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Editor's Note

Just this last month I had to advise a client with a household of cats of which one tested positive for FeLV. She had consulted two general practitioners and both gave similar advice, but with one or two vital differences regarding interpretation of the tests. In multi-cat households managing viral infections can be a nightmare and the immunology of test interpretation can be daunting, and as usual, the devil is in the detail. There is a fantastic free access website - The European Advisory Board on Cat Diseases (ABCD) (http://www.abcdcatsvets.org) which has excellent factsheets on various aspects of feline infectious diseases and husbandry. I would urge vets with a feline patient population to peruse these guidelines.

Urinary incontinence in the bitch will be with us for as long as we continue spaying them. As vets we contend that the benefits of sterilisation outweigh the risk of incontinence, however if you had a 30 kg incontinent Doberman which is not responding to medical management and is peeing all over your carpets your opinion might be different. Medical options are available and if a poor response is demonstrated, surgical options are also available, and possibly under-utilised. The techniques sound simple, but placement and tension of the sutures is critical. Specialist surgeons or those experienced in the technique should be the first port of call.

The other day I was evaluating a radiograph of a patient with a healed radius fracture which had been repaired with a plate and screws. The dog was showing lameness and I identified lysis around the screws... only to be told that it was a digital "software" artefact. This prompted me to request an article of digital radiographic artefacts for this this edition. And no ... you can't just adjust the image obtained to correct for poor exposure technique....

Thank you to Drs Mirinda van Schoor and Christelle le Roux and Prof Louis Coetzee for assisting with articles for this edition. I hope you enjoy the edition.

Liesel

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Living With FeLV-infected Cats:
A Guide for Veterinarians and Their Clients

With proper management by the owner and healthcare from the veterinary team, cats with this retrovirus can live longer, more comfortable lives.

By Glenn Olah, DVM, PhD, DABVP (feline)

Feline leukemia virus (FeLV) is an RNA gamma-retrovirus of cats found worldwide, infecting anywhere from 3% to 14% of domestic cats depending on geographic location, sex, lifestyle and general health[1]. Experts speculate that the virus evolved from rats during the late Pleistocene era up to 10 million years ago in the North African desert. Ancestral rats and cats roamed freely, and the virus was likely transmitted to cats through rat ingestion or bite.[2,3].FeLV is highly contagious, particularly in kittens, and is readily spread among cats in casual close contact, which can include sharing food and water as well as mutual grooming. However, aggression (i.e. cat-fight bites) can also readily transmit the virus. There are three primary outcome stages of FeLV infection: abortive, regressive and progressive.[4] Approximately two-thirds of cats exposed to FeLV will experience either the abortive or regressive stage of infection, and about one-third of cats develop progressive infection[5]. Here are some additional details:

Abortive stage
An abortive infection occurs when a cat clears the infection. (Cats with this stage were formerly referred to as "regressor" cats.)

Regressive stage
While a regressive infection causes a cat to become temporarily viraemic, the cat eventually clears the viraemia and does not become ill from FeLV-associated diseases. However, it does have viral DNA integrated into its genome.[6-8](Cats with regressive infection were formerly referred to as "latently infected" cats.)

Progressive stage
Progressively infected cats shed virus in their saliva, ocular and nasal secretions, urine, faeces and milk and are thus infectious to other cats. (These cats were formerly called "persistently viremic" cats.) Progressively infected cats can survive months to years, with a mean survival of 3.1 years, and may die of FeLV-associated diseases[9]. However, with proper

management and veterinary care, an FeLV-infected indoor-only cat may live much longer with a good quality of life. Focal infection may occasionally occur; it is characterized by persistent atypical local viral replication (such as in mammary glands, the bladder or the eyes)[4,10].

FeLV-associated diseases include lymphoma, leukaemia, anaemia and infectious diseases that are potentiated by the virus' immunosuppressive effects. Outcomes of FeLV infection depend on an individual cat's immune status, genetic makeup and age, the presence of any other infectious diseases, and the pathogenicity and infectious dose of the FeLV virus.

Determine FeLV status of all cats in a household

If a cat tests positive on a screening test for FeLV, it should be confirmed as "true positive" with a confirmatory test; both tests are typically performed on peripheral blood. Screening tests are usually ELISA-based tests designed to detect p27 FeLV **antigen**, and most cats will test positive within 30 days of exposure[11]. Recommended confirmatory tests are either indirect fluorescent antibody (IFA) tests that detect p27 FeLV antigen in infected leukocytes or platelets, or polymerase chain reaction (PCR)-based tests that detect FeLV provirus. IFA tests don't usually yield positive results until secondary viraemia has occurred after infection of bone marrow (about 45 to 60 days after initial infection).

The stage (abortive, regressive or progressive infection) should be determined for all FeLV-infected cats. Abortive infected cats will test FeLV-negative and IFA- or PCR-negative, but they will seroconvert and test FeLV antibody-positive; however, antibody testing isn't usually performed in a clinical setting. Regressively infected cats usually test FeLV antigen-negative no later than 16 weeks after infection, while progressively infected cats remain FeLV antigen-positive[12]. Both regressively and progressively infected cats can test PCR FeLV provirus-positive as soon as two weeks after infection and they will remain positive thereafter[13,14].

Here are some additional principles for FeLV testing

- Any new cats or kittens should be screened for FeLV infection before being introduced into a household.
- Household cats that go outdoors or share a house with cats that go outdoors should be FeLV-tested at least yearly. Also, any cat that becomes clinically ill should be tested for FeLV immediately if it shares a household with an FeLV-infected cat.
- Household cats that may have been exposed to other cats with unknown FeLV infection status should be immediately tested for FeLV and retested six weeks after exposure. In some cats it can take up to four months to figure out the stage of FeLV infection. In a multicat household, it can be difficult for the owner to confine FeLV-exposed cats, assess risk to other cats, and decide how to manage the situation. Close partnership with the veterinary team is essential in these situations.
- FeLV tests detect infection, not clinical disease. A decision for euthanasia should never be based solely on whether a cat is "confirmed" FeLV-infected. While FeLV infection can be life-threatening, proper management and prompt veterinary care can help regressively and even progressively infected cats have long, healthy lives with good quality.
- Which cats should be vaccinated for FeLV?
  The decision to vaccinate an individual cat against FeLV is based on risk assessment for infection and lifestyle. Cats that should be vaccinated include:

  1. Kittens, because they're more susceptible to infection and their lifestyle is still in flux. Note that although FeLV infection susceptibility decreases as cats get older, the risk does not necessarily reach zero; it depends highly on a cat's lifestyle and degree of viral exposure.
  2. Cats with access to the outdoors and cats that have contact with cats with access to outdoors.
  3. Cats that live with FeLV-infected cats.
  4. Cats that may encounter other cats with unknown FeLV status.

Managing healthy FeLV-positive cats

If a cat is FeLV-positive but displaying no clinical signs, it should receive a physical examination at least twice a year and at each veterinary visit, with attention paid to unintentional weight loss, enlarged lymph nodes, clinical signs of upper respiratory infection (e.g. ocular or nasal discharge) and oral health. All cats should have the anterior and posterior segments of the eye thoroughly examined. Complete blood count, biochemical profile, urinalysis, urine culture and fecal examination are indicated at least once a year. Some fragile FeLV cats may need bloodwork, urinalysis and faecal examination more frequently. Infected queens and toms should not be bred, and they should be spayed or neutered, respectively, to reduce behaviors that increase risk of disease exposure or transmission, such as escaping, fighting and roaming. Routine gastrointestinal and external parasite controls should be provided.

Some FeLV-infected cats have been shown not to mount an adequate protective response to rabies vaccination[15];therefore, it's prudent to advise owners that FeLV-infected cats should not have outdoor access, especially in rabies-endemic areas.

Regardless, FeLV-infected cats should still be vaccinated with core vaccines (rabies, feline herpesvirus,

calicivirus and panleukopenia virus) and possibly vaccinated more frequently (for example, every six months) based on an individual cat's risk assessment and lifestyle[4]. There is controversy surrounding the use of inactivated, modified-live or recombinant vaccines. Some researchers and clinicians suspect an increased risk for the development of injection-site sarcomas with the use of adjuvant killed vaccines[16], and others are concerned that modified-live vaccine viruses may regain their pathogenicity in immunocompromised cats1[7,18].

Managing clinically ill FeLV-positive cats

Early therapeutic intervention is key to a successful treatment outcome in FeLV-infected cats that display clinical signs. First, the clinician should determine whether the illness is directly associated with FeLV infection (for example, lymphoma or anemia) or a secondary disease associated with immune dysfunction (opportunistic infection or oral inflammatory disease). Intensive diagnostic testing should occur earlier during a diagnostic workup as opposed to a "wait-and-see" approach. Most FeLV-infected cats respond well to appropriate medications and treatment strategies, but they may require a longer or more aggressive course of treatment and need to be more closely monitored during recovery. While several antiviral drugs, immunomodulators and alternative therapies have been investigated for efficacy in FeLV treatment, most have been shown to be ineffective or only marginally beneficial[19,20]. To date, no treatment has been shown to reverse or cure FeLV infection in cats.

Educating owners of FeLV-infected cats

Initial diagnosis may illicit quite a bit of anxiety in an owner. To alleviate this anxiety, it's helpful to educate the owner about FeLV infection aetiology, its clinical effects, and how proper home management and veterinary care can provide the best health and quality of life for the cat. Remember the old adage that people often fear what they don't understand. Be sure to empower owners regarding management of the cat at home and have them view the veterinary clinic as a source for medical and management advice. In addition, alert owners to the 10 common feline signs of illness[21]:

1. Inappropriate elimination (urination, defaecation or both)
2. Changes in social interaction
3. Changes in activity level
4. Changes in sleeping habits
5. Changes in food and water consumption, changes in chewing and eating habits
6. Unexpected weight loss or gain
7. Malodorous breath
8. Changes in sleeping habits
9. Changes in vocalisation
10. Signs of stress (hiding, withdrawal, changes in appetite, decreased grooming, decreased social interaction, more awake time and so on).

If owners observe any of these signs, or if they notice other behavioural changes and aren't sure if they're important, they should contact the veterinary clinic for advice.

The best situation for an FeLV-infected cat is to live in an indoor-only environment and be the only cat in the household[10]. A nutritionally balanced diet is also essential. Cats are obligate carnivores and evolved from a desert environment; thus they thrive on high-quality (animal-based) protein (more than 45% by dry matter), low-carbohydrate, moderately low-fat and high-moisture diets[22].

Canned cat foods are ideal because they have high water content. It's possible to transition cats that prefer dry food to a canned food diet, but this should be done cautiously. Remember that many cats would rather starve to death than eat unfamiliar foods or foods they don't like. It's better to have a cat eat than not eat, so if dry foods must be fed, then research dry foods with a good nutrient profile. Raw diets should be avoided in FeLV-infected cats because of the increased risk of foodborne bacterial and parasitic diseases.

Although it's preferable for FeLV-infected cats to live in single-cat households, thereby avoiding viral transmission to cat housemates and preventing high-risk behavior such as cat fights, this isn't always possible. If they're to be part of a multicat household, then separation of FeLV-infected cats is ideal. If an owner is unwilling to separate the FeLV-infected cat from non-infected cats, then the non-infected cats should be adequately FeLV-vaccinated. Warn owners that vaccination does not guarantee 100% protection, especially in high-exposure environments.

No new cats should be added to the household because this would disrupt the social structure and possibly increase the risk of cat fights and bites. Since FeLV is primarily transmitted by close contact (both friendly and aggressive) and the sharing of food bowls, water bowls and litterboxes, it's unlikely that an owner will create an environment completely void of FeLV infectious virions. However, providing separate feeding stations for infected and non-infected cats may help decrease the degree of exposure.

FeLV is also labile outside of the host, remains infectious for only minutes in the environment and is readily inactivated with soap and disinfectants, so frequent cleaning of litterboxes and other potential fomites with soap and disinfectant may decrease viral load. FeLV is not zoonotic.

Recommendations in this article are based on 2008 American Association of Feline Practitioners (AAFP) Feline Retrovirus Guidelines but also include some updated material and perspectives[10]. The AAFP Feline Retrovirus Guidelines are presently in the process of being updated[23].

KEY points for FeLV infection

Terminology has changed: Persistent viraemia = progressive infection; Transient viraemia = regressive infection; Cleared virus = Abortive infection

Infections occur worldwide, but the prevalence and importance of FeLV in developed nations has decreased due to reliable tests, programmes to segregate viraemic carriers, improved understanding of FeLV pathogenesis and the introduction of effective vaccines.

The most widely used in-practice tests for FeLV are antigen ELISA and immunochromatography tests. As the prevalence of FeLV infection decreases the percentage of false positive test results tends to increase. Therefore, a positive result in a healthy cat should always be confirmed, preferably using provirus PCR offered by a reliable laboratory or by repeating up to 16 weeks after the first positive result.

A positive ELISA or immunochromatography result in a cat with clinical signs consistent with FeLV infection is more reliable, as in sick cats the prevalence of FeLV is considerably higher, and the higher the prevalence the higher the probability of a test to be correctly positive. Cats in which an antigen-positive test result has been confirmed can overcome viraemia (regressive infection) after weeks, usually by twelve weeks – in rare cases even later; in one documented case more than a year after infection.

Every antigen-test-positive healthy cat should be separated and retested after 6 or more weeks; if the cat still tests antigen-positive retesting can be repeated. Cats that clear infectious virus from the plasma will be negative by virus isolation, ELISA, immunochromatography and IFA, but will remain positive by provirus PCR. These cats should be considered regressively infected.

Often the development of viraemia as well as the establishment of a progressive infection may be overcome by a functioning immune system, resulting in transient viraemia. Such regressively infected cats are generally not at risk of developing disease, even with corticosteroid treatment in the future. In a multi-cat household without control of FeLV infection, 30-40 % of the cats develop persistent viraemia (progressive infection), 30-40 % exhibit transient viraemia (regressive infection) and 20-30 % develop antibodies without ever being detectably viraemic (abortive infection). A smaller proportion (~5 %) exhibits an atypical course of infection, displaying antigenaemia but no or only low-level viraemia.

FeLV does not survive long outside the host as it is easily destroyed by disinfectants, soap, heating and drying. Transmission via fomites is unlikely. The virus will however survive if it is kept moist at room temperature, so there is a potential for iatrogenic transmission via contaminated needles, surgical instruments or blood transfusions.

Viraemic cats are the source of infection, with shedding in saliva, nasal secretions, faeces, and milk. Risk factors are young age, high population density and poor hygiene. Transmission occurs mainly through friendly contacts like grooming. In viraemic queens, pregnancy usually results in embryonic death, stillbirth or in FeLV-viraemic kittens, which commonly fade away rapidly. In regressively infected queens, usually transmission does not take place during pregnancy. However, rarely some (but not all) kittens of these queens may become viraemic after birth, with transmission occurring from individual mammary glands.

