - New capillaries from the recipient bed unite with graft plexuses

Dick White Referrals

Process of graft taking

- Neovascularisation
  - New capillaries form and grow into the graft
- For a graft to take regeneration must overtake degeneration by day 8
- Factors that will inhibit graft taking
  - Fluid, motion, inadequate graft contact (too big or too small), infection

Dick White Referrals

Types of grafts

- Full thickness
- Half thickness
- Meshed
- unmeshed

Dick White Referrals

Grafts

- Full thickness
  - Epidermis and dermis
  - Good cosmesis
  - Easy to harvest
  - Better durability
- Half thickness
  - Epidermis and some dermis
  - Special equipment
  - No hair
  - ? better circulation

Dick White Referrals

Grafts

- Meshed
  - Multiple slits created
  - Good drainage
  - Can stretch graft
  - Can suture in slits
- Unmeshed
  - ? Better cosmesis
  - Need drain

Dick White Referrals

Non-meshed

Dick White Referrals

Practical skin grafting

- Preparation of donor bed
- Preparation of recipient bed
- Harvesting the graft
- Meshing the graft
- Applying the graft
- Postoperative care

Dick White Referrals

Preparing the donor bed

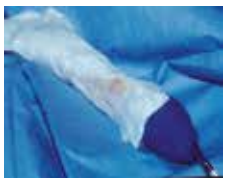
- Wide clip
- Careful prep
- Choose area
  - Skin closure
  - Hair colour and growth direction

Dick White Referrals

Preparing the recipient bed

- Careful clip and prep
- With fresh surgical wound
  - No further preparation
- With granulating wound
  - Scrape surface
  - Freshen edges
  - Blood imprint

Dick White Referrals



Dick White Referrals

Harvesting the graft

- Use blood imprint to mark area required
- Remove skin to level of subcutaneous tissue
- Stay sutures to handle graft
- Place "upside down" on cardboard and stretch out

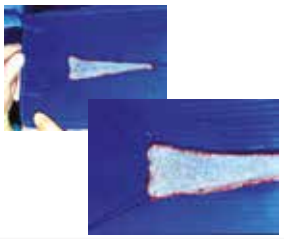
Dick White Referrals

Dick White Referrals

Preparing the graft

- Remove the subcutaneous tissue with sharp scissors
- Hair follicles should be visible easily
- Keep moist with sterile saline at all times

Dick White Referrals



Dick White Referrals

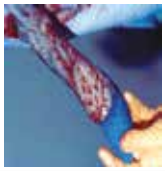
Meshing the graft

- No 11 scalpel blade
- 0.5cm length incisions
- 1cm apart
- Space evenly
- Parallel

Dick White Referrals

Application of the graft

- Place in correct orientation
  - Hair growth
- Ensure good contact
- Trim excess skin
- Suture
  - SI nylon/polypropelene
  - staples



Dick White Referrals

After care

- Layer of antibiotic ointment (no steroid)
- Non-adherent dressing
- Swabs on second layer
- Absorbent material
- Tertiary layer
- KEEP IMMOBILE
  - Consider ESF, splints, casts

Dick White Referrals

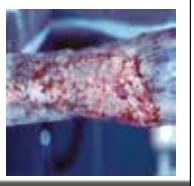
Aftercare

- Do not change bandage until 48 hours after surgery
- Sedate and extreme care in removing bandage
  - Warm saline to ease off
- Sterile bandage changes
  - Every 48 hours

Dick White Referrals

Aftercare

- Bandage until graft has taken
  - 2 weeks
- Expect colour changes
  - Purple/blue (2 days)
  - White or black – bad news!
  - Unless whole graft is necrotic leave suspicious areas until end



Dick White Referrals

Skin graft

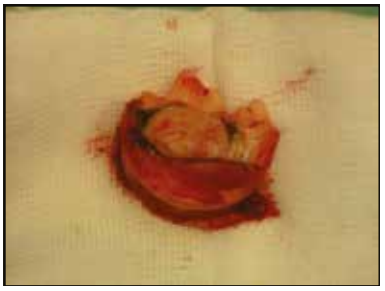
Dick White Referrals

Surgery for specific disease

Cancer surgery

- Aim to excise with margin of normal tissue
- Avoid "shelling out" even if additional tissue is then taken
- Excise "en bloc"
- Pseudocapsule not appropriate margin
- Minimum of 1cm but varies with tumour
  - E.g oral FSA versus SCC
  - No studies to evaluate size of margins with different tumour types except mast cell tumours