Young kittens are especially susceptible to FeLV infection. As cats age they become increasingly resistant, especially to progressive infection, and this is in full force by 3 years of age. The cat's age at the time of the infection is the most important factor determining the clinical progress. Viral and host factors, like the virus subgroup and the cell-mediated immune response, influence the pathogenesis in individual cats.

Cats with regressive infection appear to have the same life expectancy as cats that have never been exposed to FeLV.
The prognosis for cats with progressive FeLV infection is poor and most will develop typical clinical signs. Whether clinical signs are present or not, every cat with progressive FeLV infection is immune-suppressed with delayed and decreased primary and secondary antibody responses.

FeLV-infected cats may develop many different types of anaemia; most of them are non-regenerative and only few are regenerative. Regenerative anaemias, associated with haemolysis may be related to secondary opportunistic infections, for example by *Mycoplasma haemofelis*, or to immune-mediated destruction of red blood cells. Non-regenerative anaemias may be caused by the direct bone marrow suppressive effects of the virus, chronic inflammatory mechanisms, or myeloproliferative disease. Other cytopenias may be present, e.g., thrombocytopenia and neutropenia, caused by the same mechanisms. Many complications of FeLV infection respond well to treatment, such as secondary bacterial infections, especially with *Mycoplasma haemofelis*, which often improves with antibiotic treatment, such as doxycycline

In 2 recent studies FeLV antigen-negative cats with lymphoma had significantly longer remission times (472 ; 170 days)) than FeLV antigen-positive cats (25 days, 27 days) following treatment. The prognosis of lymphoma in cats with progressive FeLV infection is poor because of bone marrow suppression, which is usually exacerbated by chemotherapy and can frequently delay treatment.

Although progressively infected cats can be housed in the same ward as other hospitalised patients, they should be kept in individual cages. Simple precautions and routine cleaning procedures will prevent transmission in the hospital. Since they may be immune-suppressed, they should be kept separated from cats with other infectious diseases. They should not be placed in a "contagious ward" with cats suffering from e.g. viral respiratory disease.

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1. Which one of the statements listed does NOT apply to the regressive (latent) infection of FeLV?
a. occurs when a cat clears the infection.
b. causes a cat to become temporarily viraemic,
c. becomes ill from FeLV-associated diseases
d. it does have viral DNA integrated into its genome
e. shed virus in their saliva, ocular and nasal secretions, urine, faeces and milk and are thus infectious to other cats

2. Which one of the statements listed does NOT apply to the progressive stage (persistently viraemic) stage of FELV?
a. Progressively infected cats can survive months to years
b. Progressively infected cats shed virus in their saliva, ocular and nasal secretions, urine, faeces and milk
c. Are infectious to other cats.
d. Approximately two-thirds of cats exposed to FeLV will experience the progressive stage of infection
e. May die of FeLV-associated diseases

3. Which one of the disease listed below is NOT typical of cats infected with FeLV?
a. Lymphoma,
b. Leukaemia,
c. Anaemia
d. Infectious diseases
e. Neutropenia

4. Which one of the statements below is CORRECT concerning confirmatory testing for FeLV?;
a. A bone marrow sample is required for a confirmatory test for integrated viral DNA
b. Recommended confirmatory tests are either indirect fluorescent antibody (IFA) tests that pro-virus in infected leukocytes or platelets
c. Screening tests are usually ELISA-based tests and most cats will test positive within 2 weeks of exposure
d. Confirmatory polymerase chain reaction (PCR)-based tests detect p27 FeLV antigen
e. Confirmatory IFA tests don't usually yield positive results until 45 – 60 days after initial infection when secondary viraemia has occurred after infection of bone marrow.

5. Which one statement below is INCORRECT.?
a. Any new cats or kittens should be screened for FeLV infection before being introduced into a household.
b. Household cats that go outdoors or share a house with cats that go outdoors should be FeLV-tested at least yearly
c. Any cat that becomes clinically ill should be tested for FeLV immediately if it shares a household with an FeLV-infected cat.
d. Healthy cats which tests positive on the ELISA test should be euthanased
e. Household cats that may have been exposed to other cats with unknown FeLV infection status should be immediately tested for FeLV and retested six weeks after exposure.

6. Which one criteria listed below is NOT an indicator to vaccinate an individual cat against FeLV?
a. Kittens, because younger animals are more susceptible
b. Strictly indoor cats
c. Cats that have contact with cats with access to outdoors.
d. Cats that live with FeLV-infected cats.
e. Cats that may encounter other cats with unknown FeLV status.

7. Which one of the statements below regarding treatment of FeLV ill cats is INCORRECT?
a. Early therapeutic intervention is key to a successful treatment outcome in FeLV-infected cats that display clinical signs.
b. First, the clinician should determine whether the illness is directly associated with FeLV infection or secondary due to immune dysfunction.
c. Intensive diagnostic testing should occur earlier during a diagnostic workup as opposed to a "wait-and-see" approach.
d. Most FeLV-infected cats respond well to appropriate medications and treatment strategies, but they may require a longer or more aggressive course of treatment and need to be more closely monitored during recovery.
e. Antiviral drugs and immunomodulators are the cornerstone to managing cats with FeLV

8. Separation of FeLV-infected cats from uninfected cats is ideal . If an owner is not prepared to separate cats which one of the factors listed below is UNTRUE?
a. If an owner is unwilling to separate the FeLV-infected cat from non-infected cats then the non-infected cats should be adequately FeLV-vaccinated.
b. Vaccination of non-infected cats does guarantee 100% protection, even in high-exposure environments.
c. No new cats should be added to the household because this would disrupt the social structure and possibly increase the risk of cat fights and bites
d. However, providing separate feeding stations for infected and non-infected cats may help decrease the degree of exposure
e. FeLV is also labile outside of the host, remains infectious for only minutes in the environment and FeLV is readily inactivated with soap and disinfectants

.

9. Which one of the following statements is most correct?
a. A positive ELISA in a cat without clinical signs consistent with FeLV is less reliable in sick cats
b. A positive result in a healthy cat should always be confirmed
c. As the prevalence of FeLV decreases so does the percentage of false positive ELISA test results.
d. Cats with an antigen confirmed positive test cannot overcome the viraemia
e. Six weeks is the cut-off time for a cat to develop a regressive infection - ie for those with transient viraemia to overcome infection

10. Which one of the following statements is correct regarding a multi-cat household ?
a. 30 - 40% of the cats will develop persistent viraemia (progressive infection)
b. 30 - 40% of the cats will show transient viraemia (regressive infection)
c. 20 - 30% of the cats will show antibodies without being viraemic (abortive infection)
d. Regressively affected cats are not at risk for developing clinical disease
e. All of the above

The ABCs of Veterinary Dentistry

"M" is for "Malposition and Malocclusion"

In veterinary medicine, the goal of orthodontic correction isn't a pretty smile but pain-free, functional occlusion.

By Jan Bellows, DVM, DAVDC, DABVP, FAVD

"Are those braces on dogs' teeth?" This question, posed to me by a fellow passenger on my return flight from a veterinary conference, caused me to put my current task (subject-tagging images of dogs' and cats' mouths on my computer) on pause. I explained that in cats and dogs, the goal of orthodontic correction isn't a pretty smile but pain-free, functional occlusion.

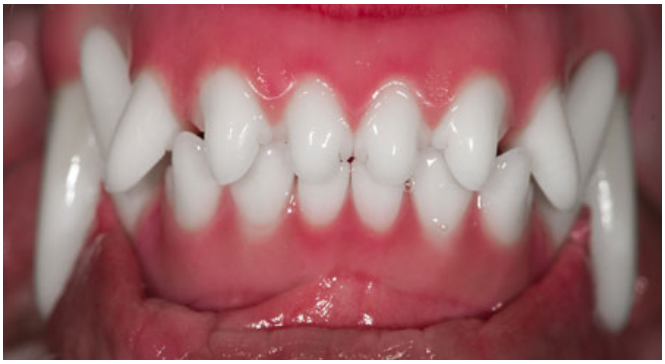


Figure 1A. Normal maxillary incisor overlap. (All images courtesy of Dr. Jan Bellows)

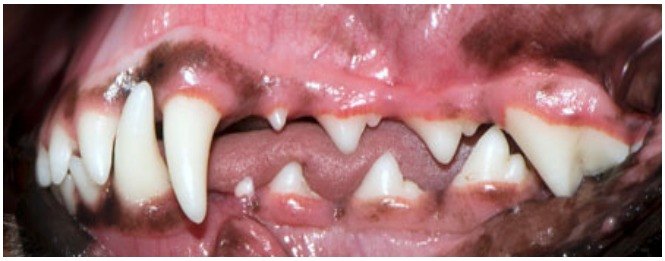


Figure 1B. A dog's left buccal view; normal interdigitation of canines and premolars.

What happens when you peek into the mouth of a patient and note that one or more teeth are out of place? Hopefully you don't quickly close the mouth, hoping that the pet owner didn't spot the problem. (Out of sight, out of mind.) It's much better to let your client know when something isn't right in their pet's mouth and explain what it will take to fix a poor or nonfunctional bite. But before you can recommend orthodontic care for your patients, you'll need to embrace the concepts of malposition and malocclusion.

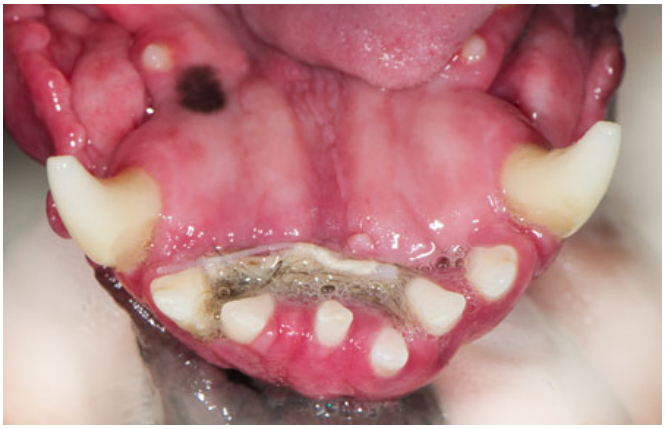
Occlusion

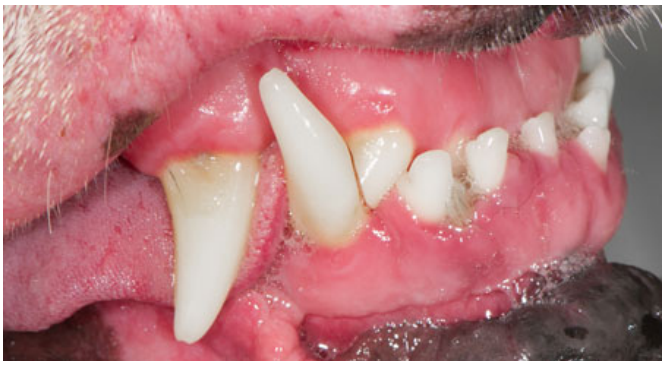
Occlusion refers to the relationship between the maxillary and mandibular teeth when they approach each other, as occurs during chewing or rest. Normal occlusion exists when the maxillary incisors just overlap the mandibular incisors *(Figure 1A),* the mandibular canines are equidistant from the maxillary third incisors and the maxillary canine teeth, and the premolar crown tips of the lower jaw point between the spaces of the upper jaw teeth in a saw-toothed fashion *(Figure 1B).*

Flat-faced breeds, such as boxers, shih tzus, Boston terriers, Lhasa apsos and Persian cats, have abnormal bites that are recognized as normal for their breed in which the mandibular jaw protrudes in front of the maxillary jaw, altering the above tooth-to-tooth relationship *(Figures 2A and 2B).*

Malocclusion and malposition

Malocclusion refers to abnormal tooth alignment. Skeletal malocclusion occurs when jaw anomalies result





Figures 2A and 2B. Normal but painful nonfunctional rostral occlusion in a boxer. Note the maxillary incisors penetrating the mandible.

in abnormal jaw alignment that causes the teeth to be out of normal orientation. Dental malposition occurs when jaw alignment is normal but one or more teeth are out of normal orientation. When dental malposition or skeletal malocclusion causes trauma to other teeth or oral soft tissues, the condition is termed poorly functional ornonfunctional and treatment is indicated.

Therapy options include moving or removing the offending or offended tooth or teeth, or surgically creating additional space for the malpositioned tooth to occupy without causing trauma.

Skeletal malocclusion

Here are some of the common terms associated with abnormal jaw alignment:

Mandibular distoclusion
(also called overbite, overjet, overshot, class 2, and mandibular brachygnathism) occurs when the lower jaw is shorter that the upper and there's a space between the upper and lower incisors when the mouth is closed. The upper premolars will be displaced rostrally (toward the nose) compared with the lower premolars. Mandibular distoclusion is never normal in any breed *(Figures 3A and 3B)*.

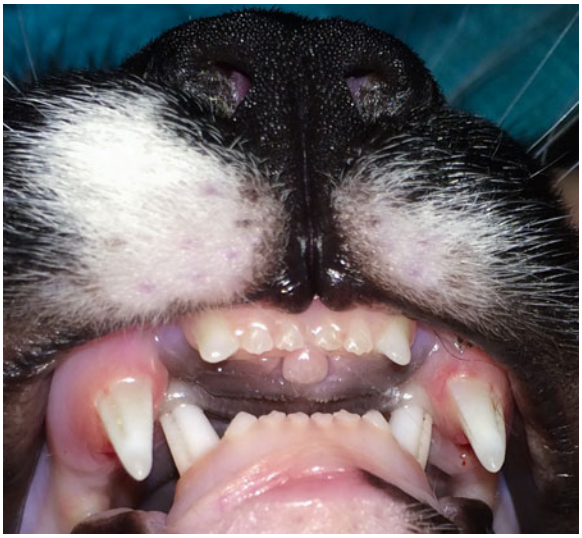


Figure 3A. A cat's mandibular distocluson and asymmetry.

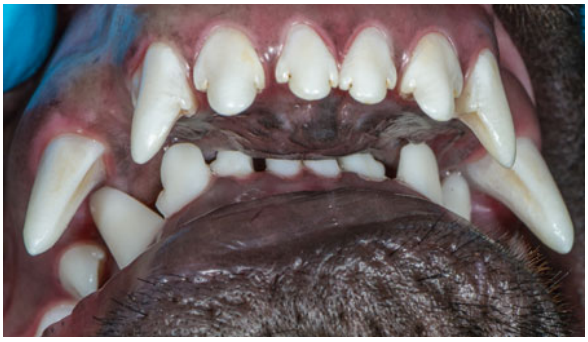


Figure 3B. A dog's mandibular distocluson.

Mandibular mesioclusion
(also called underbite, undershot, reverse scissor bite, prognathism, and class 3) occurs when the lower teeth protrude in front of the upper teeth. If the upper and lower incisor teeth meet each other edge to edge, the occlusion is an even or a level bite *(Figure 4)*.