Dick White Referrals

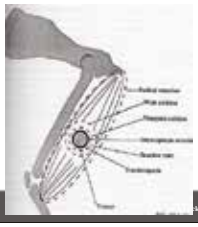


Surgical margins

- Intracapsular resection
  - Dissection within tumour capsule
  - Benign conditions
- Marginal resection
  - Through reactive zone
  - Tumour + pseudocapsule
- Wide resection
  - Margin of normal tissue
- Radical resection
  - En bloc removal of tumour and tissue compartment

Dick White Referrals

Surgical margins



Dick White Referrals

Vector borne diseases

Simone Manzocchi (IT)

DVM, res EVPC.

Dr. Manzocchi received his veterinary degree from the University of Parma Veterinary School in Parma, Italy in 2010 and started working in private practice the same year. Until 2012 he worked in several practices and veterinary hospitals focusing his activities on clinical pathology and clinical parasitology. In 2012 he became an employee of IDEXX Laboratories in Novara Day Lab, where he is currently the head of clinical biochemistry and responsible of the parasitology diagnostic service. In 2015 he was accepted as a Resident of the European College of Veterinary Parasitology (EVPC) and established active collaborations with several Universities on parasitology research projects. Dr. Manzocchi has authored peer-reviewed publications and a diagnostic guide and has been a speaker at national and international seminars.

How to carry out a cheap and perfect Knott and Baermann testing in your practice

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Infection of dogs and cats with *Dirofilaria immitis* and *Dirofilaria repens* has been diagnosed in most European countries, particularly in southern and eastern Europe, and are spreading because of several climate and social changes influencing both populations of mosquito vectors and mammalian reservoirs.[1] The exact same thing is happening for *Angiostrongylus vasorum* and for many other vector borne diseases worldwide.[2]

Diagnosis of heartworm disease in dogs is based on the detection of microfilariae in association with the detection of circulating heartworm antigens, while canine angiostrongylosis can be diagnosed by identification of 1st stage larvae in the feces or detection of specific antigen in the blood. [3,4] Detection and identification of microfilariae in the blood for filarial diseases and of nematode larvae in the feces for lungworm diseases can be performed both by specialized laboratory or by clinical practices using cheap instruments and good technical skills.[5]

For detection of microfilariae in blood, without regard to the specie we are looking for, two diagnostic techniques are considered the most sensitive: Modified Knott Test (MKT) and Filter Test.

The modified Knott's technique is the easiest and cheapest method for concentration and identification of microfilariae in blood and requires few and simple steps:

1. Draw a sample of blood into a syringe containing anticoagulant such as EDTA or heparin. Mix well the sample before use.
2. Mix 1mL of the blood with 9mL of a 2% formalin solution. If not well mixed, the red cells will not be thoroughly lysed by the hypotonic formalin solution, making the test much more difficult to read. Microfilariae, but not red cells, will be fixed by 2% formalin. If 10% formalin is used (the concentration used for fixation of tissues), red cells will also be fixed and not lysed.
3. Let it sediment for 20' or centrifuge the mixture at low RPM and discard the supernatant.
4. Add one drop of eluted methylene blue or cresyl blue to the sediment, mix well, and transfer the stained sediment to a microscope slide using a Pasteur pipette to collect the entire sample.
5. Examine using the 10× microscope objective. Microfilariae will be fixed in an extended position with nuclei stained blue.

An alternative procedure using the same amount (1ml) of blood is the filter test, which traps microfilariae on a micrometric filter that is then examined by microscopy. This technique can be quicker and more sensitive (more microfilariae detected) than MKT, but microfilariae are not so easily measurable and morphologically evaluable as in MKT.

1. Mix 1mL of blood with 9mL lysing solution in a syringe.
2. Attach the syringe to the filter holder containing a filter with a 5-µm pore size and empty the syringe.
3. Refill syringe with water and pass it through the filter to wash away remaining small debris.
4. Refill syringe with air, reattach to the filter apparatus, and express.
5. Unscrew the filter assembly, remove the filter with forceps, and place the filter on a microscope slide.
6. Add one drop of eluted methylene blue or cresyl blue, coverslip, and examine at 10×.

For both the techniques the use of 2% formalin solution is mandatory for measurements consistency. The solution may be made from 10% (or more) solutions using tap or distilled water as eluent.

Other techniques can be applied for detection of microfilariae, as wet mount blood smear, air dried and stained blood smear, hematocrit test, buffy coat examination, but those methods all achieve lower sensitivity than MKT/Filter test because they are performed on smaller amount of blood.

The Baermann test is the backbone of lungworm diagnosis. It is used to efficiently isolate larvae from fecal samples and concentrate them on a slide for microscopic evaluation and identification. Some expedients are required to maximize diagnostic performance of Baermann test. First of all, it is very important that the fecal sample is fresh, since parasitic eggs (not lungworms) may hatch and free-living nematodes may invade the sample, making nematode identification much more difficult. The volume of the fecal sample is of paramount importance to achieve good result; it should be at least 10 gr (a full spoon). Collecting multiple samples form 3 consecutive days can increase the chance of detecting larvae in case of intermittent shredding. Using warm water and a bright room is reported to increase larval migration. 24 hours in considered the correct period for having all the larvae migrated in the apparatus. Finally, the ratio between fecal volume and water should be as low as possible to decrease dispersion of larvae.

A Baermann test requires equipment to hold the fecal sample in water so that larvae can migrate out and be collected. This can be easily accomplished in veterinary practice with the use of a wine glass with a hollow stem, plastic bags, or similar equipment that can contain enough volume of samples and can be easily poured off. Additionally, the original Baermann apparatus consisting of a funnel clamped to a metal stand with a short piece of tubing with a clamp, can be easily created using home crafting items. Larvae in feces placed in the warm water migrate out of the sample and fall down into the bottom, where they can be easily collected.

1. Place at least 10 g of feces in a piece of double-layer cheesecloth. Gather the cheesecloth around the sample so that it is fully enclosed. Use a rubber band or wire to fasten the cheesecloth, and pass through the cheesecloth an object that will rest on the edges of the glass or funnel and suspend the sample (like a pencil). Alternatively, place the sample on a suspended sieve.
2. Fill the funnel or wine glass with warm water. Make sure that the corners of the cheesecloth do not hang over the edge of the funnel or glass, because they will act as wicks for the water.
3. Allow the sample to sit for at least 12 hours, preferably 24.
4. Remove the fecal sample and collect 10 ml of the material at the bottom using a Pasteur pipette or release the clamp of the funnel and collect 10 ml. Let it sediment or spin it at low RPM, discard the supernatant and examine microscopically the while pellet (0,5 ml ca). A stereomicroscope or a compound microscope with 5-10 x objective can be used.

The strong movement of the 1st stage nematode larvae will help the diagnosis.

Once you have found microfilariae in the blood or nematode larvae in the faces, a typization should be performed to achieve a specific diagnosis. This can be done by microscopy, evaluating morphometric features, or more easily by PCR.

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Is it *D. immitis*, *D. repens* or something else? Is it *Angiostrongylus*? What about antigen testing? Tips and tricks for getting out of the jungle of blood and faeces

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Infection of dogs and cats with *Dirofilaria immitis* and *Dirofilaria repens* has been diagnosed in most European countries, particularly in southern and eastern Europe.[1] Those infections are spreading because of several climate and social changes influencing both populations of mosquito vectors and mammalian reservoirs.[1] The exact same thing is happening for *Angiostrongylus vasorum* as for many other vector borne diseases worldwide.[2]

Diagnosis of heartworm disease in dogs is based on the detection of microfilariae, in association with the detection of circulating heartworm antigens. Canine angiostrongylosis can be diagnosed by identification of 1st stage larvae in the feces or by detection of specific antigen in the blood. [3,4] While concentration and detection of microfilariae and 1st stage larvae can be easily performed following strict technical procedures (Modified Knott Test, Baermann Test),[5] accurate identification of the nematode specie is cumbersome and requires some degree of expertise. The length and some morphological features must be evaluated, both in MKT and Baermann test, to achieve a specific diagnosis.[5] Specifically, morphology of the head, cephalic space and tail can be considered for differentiation of microfilariae in blood, while morphology of tail, esophagus and tip of the head can be used for 1st larvae in feces. Measurement can be done traditionally by a micrometric grid in the eyepiece of the microscope. Nevertheless measuring on digital images took from microscope is generally a quicker and more precise method, considering that the structures to measure are often not linear (polylines measurements).