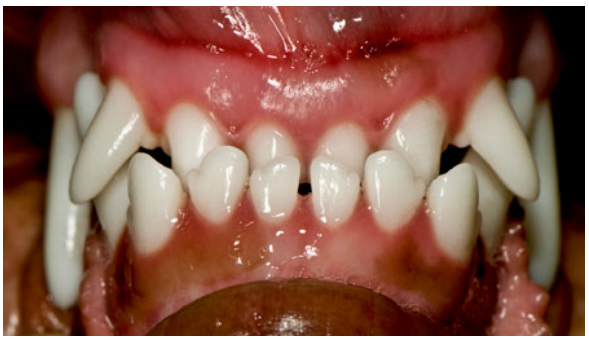


Figure 4. Mandibular mesioclusion in a dog.

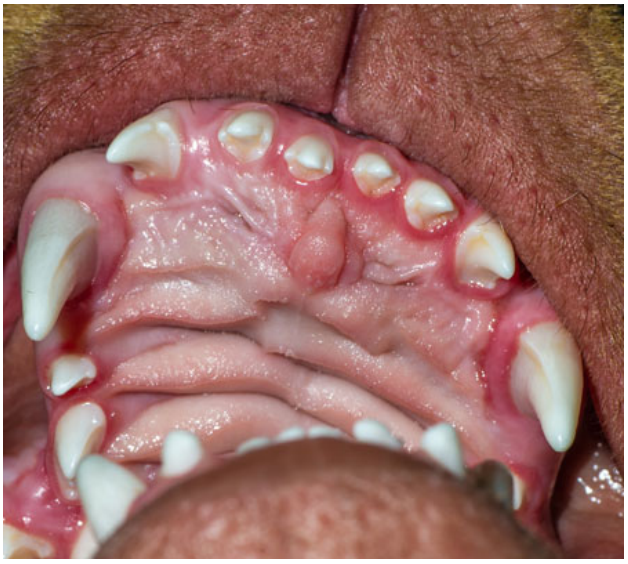


Figure 5A. Maxillary mandibular asymmetry in a dog.

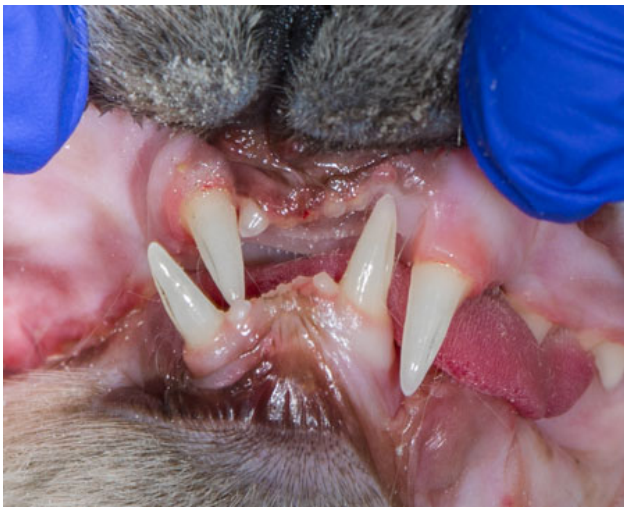


Figure 5B. Maxillary mandibular asymmetry in a cat.

Maxillary mandibular asymmetry
(also called wry bite, especially by breeders) is a skeletal malocclusion in which one side of the jaw grows differently from the other side *(Figures 5A and 5B)*

Dental malposition

Abnormally placed teeth can result in the following conditions:

Rostral cross bite
occurs when the canine and premolar teeth on both sides of the mouth are normally aligned but one or more of the lower incisors are positioned in front of the upper incisors *(Figure 6)*.

Mesioverted mandibular canines
(also called lingually displaced canines or base narrow canines) occur when the lower canine teeth protrude inward, impinging on or penetrating the maxillary gingiva *(Figure 7)*. Often this condition is due to retained deciduous teeth. The resulting trauma can be alleviated through tooth movement, crown reduction and restoration, or extraction.

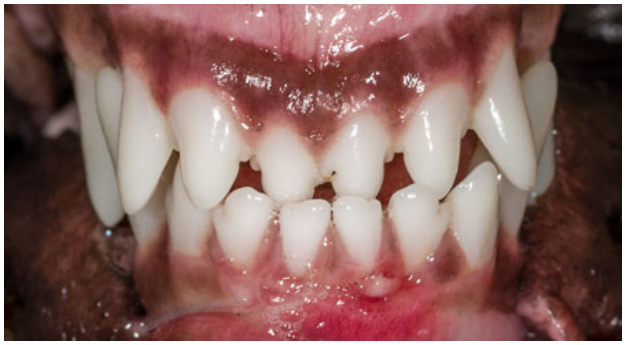


Figure 6. Rostral cross bite.

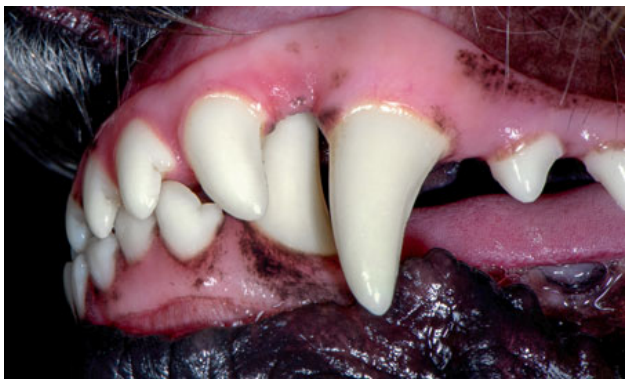


Figure 7. Mesioverted left mandibular canine.

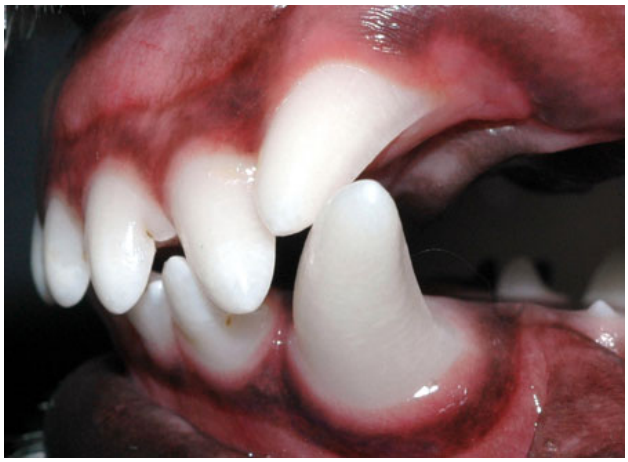


Figure 8. Rostroversion of the maxillary canine in a Shetland sheepdog.

Rostroverted maxillary canine
(also called lance canines) may be inherited (Shetland sheepdogs are prone to this condition) or developmental secondary to retained deciduous teeth *(Figure 8)*. Treatment includes moving the maxillary canine caudally with the help of orthodontic brackets and elastics, crown reduction and restoration, or extraction.

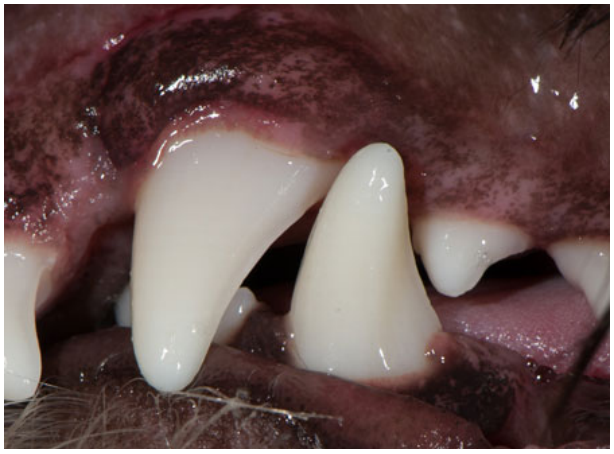


Figure 9. The left mandibular canine tooth is malpositioned but functional

Your patient has an abnormal bite. Now what?

The challenge with examining every dog and cat that comes through your practice for evidence of malocclusion or malposition is that your exams will uncover many abnormalities. However, this also means you have many more opportunities to improve your patients' health.

Consider these basic orthodontic concepts when tailoring a treatment plan for each patient with orthodontic anomalies. Is the abnormality functional?

If a tooth is out of place but isn't interfering with other teeth or with eating, and if it isn't penetrating the gingiva, a functional bite exists *(Figure 9)*. To repair a functional bite for cosmetic or show purposes isn't necessary and is considered unethical.

Options for correction

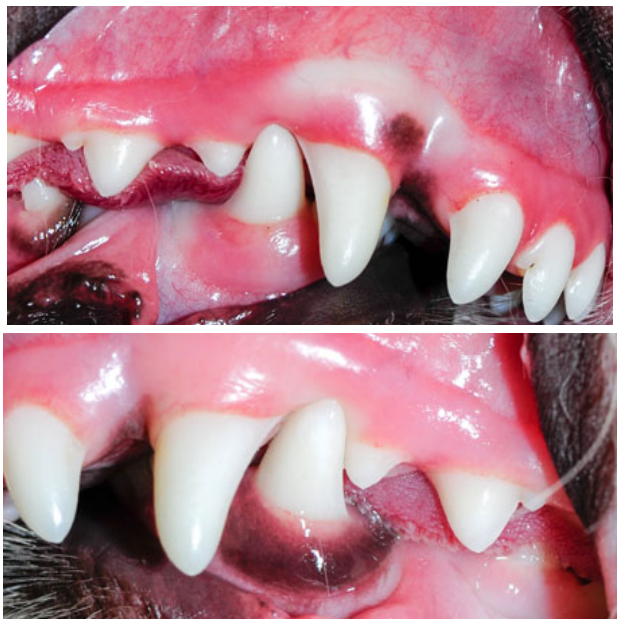
Here are the techniques and procedures that can improve quality of life in an animal with malocclusion.

1. Extraction. Extraction of the offending or offended tooth (or teeth) usually results in immediate relief. Extraction of the canines can be challenging, so consider referring if you aren't comfortable with the procedure or the possible surgical consequences

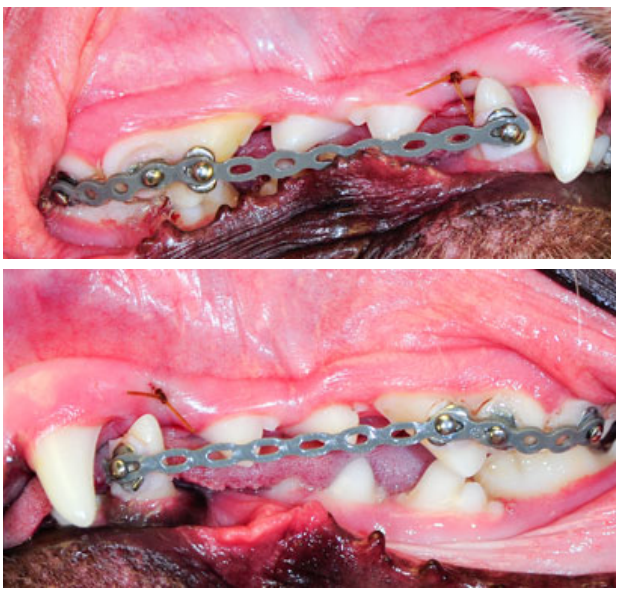
2. Tooth movement. Moving malpositioned teeth to functional positions can be both challenging and rewarding. Teeth are moved surgically or through the use of inclined planes, orthodontic brackets and elastics *(Figures 10A-10G)*. Orthodontic movement is an advanced dental procedure that should be performed only by someone with a thorough understanding of dental anatomy, physiology and orthodontic principles.

3. Crown reduction and restoration. Decreasing canine or incisor crown height will often resolve gingival impingement or penetration. This procedure preserves the vitality of the tooth through vital pulp or root canal therapy and restoration with light-cured composite (Figures 11A-11C). You can place a metallic crown for extra protection.

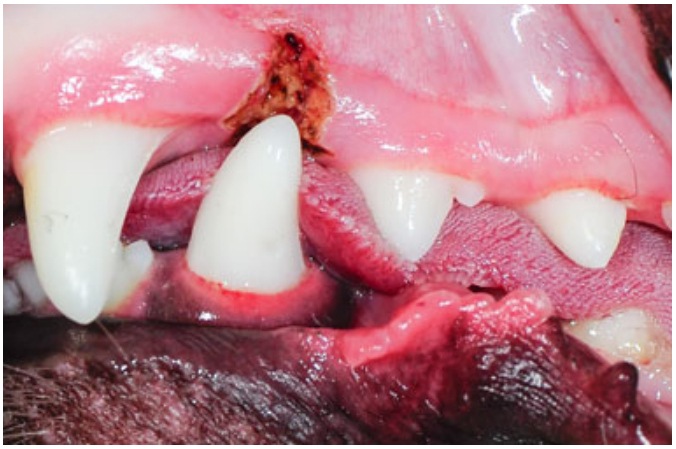
Understanding and embracing orthodontic correction will create smiles on your clients', patients' and team members' faces. Everyone wins.



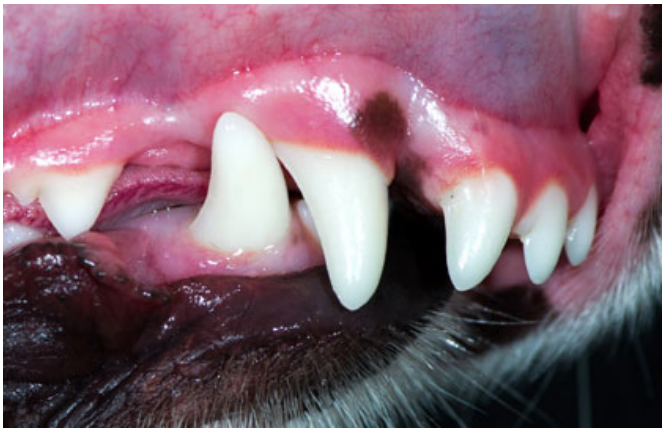
Figures 10A and 10B. Right and left mandibular canines impinging on maxillary gingiva

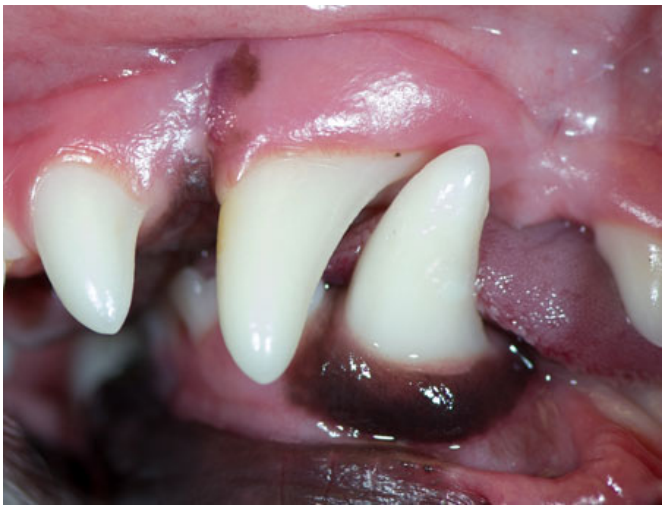


Figures 10C and 10D. Orthodontic brackets and elastics are used to move a maxillary canine caudally after extraction of the right and left maxillary first premolars.