Specie	Length (µm)	Head	Tail	Cephalic Space
D. immitis	~300	tapered	more straight	long
D. repens	~350	blunt	more hooked	short
Acantocheilonema spp	~250	knob-like	variable	very long

Table 1. Microscopic features of different species of microfilariae in Modified Knott Test.

Specie	Length (µm)	Esophagus	Head	Tail
Angiostrongylus vasorum	310-400	Non rhabditiform	Cephalic button, terminal apaerture	Kinked, dorsal indentation with spine, secondary ventral indentation
Oslerus/Filaroides	~250	Non rhabditiform		S-shape-kinked, single indentation, no spine
Crenosoma vulpis	240-310	Non rhabditiform	Pointed, subterminal aperture	pointed and straight
Strongyloides stercoralis	150-390	Rhabditiform	short buccal tube	pointed and straight
Ancylosoma caninum	~325	Rhabditiform	long buccal tube	pointed and straight

Table 2. Microscopic features of different species of 1st stage nematode larvae in Baermann Test.

In case of difficult or uncertain identification, a biomolecular approach (PCR) can be used. For best results is advised to forward sample with high density of parasitic elements, as microfilaremic blood, sediment from positive Knott Test, filter from positive Filter Test, sediment from positive Baermann Test.

Once you have achieved a specific diagnosis or a negative result for microfilariae or 1st stage larvae, the results should be correlated and interpreted in association with antigen tests. The paired use of microscopic and antigen test maximizes the diagnostic performance, increasing both sensitivity and specificity of the overall diagnostic outcome.[6] Generally, a positive result on both tests confirm infection. Negative concordant results suggests an uninfected animal or that both the tests were performed before patency and antigen development (less than 6 months for heartworm disease, less than 4 weeks for angiostrongilosis). In case of discordant results an accurate interpretation is mandatory. Careful evaluation of possible false positivity and false negativity of both tests should be performed together with clinical examination, thoracic radiography and echocardiography.[6]

Antigen	MKT	Possible interpretations
+	-	• Occult infection (common) • Antigenic cross reactivity (rare, with *Angiostrongylus*)
-	+	• Low burden or single sex infection • Immunocomplexes (antigen entrapment)

Table 3. Possible interpretations of discrepancy between test for heartworm disease.

Antigen	Baermann	Possible interpretations
+	-	• Intermittent excretion
-	+	• Low antigenic density in feces

Table 3. Possible interpretations of discrepancy between test for angiostrongylosis.

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He is the author and co-author of more than thirty original articles on parasitic diseases and cardiology in International peer reviewed Journal and author of several chapters in books on the same topic.

It presented more than 100 papers at conferences in Italy, Spain, UK, Russia, Croatia, Serbia, Bulgaria, Hungary, Romania, Slovenia, Czech Republic, Poland, USA, Japan and Cuba.

He is an European Veterinary Parasitology College Diplomate, President of the European Society of Dirofilariosis and Angiostrongylosis, Honorary member of the Bulgarian Veterinary Cardiology Society and Referee of peer reviewed Journals of Parasitology both veterinary and human.

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What can I flush out from echocardiography and thoracic radiographs in heartworm infected dogs?

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Despite the name "Heartworm" suggests a primitive cardiac involvement as the main localization of the worms and the first damages are in pulmonary arteries , heartworm disease should be considered a pulmonary disease that in the last stage only may involve right cardiac chambers.

In Heartworm infected dogs the reduction of compliance and gauge of pulmonary arteries, that can be also occluded by either thromboembolism or severe villous proliferation, results in a hypertensive pulmonary state and , as a consequence , in an increased after load for the right ventricle which can induce "cor pulmonare" and right cardiac congestive heart failure[1,2].

Based on the pathogenesis the clinical evolution of heartworm disease in dogs is usually chronic.

Most infected dogs do not shows any symptoms of the disease for a long time, months or years, depending on worm burden , individual reactivity and exercise , as arterial damages are more severe in dogs with intensive exercise than in dogs at rest. Signs of the disease develop gradually and may begin with a chronic cough.

Together with antigen with history, clinical examination and antigen testing thoracic radiographs and echocardiography are pivotal for staging the disease and choosing the best therapy in infected dogs.

Thoracic radiographs

Thoracic radiographs may show, in the advanced stage, enlargement of the pulmonary arteries , abnormal pulmonary patterns and in the worst cases right sided cardiomegaly If congestive right heart failure is present peritoneal and pleural effusion can be noted They are useful to asses the severity of the pulmonary lesions but not for evaluating worm burden[3]. Since radiographic signs of advanced pulmonary vascular disease may persist long after an infection has run its course, some of the most severely diseased dogs may have disproportionately low worm burden. On the contrary some inactive dogs may have large worm burdens and be clinically asymptomatic with no or trivial radiographic lesions.

Echocardiography

Echocardiography allows a direct visualization of cardiac chambers and connected vessels[4].

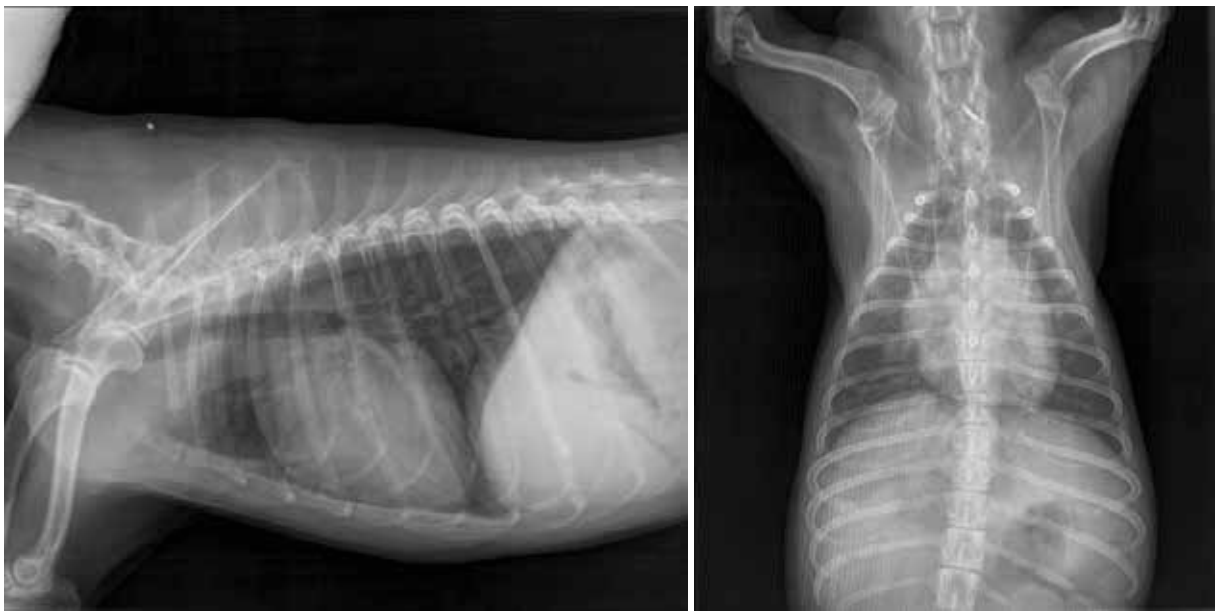
It also allows the visualization of parasites in right cardiac chambers, caudal vena cava, main pulmonary artery and proximal tract of both caudal pulmonary arteries. The heartworms are visualized as double, linear parallel objects floating in the right cardiac chambers or into the lumen of vessels[4,5].

It is performed mainly in cases where clinical and radiographic findings suggest severe disease.

Cardiac ultrasound can increase the accuracy in staging the disease and estimating the worm burden, both of which affect the treatment program and the prognosis[3].

Doppler echocardiography by measuring the velocity of tricuspidal and or pulmonic valve regurgitation furthermore allows a not invasive correct assessment of pulmonary pressure. When regurgitation absent Right Pulmonary Distensibility Index (Right pulmonary artery Diameter in Systole less Right pulmonary artery Diameter in Systole/ Right pulmonary artery Diameter in Systole) is useful for pulmonary artery pressure estimation in heartworm infected Dogs.