Figures 10E. Laser gingivoplasty creates an inclined plane to push mandibular canines labially.





Figures 10F and 10G. Functional occlusion is created.

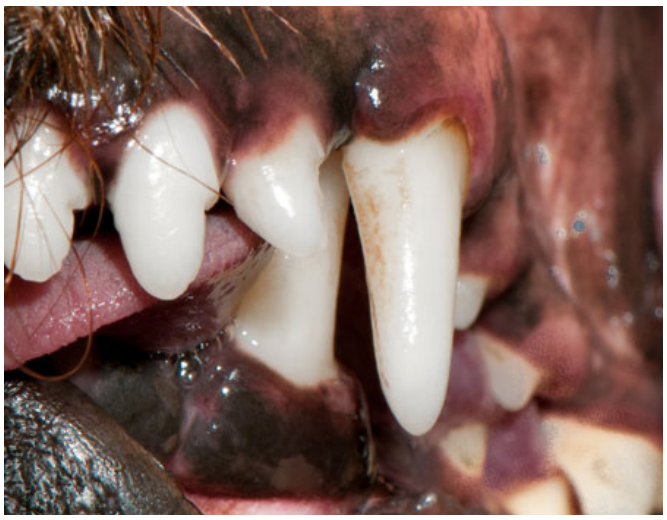


Figure 11A. Malpositioned left mandibular canine impinging on palatal gingiva.

Figure 11B. Left mandibular canine with reduced crown before restoration.

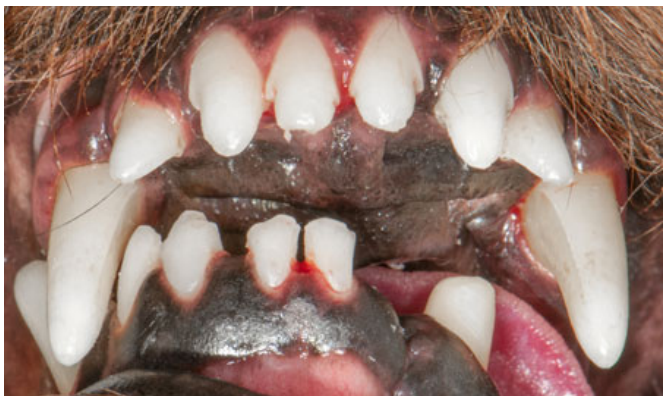


Figure 11C. Restored crown on reduced canine; impingement resolved.

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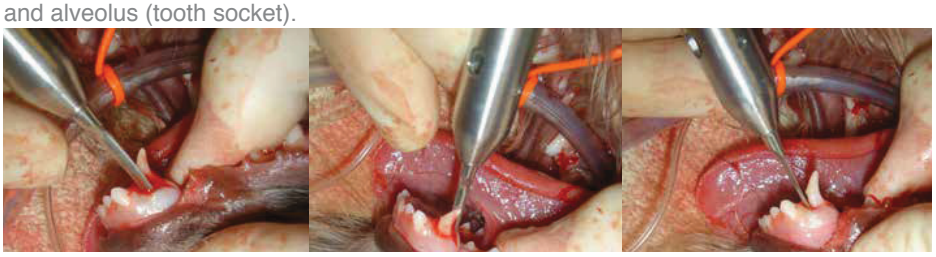

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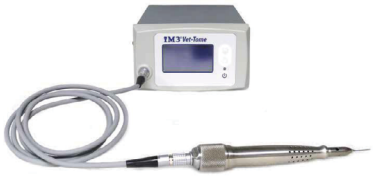

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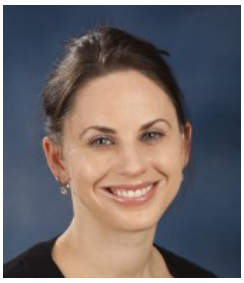
Artefacts in Radiography

An Old Topic Digitally Revisited



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One of the greatest advances in radiography over the thirty years is the advent of digital radiographic systems. These systems are generally divided into computed radiography (CR) and digital radiography (DR)

The linear response to radiation exposure by digital radiography systems, and hence wide dynamic range, is one of its biggest advantages and therefore these systems are more lenient of exposure errors compared to film/screen systems.[1] The ability to use image processing is also one of digital radiography's chief benefits, compared to traditional film-screen systems, in which the image could not be altered once acquired.[2]

The term "image processing" includes all of the procedures or processes (usually mathematical) applied to the image detector and the raw data collected by the system in the course of image acquisition.[2]

However, with the introduction of digital radiography, came the unavoidable introduction of its own set of artefacts. An artefact is an observed effect on the image or a structure that may spoil image quality, or may mimic or hide clinical pathology[1], hence their recognition and avoidance are important. The artefacts that can occur with traditional film screen radiography, such as motion artefact, poor patient positioning, incorrect identification, and poor patient preparation (such as debris on the skin)[1], can still occur in digital radiography and cannot be overcome.

This article will discuss the most common artefacts found in, and that are specific to, CR systems. Artefacts common to film/screen radiography will be mentioned briefly. Digital radiography (DR) will be discussed at another time.

Basic physics of CR

Computed radiography systems are cassette-based with a reading step after image acquisition. It uses a photostimulable phosphor imaging plate (IP) that is housed in the cassette for image acquisition. [3,4] When the IP is exposed to x-rays using standard radiographic equipment, electrons are excited to a higher energy state, which forms the latent image. [4]

The cassette is then placed into a CR reader, which opens the cassette and extracts the IP from within. Rollers within the reader move the IP so that it can be scanned by a red laser beam. This laser light frees the trapped electrons and allows them to drop back to a lower energy state, with the emission of visible green light as a result.[4]

The laser light and the released light photons have different wavelengths (longer and shorter, respectively), which allows the CR reader to distinguish the different light signals from each other. The shorter wavelength green light released from the IP is collected by a fibre-optic light guide and strikes the photomultiplier tubes (PMT). Here it produces an electronic signal, which is digitized and is stored in a display monitor, and can be sent to a picture archiving and communication system (PACS) and viewing stations.

Lastly, the IP must be exposed to a high-intensity (white light) lamp, which erases any remaining energy from a previous exposure. The IP is then replaced into the cassette and then can be re-used for the next x-ray acquisition.[4]

Artefact classification

Artefacts can be roughly classified according to the stage of imaging where they occurred. This may be prior to image acquisition (storage and handling of the cassette), during image acquisition (making of the radiograph by the user or radiographer), or during image processing (from extraction of the IP into the reader, to the computer processing on the workstation).[3,4] By identifying the step where the artefact occurred, it can be more easily corrected.
The most logical classification system may be that utilised by Jiménez and Armbrust[3,5] who divides the artefacts into pre-exposure, exposure, post-exposure, reading, and workstation artefacts.

Pre-exposure artefacts

These artefacts occur before the cassette is used for a study, and may occur with something as innocuous as storage of the cassette.

Storage scatter

Exposure of the cassette to extraneous radiation, such as storing the cassette nearby the x-ray unit when it is in use, will result in a degree of exposure of the IP. This is likened to "fogging" in film-screen radiography. It may result in a generalised increase in darkening of the image and may be difficult to identify, or may result in a distinct pattern of exposure, which may occur if the cassette was stored behind/under a structure that attenuates the beam (a shelf or table as examples) *(Figure 1).* Ways to avoid this artefact include storing the cassettes away from the x-ray unit, and erasing the cassettes if they have not been used for a period of time, as storage scatter can accumulate over time.

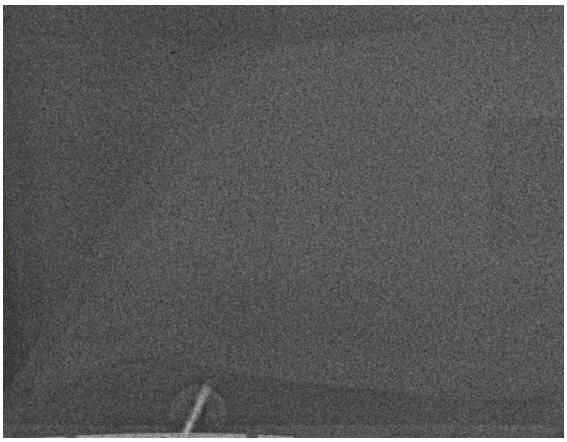

Figure 1: Note the marked generalised increased image darkening, as result of multiple small grey opacity speckles on the imaging plate. There are also patterns seen within the image, because the cassette was stored behind an unknown object when radiographs were made.

Damage to the imaging plate, "cracks" or "roller artefacts"

Physical damage to the IP plate is usually seen as focal or linear white artefacts on the image, and are referred to as "cracks" or "kinks" *(Figure 2).* Damage may arise from wear-and-tear over time, as the IP is removed from the cassette during the reading process and transported with rollers through the reader over and over with use. Damage can also occur if the IP is removed from the cassette during maintenance or cleaning.

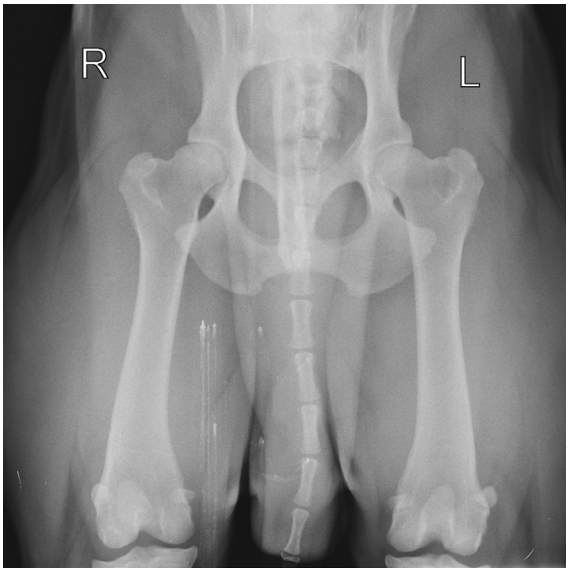

Figure 2: Damaged imaging plate. Note the interrupted vertical white lines on the image, medial to the right femur, as well as two curved artefacts are also seen on either side of the image, in the left and right-had bottom corners. Roller marks may be similar in appearance, and may span the entire length of the image.

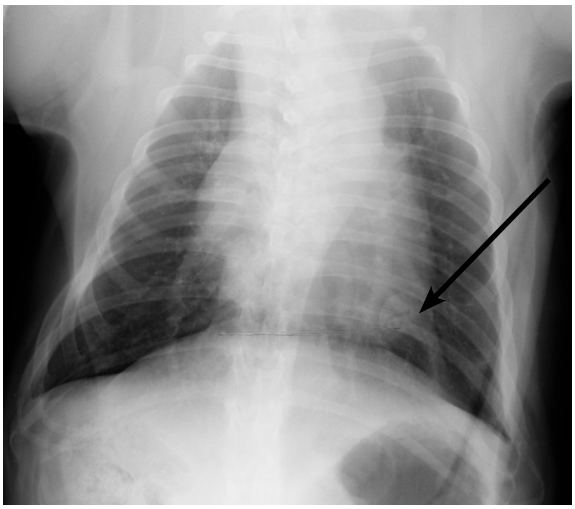

Figure 3: Partial erasure artefact. The imaging plate was not adequately erased prior to obtaining the next image. Note the faint body marker disk visible in the left caudal hemithorax over the cardiac silhouette as indicated by arrow.

Once cracks have occurred, the plates need to be replaced. Routine maintenance of the reader and careful handling of the IP, preferably with cotton gloves during cleaning, is recommended to prolong the life of the IP.

Partial erasure artefact

If the IP is not completely erased by exposure to a bright white light, which occurs after reading, the previous image acquired may be retained on the IP as a faint image superimposed over the recently acquired one *(Figure 3)*. This artefact may occur if the white light is faulty (dirty, damaged) or if excessive radiation doses were used, which result in incomplete erasure of the image due to excessive energy stored on the IP. Regular maintenance of the cassette reader, and avoidance of over-exposure, will remedy this artefact.

Phantom image artefact

This artefact may appear very similar to the partial erasure artefact, but is created in a different manner. If the IP is unused for a few days prior to image acquisition, the previous latent image may be seen faintly over the newly acquired one. Erasing the IP before use will avoid this artefact.

Exposure artefacts

These artefacts occur during image acquisition, and several are like those occurring with film/screen radiography, such as upside-down placement of the cassette, grid cut-off, back-scatter, and double exposures. One additional remark is that, unlike with film/screen radiography, a double-exposure with CR does not result in increased image darkening, and hence identification of this artefact may be hampered.

Quantum mottle and underexposure

The fluctuations in the number of x-ray photons throughout the image, called quantum mottle, becomes more prominent when too few data (or x-ray photons) reach the IP (underexposure) *(Figure 4a)*. If low mAs techniques are used or if there is increased attenuation of the primary x-ray beam, underexposure will result which leads to a pixelated or grainy speckled appearance of the image. Using a technique chart to determine to optimum exposure values for each patient is recommended.

Saturation and overexposure

Imaging plates record an image by storing energy proportional to the amount of x-ray photons incident per unit area of the plate. When the maximum storage capacity of a CR IP is reached, the corresponding pixel produced appears entirely black, hence there are physical limits to the amount of energy that can be stored *(Figure 4b)*. Further increasing the exposure will not result in any change to the image, but will increase patient exposure.

Despite the forgiving nature of CR with regards to exposure mistakes, processing will not be able to reverse the darkening of the image. Reducing exposure levels, and using a technique chart, is recommended.

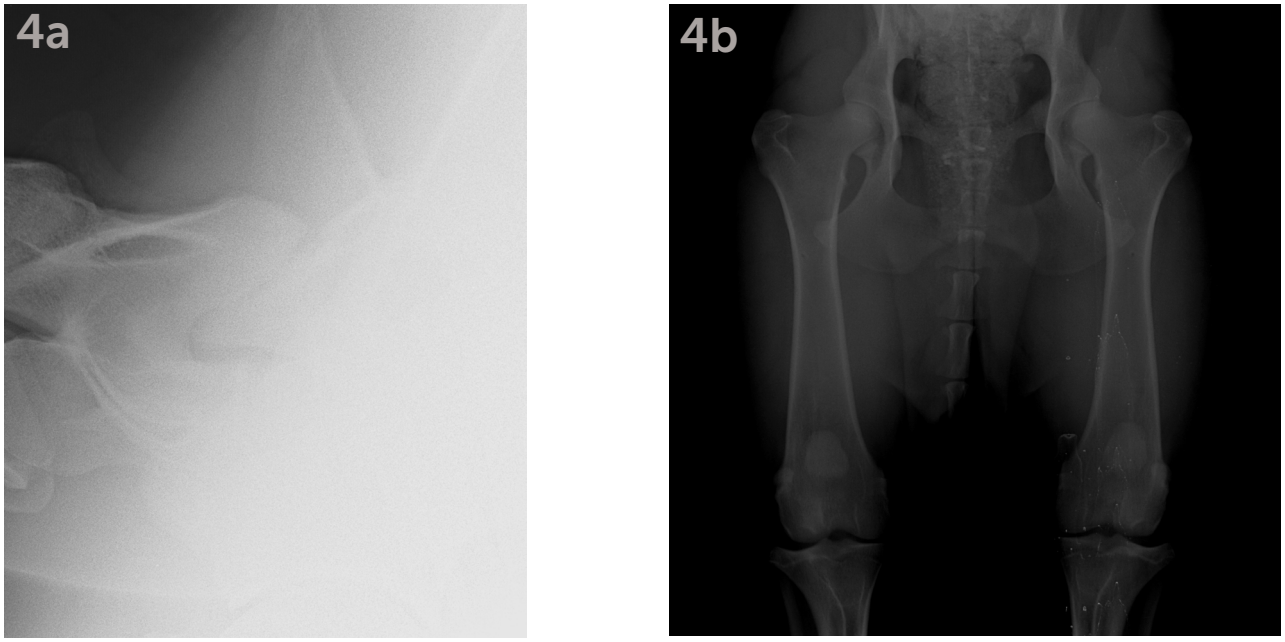

Figure 4: Underexposure (a) and over-exposure (b). The image on the left (a) is of the caudal cervical and cranial thoracic vertebra of an equine, which is typically where under-exposure occurs due to the size of the patient. However, the appearance is similar in small animals, and careful inspection of the image reveals a fine grainy appearance over the underexposed regions, especially the right-half of the image. The image on the right (b) demonstrated marked over-exposure and loss of soft tissue and bony detail especially of the thinner body parts. Neither images can be corrected by processing.

Incorrect collimation

Too much or too little collimation can affect image quality. Without any collimation, the IP reader assumes that the images have been overexposed and makes the image too light as result. With too much collimation, the plate reader assumes the image is underexposed and will correct for it by making the image dark. These artefacts are remedied by adequately collimating to the region of interest, and using the smallest cassette necessary for the part of interest.

Post-exposure artefacts

Light leak

White light is utilised by the CR reader to erase the image, prior to using the cassette for the next exposure. Exposure to external ambient light, before reading the IP, will have a similar effect, and results in removal of part of the image and a white artefact or underexposure on the image. It may take several minutes to occur, and the severity depends on the size of the portion of the IP exposed to light. The appearance of this artefact is opposite to that seen in film/screen radiography, where light leaks lead to severe darkening of the film. It can be avoided by ensuring that the cassette is adequately sealed and not opened prior to reading the IP.

Fading, or delayed scanning

Putting an exposed IP plate through the reader several days after image acquisition, will result in a grainy, underexposed and poor-quality image *(Figure 5)*. This is because the excited molecules within the IP gradually lose their energy over time through spontaneous phosphorescence, resulting in loss of the image.

It has been suggested that 25% stored signals will be lost within 8 hours after exposure and that statistically significant degradation of contrast occurs in IPs scanned merely 30 minutes after exposure. These artefacts are avoidable by reading the IP immediately after each exposure/image acquisition.

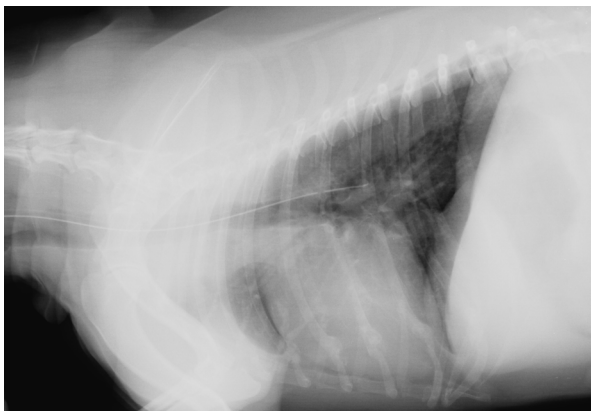

Figure 5: Fading artefact. The image was obtained 48 hours prior to reading, and shows overall loss of contrast with an underexposed appearance as result of fading.

Reading artefacts

Similar to film/screen radiography, debris such as hair or dirt within the cassette, will interfere with reading of the IP and result in white artefacts on the acquired image. Any foreign material (hair, dirt) will block light from being read and results in a well-defined/crisp white artefact on the image.

Dirty light guides

A red laser strikes the IP one line at a time during reading, and the IP releases its stored energy as green light. A light guide is needed to direct this light into the photomultiplier tube. If any light is blocked within the light guide, it will not reach the photomultiplier tube, resulting in a thin white line along the image, in line with the direction of the IP translation through the reader. Routine maintenance of the reader will include cleaning of the light guide.

Skipped scan lines

The IP is moved at a programmed and consistent rate through the reader as it is struck by the red laser. If there is any interruption of this movement, image information may be omitted, misplaced or incorrectly represented. This artefact is evident as the absence of a thin segment of the image, but with positioning of the remaining image on either side of this. Irregular movement of the IP plate may be due to power supply fluctuations to the reader, or due to the IP becoming jammed or obstructed.

Moiré pattern and grid usage

Computed radiography images are read one line at a time. The sampling frequency of a CR unit refers to the number of lines read per unit distance, and may differ between CR units. A grid also has regularly repeating attenuation due to the parallel lead strips, and hence also has an inherent frequency equal to the number of strips per unit distance. If the grid frequency and sampling frequency of the CR intersect, a series

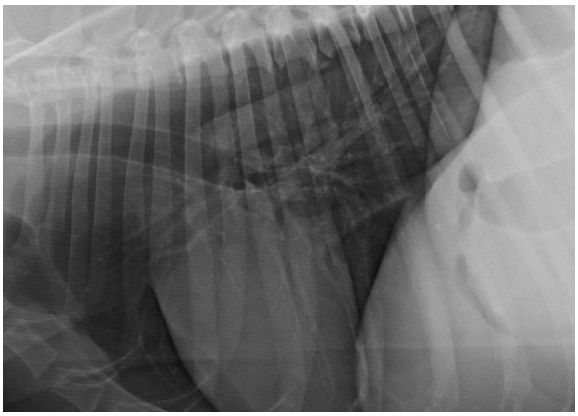

Figure 6: Moiré artefact. Note the parallel horizontal white lines on this thoracic radiograph, consistent with Moiré lines.

of points of higher attenuation results which has the appearance of straight or curved lines on the resultant image *(Figure 6)*.

This artefact typically occurs with stationery grids with a low grid line rate/frequency, or malfunction of an oscillating grid. Several actions can be taken to remedy this artefact, depending on what grid is used, but includes: using an oscillating grid (Potter-Bucky), using a grid with high ratio, ensuring correct perpendicular alignment of the grid to the sampling direction, or increasing exposure times (if an oscillating grid is already used).

Workstation artefacts

Faulty transfer

After image acquisition in CR, the data from the IP plate needs to be transferred to the workstation. Any disruption in this process can result in misplacement of distortion of body parts or regions of interest on the resultant image. Parallel streaks, elongation, or replacement of portions of the image with areas that are completely black or white can occur. Parts of the radiograph may also be incorrectly localised on the image, or duplicated or superimpose over each other, or pixels may be missing. Stable data transfer, a stable connection and reliable power source is required to prevent this artefact.

Border detection

In CR, there is no direct communication between the x-ray tube and the cassette, and thus the workstation does not "know" how much collimation has occurred or information about the image size. Thus, the workstation is automated to detect the collimated margin and crop the image accordingly, however if this is done incorrectly may lead to omission of parts of the image or inclusion of the area outside the primary x-ray field. This has a significant effect on post-processing, as the regions excluded from the image are not processed. This artefact usually occurs at the margin of highly attenuating objects/structures, with an off-centred object of interest, and when the IP is rotated even slightly relative to the collimated field.

Clinically, it is most commonly seen when trying to obtain multiple images on the same cassette, which is typically not a problem in film/screen radiography. If this artefact is present, then processing on the workstation needs to be performed, either by removing border delineation or by reprocessing the image with deactivated border detection.

Diagnostic specifier

"Post-processing", or the ability to alter the image after obtaining it, is one of CR's major advantages. Some of this is done automatically by the workstation. A histogram, or a graphic representation of all of the opacities on the image, is generated and analysed by means of a lookup table (LUT). LUTs are designed to display optimal grayscale, contrast, and detail, and are based on specific diagnostic regions. The LUT for an

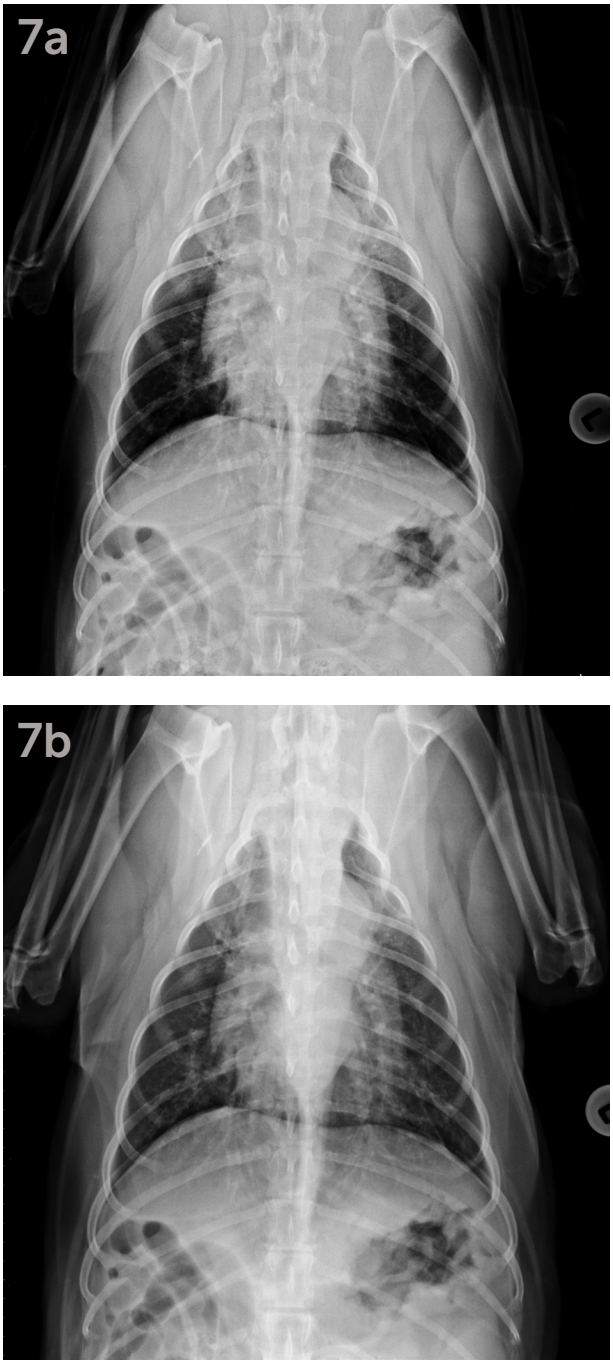

Figure 7: Selection of the incorrect lookup table (LUT). The two images were obtained using identical exposure factors and are of the same patient, but with different LUTs selected. The image on the top (a) was obtained using an "abdomen" LUT, while the image on the bottom (b) was obtained with the correct "thorax" LUT. Note that the "abdomen" LUT results in loss of the peripheral soft tissues in the image, as well as loss of the pulmonary detail, especially noticeable in the caudal lung lobes, and overall gives a higher contrast image which is undesirable in the thorax.

abdomen, thorax, or extremity, are all different and application of the incorrect one to a region of interest (for example, selecting thorax when imaging the abdomen) will result in decreased overall quality, and modification of opacity, contrast, or detail. To mitigate this artefact, manual processing can be performed at the viewing station, or the image reprocessed with the correct LUT lookup table at the workstation (i.e. selecting thorax for a thoracic study). Repeating the radiograph is usually unnecessary *(Figure 7 a and b)*.

Clipping

Raw CR files are usually obtained in 12 to 14 bits, resulting in very large files that are cumbersome to transfer. Thus, they generally are "clipped" to 10 or 12 bits by discarding some information, but can result in complete darkening of areas of higher x-ray exposure. This is different to "saturation" artefact, which is as result of overexposure. Clipping occurs within the normal or correct dynamic range of the x-ray, and is as result of post-processing and does not occur during the acquisition phase. This artefact can be remedied at the workstation, but once the images are sent to the viewing station, cannot be corrected as the lost information cannot be retrieved.

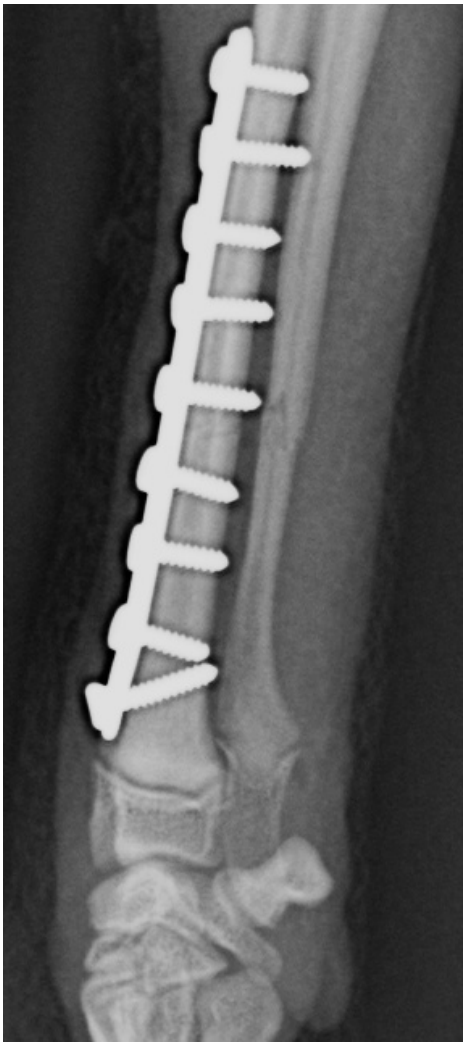

Figure 8: Note the fine radiolucent zone surrounding the surgical plate and each screw. The fact that it extends into the interosseous space between the radius and ulna, and is present dorsal to the plate, also helps to identify this as an artefact rather than lysis.