In conclusion both Thoracic radiographs and Echocardiography provide useful information about the Heartworm infected dogs and both of them should be used for staging the disease



Figs 1a and 1 b – Thoracic radiographs (right lateral and dorsoventral view) of a 4 year old cross breed Heartworm infected dogs. Not obvious radiographic changing but mild enlargement of caudal pulmonary arteries in dorsoventral view.

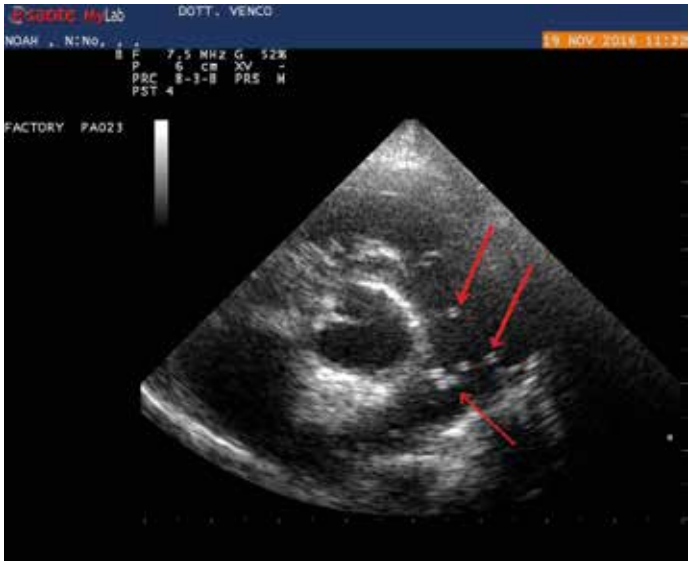


Fig 2 – Ecocadiography (Right Parasternal Short Axis view) of the same dogs. Several echoes (arrows) coming from adult worm into the main pulmonary artery

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In the middle of a boring day the canine *Angiostrongylus* patient: a difficult diagnosis for a simple therapy

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Angiostrongylus vasorum also known as "French worm" is a Nematode that is located at the adult stage in the lungs of dogs and other canides such as foxes (the main reservoir) and wolves. In Europe, this parasite has recently become a priority for veterinarians because of a number of reasons[1,2]. First, *A. vasorum* is spreading by increasing its prevalence in regions already known as endemics and reaches areas previously exempt. Secondly, Angiostrongilosis has a serious and significant impact on the health of infested dogs. Thirdly, although therapy is often simple and successful, proper diagnosis can be problematic or inadequate.

Biology

Angiostrongylus vasorum has an indirect life cycle that is based on land gasteropods (slugs and snails) as intermediate hosts. Some animals, such as amphibians and birds, can act as paratenic hsots. The *A. vasorum* first generation larvae (L1) reach the environment through the feces of infested dogs and penetrate into the foot of the competent gastropods, reaching the third stage (L3), the infectious stage for vertebrates at risk. Dogs are infected by swallowing the intermediate host for direct predation or for accidental swallowing during digging or while eating grass, or very seldom eating a paratenic host. After ingestion, the L3 penetrate the intestinal mucosa, migrate to the mesenteric lymph nodes, where they molt twice to the pre-adult stage, then reach the right ventricle and pulmonary arteries through the lymphatic vessels. Worms reach the adult stage into the pulmonary arteries. The eggs laid by adult *A. vasorum* females disclose the L1 larvae that penetrate into bronchioles and alveoli through the lung capillaries, thus exploiting the ciliary mucus clearance and following coughing are ingested and then released with faeces in the environment. The prepatent period ranges from 1 to 4 months, with an average of 1-2 months.

Geographical distribution

Angiostrongylus vasorum has long been considered to have a leopard stain distribution exclusively in certain endemic European regions such as southwestern France, south of the UK, Denmark and Switzerland, with occasional and sporadic reports outside these areas. Over the last decade, there has been an increase in the number of cases documented in the classically endemic territories, and at the same time, new infection outbreaks have appeared in several previously endemic countries and areas. For example, in the United Kingdom, *A. vasorum* is spread throughout the country, and increased reports of individual clinical cases or significant prevalence rates have been reported in the North (Denmark) and Central Europe (Poland, Germany) and in the Mediterranean area (Greece, Italy) [1,2].