Density threshold

When objects of very high density, such as metallic implants, are included in histogram analysis and application of the LUT, the displayed grayscale is widened to include these objects. The remaining biologic tissues appear dark and have decreased contrast between them because of this widened greyscale and thus decreased contrast levels. Application of a "density threshold" will allow these structures to be excluded, by processing the image such that anything above a certain density will be excluded from the histogram, and appear white. This allows biological tissues (the region of actual interest on the radiograph) to be displayed with the correct contrast levels and opacity.

Uberschwinger, "rebound" or "halo" artefact

This artefact occurs as a thin black line surrounding objects of higher attenuation (such as metallic implants), which runs evenly and parallel to the object and are adjacent to areas of lower attenuation (for example, a metallic screw placed within bone for fracture repair) *(Figure 8)*.

The radiolucent zone around the implant may be mistaken for osteolysis, which would lead one to the erroneous diagnosis of implant loosening or infection. In contrast to osteolysis, uberschwinger is of even thickness and surrounds all metallic implants on the radiograph, versus more irregular or focal lucencies of infection or loosening.

Conclusion

Identification of and pinpointing the source and type of CR artefacts is important to prevent diagnostic errors with the possibility of medicolegal implications. At best, the decreased image quality is merely an irritation to the veterinarian evaluating the image. Several artefacts are due to operator error, and thus proper training of the staff acquiring the images is necessary, as well as careful use of the equipment and regular maintenance.

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Introduction to Direct Digital Radiography (DR)

Kat Evans

Veterinary practices are frequently described as having digital radiography, however, this can be a broad and often inaccurate description. To prevent confusion, it is recommended to use the terminology Direct Digital Radiography (DR) or Computed Radiography (CR) when describing 'digital radiography'. In this article, we will discuss the theory behind DR, and answer some frequently asked questions.

What is DR?

Put simply, DR systems convert x-rays directly into a digital signal after they have passed through the patient. This information is then relayed to a computer to enable you to see the radiograph on a monitor. This differs from CR which requires a cassette to be read or processed to enable visualisation of the image.

All digital systems will also apply algorithms, which are also known as LUTs (Look Up Tables). These take the raw image, which is normally very flat, and enhance it to give us a usable image. Algorithms, and therefore image appearance, can vary enormously depending upon the quality of the equipment from different manufacturers. Different DR systems will use different receptor technologies to capture the raw image, but most are based around a layer of Thin Film Transistors, known as flat panel detectors. These are further sub-divided into direct conversion and indirect conversion detectors.

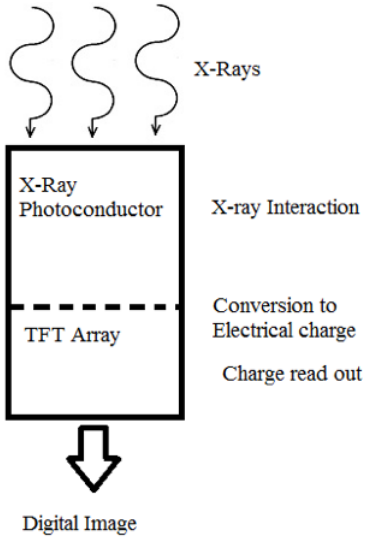

Figure 1. Direct conversion DR detector

Direct conversion DR detector

In direct conversion detectors (Figure 1) x-ray photons hit the receptor and interact with the atoms of the photoconductor, converting the energy from the x-ray photons into an electrical charge. The electrical charge is then used by the thin film transistor array to produce a digital image. The information that is used to create the image is the location and strength with which the photons hit the TFT layer. This interaction works most effectively if the incoming x-ray photons have a high energy level, *i.e.* are produced using a high kV.

Physical manufacturing limitations restrict the resolution achievable by direct conversion. An array of tiny detectors is used because it is physically impossible to manufacture one single large detector and inevitably this results in small 'gaps' between the individual detectors. The photoconductor is designed to maximise resolution by channelling incoming ions towards the detectors. There are a range of photoconductors used (the most common is amorphous selenium) which vary in their sensitivity to radiation, resolution and their ability to cope with environmental stresses.

Indirect conversion DR receptor

Indirect DR receptors (Figure 2) have more layers than direct DR receptors. There is a layer of scintillation

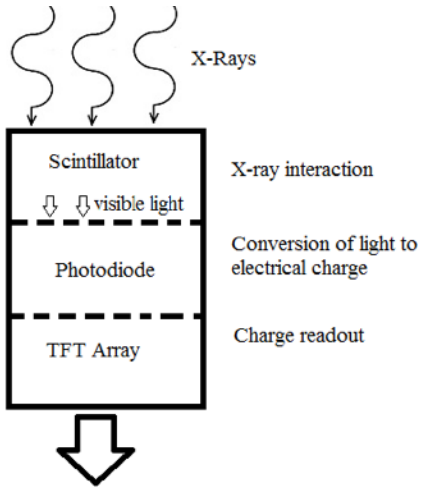

Figure 2. Indirect conversion DR receptor

material which covers the front of the photodiode, this effectively works in the same way as the intensifying screen in a traditional X-ray cassette. The scintillation material emits light when stimulated by radiation. The indirect stage of the conversion is then the transmission of this light by the photodiode into an electrical signal. In comparison to direct conversion DR receptors, the 'extra layer' allows a lower exposure to be used when acquiring an image. The increase in number of layers (or times the information is 'processed') before an image is generated also results in a slight loss of resolution. Therefore DR receptors utilising indirect conversion will inherently result in images with lower resolution than direct conversion DR receptors.

DR FREQUENTLY ASKED QUESTIONS

Which should I choose?
Historically, indirect conversion receptors have been preferred due to the lower radiation dose required. Direct conversion is now becoming more popular as the sensitivity of TFT layers improves, however, there have been more issues related to the stability of the direct conversion systems; some detector panels fail if they are taken outside certain temperature ranges!
Currently indirect conversion is still more common in human medicine, with very few direct panels in use.

Why do some panels cost more than others?
Very often quality is the answer. In the same way a television or computer monitor is made, there will always be a number of 'duff' pixels on a DR panel. When quality assurance is applied, they are then categorised by the failure rate, i.e. the less failed pixels, the lower the failure rate and the higher quality the panel. When calibrating a detector panel, the DR system builds up a map of defective pixels and then hides them, so that you don't see them on the image, but this doesn't mean they are not there!
Fundamentally, higher resolution panels are harder to make, so this will impact cost.

Why does temperature matter?
All DDR panels are sensitive to temperature changes and this can affect the required X-ray exposure. To compensate for this, many systems will recalibrate if they detect a temperature change, however, all will perform less well in extreme cold because they were originally designed to be used at room temperature. Therefore, we recommend never leaving them in unheated vehicles overnight. It is also worth remembering that wireless systems rely on batteries within the plate. Modern, rechargeable batteries have improved a great deal, but still don't cope well with the cold which can cause rapid loss of charge.

Why do they break when dropped?
The scintillation layer is normally coated onto a sheet of glass. It needs to be affixed to a thin, strong clear surface and glass functions well. The panel can cope with normal use and much work has gone into making them drop resistant, but they are very complex inside. Direct blows from a horse's hoof or repeatedly being dropped onto a hard surface will take its toll. Most are designed to hold the weight of a human patient with the weight spread evenly over the plate. The higher risk of damage comes when pressure is applied to a point. A kick is likely to be most damaging, but this risk can be reduced by using a plate protector.

Why does image quality change over time?
All DDR technologies will age over time as radiation can be harmful to electronics, eventually affecting the sensitivity of a system. This impact can be reduced by servicing, performing gain calibrations and offset recalibrations. This will also help with any pixel drop out that has occurred over time.

Why does it take so long to repairs plates?
Unlike repairs to most systems, which may be possible via computer remote, or in-house if electrical, or maybe a simple part replacement, the repair of a DR receptor panel requires a very specific environment (sterile, static-free, dust-free and airlocked) and technical skill level. This level of repair is typically only available direct from the panel production facility.

IMV imaging supplies a variety of fixed and portable DR systems into the South African market, all with specialist vet software and pricing options to fit your practice best. Contact us via our website http://www.imv-imaging.co.za/ for more information, or call Tim on 082 616 4685.

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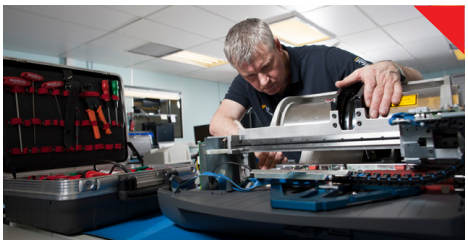

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Urinary Incontinence in Dogs

Medical Management

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Introduction

The urinary bladder is responsible for storing urine until it is convenient and appropriate to void urine. Both urine storage and bladder emptying require normal functioning and interaction of the autonomic and somatic nervous systems. In addition, these two processes also require that the parts of the lower urinary tract (ureters, bladder, bladder sphincter, urethra) are intact and functioning normally. For an animal to consciously and voluntarily initiate voiding, the cerebrum, brainstem, lumbar and sacral spinal cord also need to be intact and functioning.

Urinary incontinence is defined as the loss of voluntary control over voiding. Rather than intentionally voiding urine and emptying the bladder, the patient suffers from intermittent or continuous dribbling of urine. Most animals are still able to voluntarily empty their bladder at appropriate times and in appropriate places but dribbling of urine occurs throughout the day and night in inappropriate places, especially when the animal is lying down or sleeping. Many pets are not aware of the fact that they are dribbling urine. The prevalence of urinary incontinence varies between 5-20%.

Diagnostic approach

Urinary incontinence can result from a large number of neurogenic or non-neurogenic causes with non-neurogenic incontinence being the most common cause in small animal practice. Of all the non-neurogenic causes of urinary incontinence, primary urethral sphincter mechanism incompetence is the most common urinary storage disorder in dogs.

When a patient is presented with a complaint of urinary incontinence it is important to have a systematic approach during the diagnostic process. Information that needs to be collected includes the signalment, complete medical and surgical history, observation of the voiding process, a complete physical examination, a detailed neurological examination, palpation of the bladder, complete urine analysis with urine culture and antibiogram, haematology and serum chemistry if indicated, imaging of the urinary tract which may include radiography, ultrasonography, contrast studies (cystography and urethrography) and urethroscopy and cystoscopy.

When inappropriate urination is reported the clinician will need to distinguish between loss of voluntary control versus behavioural causes (submissive urination), urge incontinence, consciously urinating in inappropriate places or diseases resulting in polyuria and polydipsia such as chronic renal failure. The owner also needs to be questioned about the dog's ability to voluntarily initiate a urine stream, the force of the urine stream, interruptions in the urine stream during urination, presence of haematuria and any pain the dog may be experiencing during urination. Perineal soiling and urine scalding needs to be investigated.

Physical examination may reveal an enlarged/overdistended bladder which is more commonly seen in cases with neurologic disease or partial urinary tract obstruction. In cases with non-neurogenic disease the bladder is usually normal sized or small at examination. Palpation of a fairly full bladder after voiding may indicate incomplete voiding and urine retention. Passing a urinary catheter may determine the possibility of a partial urinary tract obstruction.

Neurogenic causes include upper motor neuron diseases of the brain or spinal cord, lower motor neuron damage seen in trauma for example and reflex dyssynergia. A complete neurological examination will aid in diagnosing these causes. A rectal examination to assess anal tone may also be useful in determining neurologic diseases (lower motor neuron) as the cause of the incontinence.

Although the most common non-neurogenic cause is urethral sphincter mechanism incompetence (USMI), other non-neurogenic causes need to be investigated and excluded. These causes include urinary tract inflammation and infection (which results in urge incontinence), ectopic ureters, urethral diverticulum, pelvic bladder, post-prostatectomy incompetence in male dogs, ureterocoele and a few other less common causes. The rest of this paper will focus on the management of USMI and the reader is referred to other texts for detailed discussions on diagnostic procedures and management of other causes of inappropriate urination and urinary incontinence mentioned in this section.

Urethral sphincter mechanism incompetence (USMI)
As mentioned before, USMI is the most common cause of urinary incontinence in small animal practice (60% of urinary incontinence cases) and is most commonly seen in adult spayed female dogs.

Optimal functioning of the urethral sphincter depends on the smooth muscle of the urethra as well as tissues surrounding the urethra, the urethral submucosal vasculature, collagen content and the urothelium. Decreased tone and responsiveness of the muscle together with changes in the peri-urethral tissue results in sphincter failure. In female dogs these changes are associated with a reduction in oestrogen and oestrogen receptors and an increase in follicle-stimulating hormone and luteinizing hormone after being spayed. The condition was previously referred to as hormone responsive or oestrogen responsive incontinence, but with a better understanding of the complexity of the pathophysiology involved, this name has mostly been abandoned.

USMI generally occurs around from about 3 years after ovariohysterectomy in up to 20% of female dogs spayed between the first and second heat. Some authors report that dogs spayed before the first heat had a 9.7% incidence of USMI. Urethral closure pressure was shown to decrease within 12-18 months after spaying a bitch. In one study in dogs spayed before 3 months of age the risk of USMI appears to be increased, but other studies have failed to show any relationship to the age at which the dogs were spayed. Dogs showing signs of incontinence since birth or since acquiring the puppy shortly after weaning should be examined for congenital deformities such as ectopic ureters.

A recent study investigated the relationship of age at spaying and the projected adult weight of the dogs. This study found that dogs weighing more than 25 kg adult weight were more likely to develop USMI if they were spayed at a younger age. It is possible that the impact of decreasing oestrogen may have less of an effect on developed adult tissues compared to developing tissues.

Several breeds seem to be predisposed to USMI and the incidence in large breed dogs weighing more than 20 kg is reported to be up to 30%. Doberman Pinchers, Giant Schnauzers, Old English Sheep Dogs, Rottweilers, Weimeraners and Boxers are some of the breeds that seem to be overrepresented. Female dogs of predisposed breeds may benefit from delaying time of spaying and thus decrease the risk of USMI. The same tendency was not seen in small breed dogs and therefor timing of spaying can still be based on potential benefits such as decreased risk of mammary neoplasia with early spaying.

Previously body condition score (BCS) was thought to be associated with development of USMI with obese dogs supposedly being predisposed to UMSI. The latest study did not confirm this relationship, but the authors commented that assessment of BCS is not standardized and future studies need to consider using more accurate and standardized evaluation methods before any concrete conclusions can be made regarding the role of obesity.

Conformational predisposing factors may also increase the risk of PSMI. These include pelvic bladder, short urethra and recessed/juvenile vulva. Dogs with USMI usually have adequate bladder capacity and are able to empty the bladder normally. Incontinence in these cases occur due to increased pressure intra-abdominally. This occurs for example when an animal lies down, especially when it is relaxed, or when pressure increases due to barking or coughing or after exertion.

The reasons for incontinence in male dogs is poorly understood, but a decrease in testosterone leading to an increase in follicle-stimulating hormone and luteinizing hormone may play a role.

In a clinically healthy spayed female dog that was urinary continent before being spayed and has a normal result on complete urine analysis, a presumptive diagnosis of USMI is justified. In all other cases a more intensive investigation is warranted.

Medical treatment of USMI

Medical management of USMI is usually considered the first line of treatment, but some clinicians prefer surgical options and in cases that do not respond to medical management surgical options should

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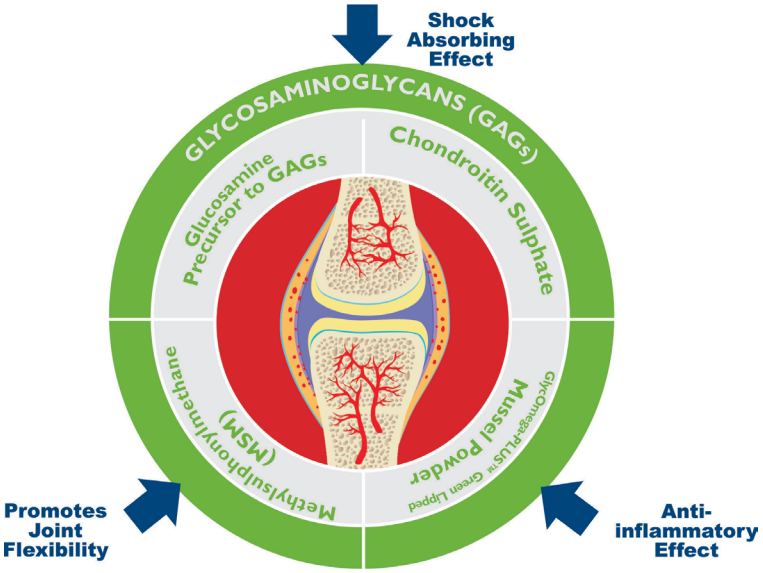

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be considered. The aim of medical management is to either increase the sensitivity of alpha-receptors, increase the number of alpha-receptors in the urethral sphincter or both.

Alpha-adrenergic drugs

- **Phenylpropanolamine**
  Many clinicians consider phenylpropanolamine (PPA) to be the first choice in the treatment of USMI. PPA is an alpha-adrenergic agonist which is associated with treatment success rates of 75-90%. The dosage for individual animals may vary quite widely but starting doses of 1.0-1.5 mg/kg *per os* every 8-12 hours appears to be effective in most cases. Over time the response to treatment may decrease and increases in dosage may be required. A 2011 study showed that once daily dosing may be adequate. A single daily dose of 1.5 mg/kg was successful in controlling incontinence in 88.9% of clinical cases in this study. It was shown in healthy beagles that PPA reached therapeutic levels 2 hours after a single dose and then progressively decreased to baseline by 24 hours. Despite higher plasma levels of PPA with more frequent dosage, urethral pressure profiles were not significantly different. The reasons for clinical efficacy is not clearly understood, but it may be possible that certain individuals can be managed with once daily dosing which is more convenient and cost effective for owners.

  In South Africa PPA can be used in the form of a cold and flu mixture sold as Rinex®. Rinex® contains phenylpropanolamine hydrochloride, chlorpheniramine maleate (an antihistamine) and phenylephrine hydrochloride (another alpha-adrenergic agonist). The drug is available in capsules (30mg PPA per capsule), syrup (1mg PPA per ml) and paediatric syrup (0.5 mg/ml). Most patients have some kind of response to this drug. The dosage may need to be increased over time as the drug does seem to become less effective over time. Side effects may include restlessness, hypertension, aggressiveness, changes in sleeping patterns and gastrointestinal side effects. These can usually be limited by decreasing the dose.

  Contraindications for the use of PPA include pre-existing cardiac disease, chronic kidney disease, protein losing nephropathy, hyperthyroidism and hypertension. Although hypertension does not seem to develop to a clinically significant degree in dogs receiving PPA it is recommended that blood pressure is monitored regularly. In many cases PPA is combined with oestrogen drugs and there seems to be a synergistic effect when these two drugs are used together.

  PPA is also usually prescribed in incontinent male dogs. Some clinicians combine PPA with testosterone, but efficacy of this approach has not been well documented. Approximately 43% of male dogs are responsive to PPA, much lower than what is seen in female dogs.

- **Phenylephrine**
  This drug is an alpha-adrenergic agonist which is used to treat hypotension. It is usually used as a constant rate infusion to improve peripheral vascular resistance and increase mean arterial pressure. The drug is compounded and sold as an oral solution to treat USMI in incontinent dogs. In South Africa it is compounded by Kyron laboratories as a syrup which contains 20 mg/ml. The recommended dose is 1.2 mg/kg three times daily. As with PPA, it is contraindicated in cases with cardiac disease, renal disease or hypertension. It is advisable to monitor blood pressure regularly.

Oestrogens

Oestrogens can be administered less frequently than PPA which may be more convenient for most owners. However, with some formulations response rate is lower than with PPA and only 60-65% of patients respond to treatment with diethylstilboesterol. For the newer oestriol formulation a response rate of up to 80% or higher was reported.

Oestrogens increase the sensitivity of the urethral alpha-adrenergic receptors to catecholamines and possibly also increase the number of alpha-adrenergic receptors. Diethylstilboesterol (DES) and oestriol are the most commonly used oestrogens.

Side effects of oestrogen therapy could include signs of oestrus such as mammary gland enlargement, vulvar swelling and attractiveness to intact males; perineal alopecia and bone marrow suppression. In most cases bone marrow suppression is not a clinically significant complication unless dosages of around 10 times the recommended doses are used and it is not listed as a side effect for oestriol. It is recommended that as a precaution a complete blood count should be performed prior to treatment and repeated a month after initiating treatment and again at 3 months and 5 months. Its use is contraindicated in intact female dogs and the drug should be used with extreme caution in male dogs as it could result in prostatic metaplasia.

- **Diethylstilboesterol**
  DES is compounded by compounding pharmacies such as V-Tech. DES is dosed at 0.1-1 mg (0.02 mg/kg) per dog perios for 3-5 days and then every 3-7 days depending on the response. Once a week dosing may be more convenient for some owners. After two months the dosing frequency can be decreased to once every 10 days, and in some cases even to every 14 days. The formulations available from V-Tech in South Africa include 30

mg capsules and a syrup which contains 1mg/ml.

- **Oestriol**
  Oestriol (Incurin®) is a naturally occurring short acting oestrogen which is registered for use in dogs. Each tablet contains 1 mg oestriol. It is dosed at 1 tablet per dog per day for 1-2 weeks after which the dose is reduced to the lowest controlling/effective daily dose, usually ½ tablet per dog per day. After this stage, alternate day dosing can be implemented. Dose is not related to body weight and needs to be adjusted for the individual animal. In cases that do not respond to 1 tablet per day the dose can be doubled to 2 tablets per dog per day. If this regimen still has no effect on the incontinence the diagnosis should be reconsidered and a more intensive diagnostic approach should be taken.

- **Conjugated oestrogens**
  Premarin® tablets (0.3 mg, 0.625 mg, 1.25 mg) is a preparation used in postmenopausal women. It is derived from pregnant mare urine and contains conjugated oestrogens which are converted to oestradiol. This drug can be dosed at 20µg/kg *per os* every 3 or 4 days.

Testosterone analogues

- **Testosterone cypionate**
  Testosterone injections have anecdotally been reported to improve incontinence in male dogs. It is given at 1.5 mg/kg every 4 weeks. The exact mechanisms which result in an effect are unclear, but could involve increased urethral smooth muscle tone, improved tone in urethral supporting structures and increased prostatic urethral resistance. It is reported to be less effective than PPA. Potential side effects in male dogs include development of perianal adenomas and prostatic enlargement. The use of testosterone in female dogs cannot be recommended as its use may lead to clitoral hypertrophy, masculinisation and aggression. It is also contraindicated in dogs with renal disease, cardiac disease and hepatic insufficiency.

- **Gonadotropin releasing hormones**
  Although not often recommended and not commonly used, gonadotropin releasing hormone (GnRH) analogues may be effective in some cases of USMI. GnRH analogues decrease follicle stimulating hormone (FSH) and luteinizing hormone (LH) by down regulating gonadotropin receptors in the pituitary gland. Because spaying causes a decrease in oestrogens and a subsequent increase in FSH and LH, GnRH may have a role to play in the management of post ovariohysterectomy USMI. This medication will need further investigation as analysis to date showed that FSH and LH in spayed incontinent dogs were actually lower than FSH and LH in spayed continent dogs. GnRH analogues had no effect on maximum urethral closing pressure and urethral pressure profiles compared to PPA and oestrogens.

  In a small group of dogs, a response rate of (7/11 dogs) 60% was seen when using leuprolide. The fact that a response is seen clinically when using the drug lead to the speculation than GnRH may have a direct effect on the bladder which causes detrusor muscle relaxation. The GnRH analogue, leuprolide (Lupron®) can be used at 5-10 mg intramuscularly every 3 months. This drug may be useful in cases where oetrogens and PPA are contraindicated.

Tricyclic antidepressants

- **Imipramine**
  Imipramine (Tofranil®) is a norepinephrine reuptake inhibitor and potential anticholinergic. It is available in 10 mg, 25 mg and 50 mg tablets. It indirectly increases alpha-receptor stimulation in urethral smooth muscle and may lead to an increase in bladder relaxation. It is dosed at 5-15 mg/dog (½ to 1 ½ of the 10mg tablets) *per os* every 12 hours. It is not commonly used in the treatment of PSMI. Side effects include vomiting, diarrhoea, hyperexcitability, seizures and sedation. It decreases the seizure threshold and should not be used in epileptic animals.

Interventional procedures for USMI

A complete discussion on these procedures are beyond the scope of this text as this paper describes the medical management of USMI. However, it is worth mentioning the surgical options available for patients in South Africa. Various surgical specialists can be contacted regarding these procedures should a patient require surgical intervention.

- **Urethral bulking**
  This procedure involves the injection of bulking agents such as bovine crosslinked collagen submucosally into the proximal urethra during cystoscopy. By bulking up the tissues an increase in resting urethral pressure is achieved. There is a variable response rate (50-80%) and duration of effect with response periods varying from 8 months to 2 years. This procedure can be combined with medical management and renders PPA more effective.

See the article on *Surgical Procedures for Urethral Sphincter Mechanism Incompetence in Bitches* on page 30-34 in this edition

References on www.vetlink.vet360.co.za

Surgical Procedures for Urethral Sphincter Mechanism Incompetence in Bitches

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Acquired urethral sphincter mechanism incompetence (USMI) is the most common storage disorder in neutered female dogs[1]. The urethral sphincter mechanism involves the smooth muscle of the urethra as well as the surrounding support tissues, sub-mucosal vasculature, and urothelium[7].

Female dogs have no true bladder neck sphincter and urinary continence is maintained by a complex mechanism of interacting factors. Urethral sphincter mechanism incompetence (USMI) is the most common diagnosis made in adult female dogs and is second only to ureteral ectopia in juvenile dogs, especially in females[1-3]. USMI in male dogs in uncommon, but if present it may be associated with gross prostatic or pelvic urethral deformities such as prostatic urethral diverticulae[4].

Normal control of urinary continence

During filling of the urinary bladder, urethral resistance is maintained by a complex mechanism of factors. These include tone in the urethral smooth muscle (the internal bladder neck sphincter), tone in the urethral striated muscle (the external sphincter or ischio-urethralis muscle), natural elasticity of the urethral wall tissues (not only musculature), physical properties of the urethra (length and diameter of the urethra) and the degree of engorgement of the sub-urethral venous plexuses[7]. In a variety of species, including dogs, direct sympathetic and parasympathetic interactions occur in the urethra and bladder during storing of urine and during voiding.

Urethral tone

Poor urethral tone is implicated in urinary incontinence based on investigations of urethral pressure profilometry. Urethral tone is maintained by a complex interaction of neuromuscular, vascular, and passive elastic components; it is unclear which of these is deficient in female dogs with urethral sphincter mechanism incompetence[1].

Urethral length

Urethral length varies considerably among female dogs of different sizes. When body size is taken into consideration, then bitches with urethral sphincter mechanism incompetence tend to have shorter urethras than continent animals[1, 2].

Bladder neck position

Radiographic finding of a "pelvic bladder" and a more caudal positioning of the bladder neck when a bitch moves from a standing to a relaxed recumbent position is more pronounced in bitches with bladder sphincter mechanism incompetence than in normal animals[2, 6]. *Figure 1 & 2.* There may be a deficiency in supporting mechanisms in the lower urinary tract of affected animals. The role of the bladder neck position in bitches with USMI is thought to be from changes in transference of abdominal pressure to the urethra[6].

SURGICAL OPTIONS

The main objective of surgical treatment is to increase the urethral outflow resistance or the urethral length or to relocate the intra-pelvic bladder to a more intra-abdominal position.

Options for increasing urethral outflow resistance include peri-urethral slings, artificial sphincters, or intra-urethral injection of collagen bulking agents with the use of an urethro-cystoscopy technique[8]. The urethral length can be increased with bladder neck reconstruction techniques. The bladder neck can be relocated to a more intra-

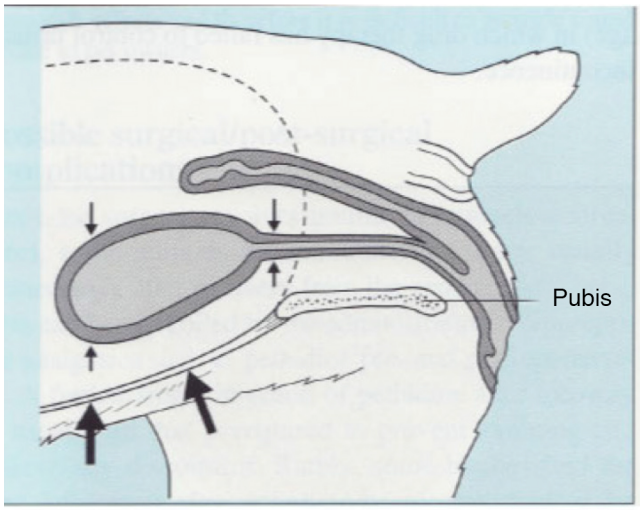

Figure 1. If the bladder and the proximal urethra lies intra-abdominally, then any increased abdominal pressure will cause equal pressure on the bladder wall as well as on the urethra.