In addition, the first indigenous cases of Angiostrongilosis in eastern countries (Slovakia and Serbia) have been described. Other European countries where *A. vasorum* has been recently found or is endemic with variable rates are Belgium, Hungary, Finland, the Netherlands and Spain[1]. Veterinarians should always remember that the supposed absence of *A. vasorum* in a given geographical region does not imply that the parasite is really absent. Therefore, the geographical area should not be considered as a criterion for including or excluding *A. vasorum* in dogs with compatible clinical signs.

Clinical signs

All dogs of all ages and breeds are potentially at risk of infection throughout the whole year. However, younger dogs (under 2 years) are at higher risk, probably due to the behavior that leads them to swallow more often the intermediate host. Even some breeds (Bull Terrier, American Staffordshire, Labrador, Beagle, Cavalier King Charles, Jack russel) are most likely reported to be infected for the same reason such as hunting dogs and truffles searching dog for lifestyle. Clinical forms can occur throughout the whole year but winter and spring are the seasons in which they are more frequently observed due to the length of the prepatent period. Clinical manifestations of angiostrongilosis in the dog are usually acute, but many subjects may live with infestation without apparent symptoms for a long time, and in some cases suddenly present serious clinical forms[3]. The most common clinical scenario is characterized by respiratory symptoms: cough, dyspnea, tachypnea, pulmonary hypertension (Cor pulmonale), tachycardia, pale mucous membranes and syncope. Pulmonary hypertension is less commonly observed and usually disappears after treatment but significant increases in pulmonary pressure values are possible and associated with poor prognosis. *A. vasorum* often causes coagulation) disorders. Affected dogs may have petecchie or ecchymosis, but also spontaneous blleding form the nose, hematomas, post-surgical haematomas, anemia and / or chronic thrombocytopenia[3,4]. Potentially fatal hemorrhages may occur in the central nervous system[5].

Diagnosis

Canine angiostrongilosis should always be included in differential diagnosis in dogs that also have a single compatible clinical sign, regardless of whether the parasite is endemic or not in a given area, or if it is more likely that pathologies more frequently diagnosed in the past (taking It is noteworthy that a large part of the dogs as in most parasitic diseases can be asymptomatic or with subclinical symptoms.) A definitive diagnosis can only be obtained by detecting *A. vasorum* larvae (L1) in the feces and/or circulating antigen of the parasite in serum of the infected dogs[6].

Clinical examination, thoracic radiographs echocardiographic findings can lead to a strong suspicion. The same applies to hematologic or biochemical results. Specifically at the radiograph level, the most commonly detected pattern is a focal interstitial pulmonary with centripetal distribution sometimes associated with increased size of the major pulmonary artery[3,4].

Treatment and control

The treatment is based on the use of benzimidazoles (Fenbendazole) and some macrocyclic lactones (Moxidectin, Milbemycin oxime). Fenbendazole oral at 25-50 mg / kg for 5-21 consecutive days is an effective and well-received treatment, although in many countries this molecule has no claim for the treatment of A. vasorum[3,4]. At present, in Europe, medicines authorized for the treatment of angiogenesis in the dog are two: the milbemycin / praziquantel oral combination and the imidacloprid / moxidectin in spot formulation. Although it is evident that activity against parasites is carried out by macrocyclic lattons in these associations. Symptomatic therapy that may be associated varies in relation to each clinical scenario. In dogs with severe dyspnoea, oxygen supplementation, corticosteroids and bronchodilators may reduce the severity of the symptoms. Corticosteroids at immunosuppressive doses may also be useful for the treatment of secondary immune mediated thrombocytopenia. Fresh whole blood transfusion or frozen plasma is indicated in dogs with hemorrhagic diathesis while inhibitors of type 5 phosphodiesterase (sildenafil) in case of severe pulmonary hypertension. The routine use of heparin therapy in case of DIC secondary to infestation due to the underlying inflammatory state is questionable and not recommended. Dogs at risk of infection, especially those living in the same environment in which they have been reported, even earlier, individual clinical cases should undergo chemoprophylaxis. Again, monthly administration of Moxidectin in spot formulation and Milbemycin oxime in oral formulation are considered effective. The use of specific poisons against slugs and snail for environmental prophylaxis must be avoided because it does not guarantee a true reduction of the gastropod populations, being at the same time dangerous for the environment and for dogs, and leading also paradoxically to a more frequent access of dogs to the dead snails[1,2].

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Notes