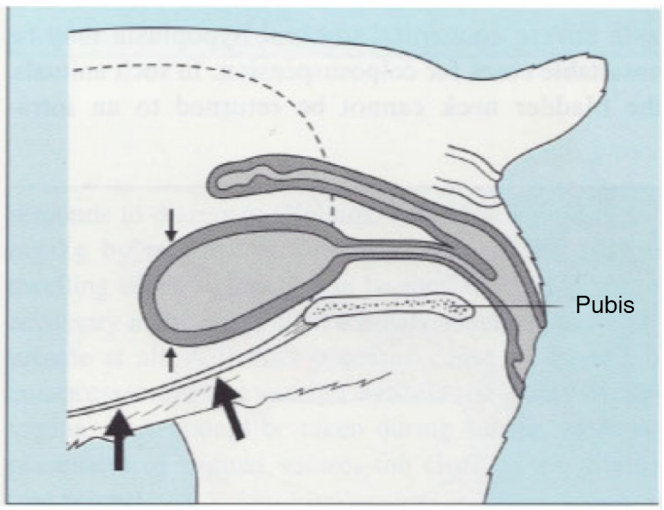

Figure 2. If the bladder is located intra-pelvic, the abdominal pressure is only active on the cranial bladder wall and forces urine out of the short urethra, making the dog incontinent.

abdominal position by means of a colposuspension or with an urethropexy [9-11]. Colposuspension provides firmer anchorage of the lower urogenital tract than urethropexy and it also avoids urethral trauma[11].

1. Colposuspension

This moves the bladder neck cranially and relocates the bladder in a more intra-abdominal position to improve transference of abdominal pressures to the bladder neck and proximal urethra [2, 3].

This operation was developed for woman with stress incontinence and is used in bitches with USMI since 1985[2]. The vagina on either side of the urethra is anchored to the pre-pubic tendon with non-absorbable monofilament nylon sutures [2, 3].

Surgical technique

The bitch is placed in dorsal recumbency and an 8FG Foley catheter (smaller bitch) or 10FG (larger bitch) is inserted through the urethra into the bladder. The cuff is inflated and the catheter gently withdrawn until its cuff rests in the bladder neck. The catheter facilitates identification of the urethra and bladder neck during surgery.

A caudal, midline abdominal approach just cranial to the pubis, is made and the linea alba is incised caudally to the level of the pelvic brim. The pre-pubic tendons and external pudendal vessels are identified so they can be avoided.

Cranial traction on the bladder with a stay suture allows the bladder neck with the inflated Foley catheter cuff to be identified. The retroperitoneal fat around the bladder neck and urethra is gently rubbed off with a swab twisted around a straight Spencer Welch artery forceps. (Fig 3 a, Fig 3 b)

The vagina is displaced cranially with a gloved finger inserted into the vagina (large bitch) or a well-lubricated test tube or Poole suction tip (smaller bitch)[11]. The urethra is palpated through the ventral vaginal wall and displaced to the left side of the patient while the vaginal wall over the fingertip to the right side of the urethra is pushed cranially towards the caudal aspect of the linea alba incision. The exposed vaginal wall is now grasped with an Adson tissue forceps

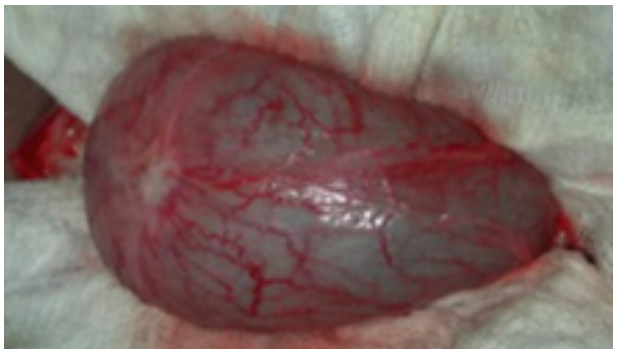

Figure 3a. Bladder position before traction cranially

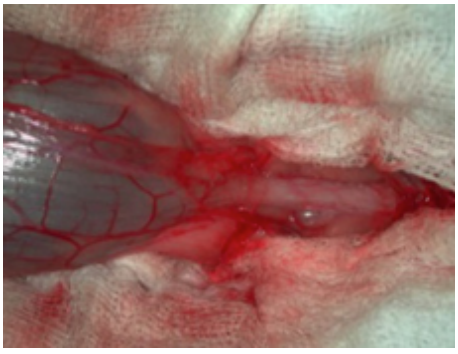

Figure 3b. Bladder position after traction

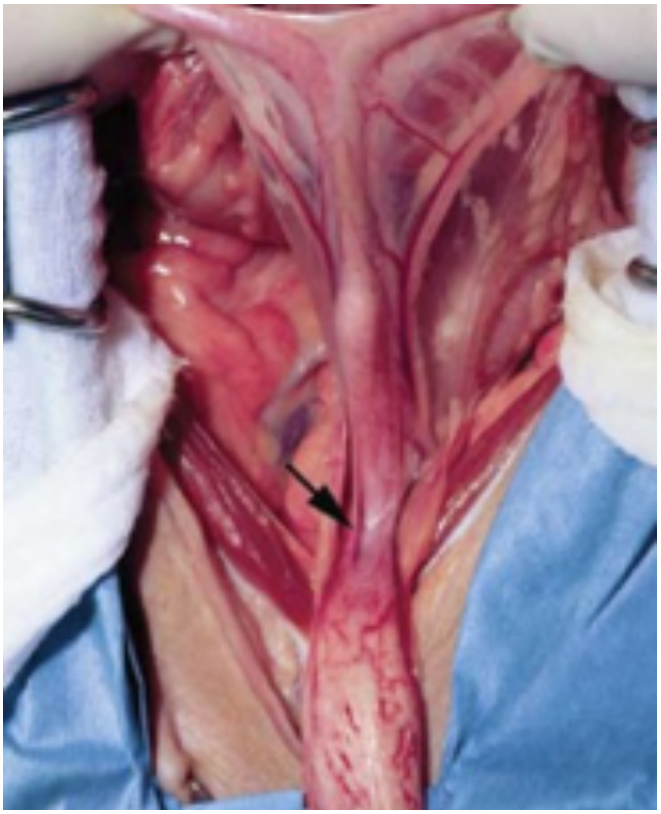

Figure 4. The right lateral aspect of the vagina is exposed (arrow) underneath the urethra.

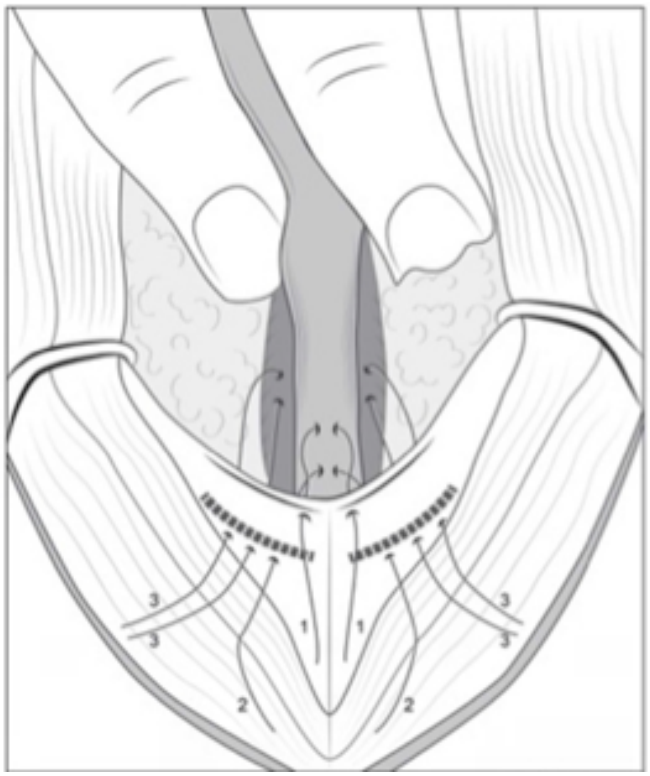

Figure 5 - Colposuspension with nylon sutures through the lateral vaginal wall to form a vaginal sling that is pressing the urethra against the pubis

and pulled cranially. *(Figure 4)* The most lateral part of the right side vagina is sutured to the pre-pubic tendon approximately 1.0 to 1.5 cm away from the midline with 0/0 monofilament nylon. Two to three sutures are placed on the right and the left sides through the lateral vaginal wall and through the pre-pubic tendons on either side of the urethra so that the knots are on the outside of the rectus sheath[10, 11]. The nylon sutures may enter the vaginal lumen during this procedure; therefore the vagina must be aseptically flushed out with diluted aqueous povidone-iodine solution during preparation of the patient.

When all the nylon sutures have been pre-placed, a curved Spencer Welch artery forceps or Kilner needle holder is placed ventrally between the ventral aspect of the urethra and the pubic symphysis to ensure sufficient space between the urethra and the pubic bone when these vaginal sling sutures are tightened[10, 11]. *(Figure 5)*

The urethra must be evaluated all the time as the left and right side nylon sutures are tightened to prevent compression of the urethra against the cranial aspect of the pubis, because the animal may develop postoperative dysuria. If the urethral position is adequate, the cuff of the Foley catheter is deflated and the catheter is removed. The ventral abdomen is closed routinely.

2. Urethropexy

Urethropexy is an alternative to colposuspension that is aimed at restoring the bladder neck and proximal urethra to an intra-abdominal position while simultaneously increasing resistance to urine flow by reducing the diameter of the urethral lumen. The urethra is approached through a caudal midline coeliotomy incision that extends to the pubic brim. The bladder is retracted cranially and the ventral aspect of the urethra is exposed at the level of the cranial pubic brim.

With the bladder neck and urethra retracted cranially, 2/0 or 0/0 polypropylene (Prolene®) sutures are pre–placed: they should enter the abdominal cavity, passing full thickness through the ventral abdominal wall, including the rectus fascia, they should then pass through the seromuscular layer of the urethra in a horizontal mattress pattern at either the nine or three o'clock position in the transverse section without penetrating the urethral lumen.

The sutures then exit from the abdominal cavity through the abdominal wall, including the rectus fascia, on the same side. The two most caudal sutures on either side of the urethra are engaged through the prepubic tendon as they enter and exit the abdomen. After the bladder has been replaced into the abdomen tighten and tie the pre-placed sutures from caudal to cranial on each side of the urethra. The abdomen is closed routinely.

3. Bladder sling urethroplasty

The bladder and urethra is approached through a caudal midline coeliotomy incision that extends to the pubic

brim. The bladder is retracted cranially and the ventral aspect of the urethra is exposed to the level of the cranial pubic brim. Stay sutures are placed on either side of the ventral bladder neck and urethra before a ventral cystourethrotomy is performed in the ventral midline.

Two full-thickness bladder wall slings are harvested from the left and right sides of the urethral wall. Stay sutures are placed in the cranial ends of each bladder sling. A 10 FG Foley catheter is inserted into the bladder with its cuff in the bladder lumen before the narrowed urethral wall is sutured closed over the Foley catheter that is used as a stent. *(Figure 6)*

Once the entire urethra and the neck of the bladder have been sutured closed, the two urethral wall slings are pulled underneath the exposed urethra to form a figure-of-eight pattern around the urethra *(Figure 7)*. The cranial end of each sling is finally sutured to the ventral bladder neck area to maintain them in this position *(Figure 8)*. The cuff of the Foley catheter is inflated and gently pulled caudally until the cuff lies in the bladder neck. All the stay sutures are removed and the ventral abdominal wall is closed routinely.

The Foley catheter is secured to the tail of the bitch and maintained in place for four to six days to allow resolution of postoperative urethral swelling. A NSAID is administered for the first 5 days while the dog is hospitalised. No antibiotics are given while the indwelling catheter is in place. After 4 to 6 days the Foley's catheter cuff is deflated and the catheter removed. The tip of the catheter can be used for bacterial culture and antibiotic sensitivity testing if cystitis is expected.

Most dogs will have some dysuria for a day or two due to increased outflow resistance. Bethane col (Urecholin®) with diazepam (Valium®) can be administered for 7 to 10 days to alleviate this. The owner must carefully observe if the bitch is able to urinate with a stream or if the animal is showing pollakiuria with only small volumes of urine during voiding. In 53 dogs operated by myself with this sling urethroplasty technique over the past decade, 45 animals did not need any additional phenylpropanolamine or estradiol to maintain urinary continence.

4. Polypropylene tape used as an artificial urethral sling

The first work was published by Stephanie Claeys et al. (2010) from Belgium[12]. The trans-obturator vaginal tape inside out (TVT-O) has later been used in 7 incontinent female dogs with this polypropylene tape that is actually used for stress incontinence in women and was found fairly easy to install with good clinical effect[13].

A median episiotomy is first performed to reach the urethra and to install the polypropylene tape around the urethra after an incision has been made into the floor of the vagina just proximal to the external urethral meatus. The blue polypropylene tape is covered in a thin plastic

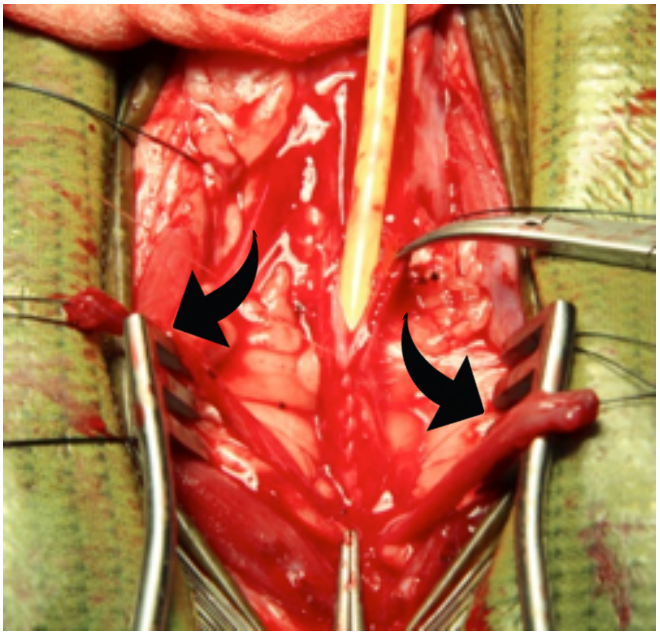

Figure 6. Two full-thickness urethral wall slings with stay sutures in their cranial ends are retracted sideways () while the narrowed urethra is sutured closed over the Foley® catheter with 4/0 PDS in a simple continuous suture pattern. (The patient's head is on top of the photo)

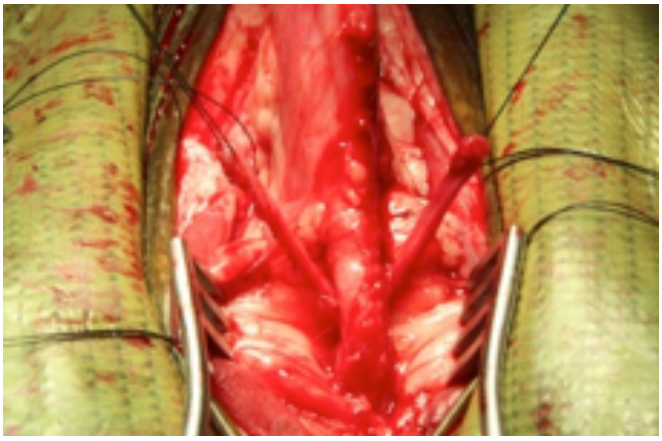

Figure 7. Two stay sutures maintain the two urethral slings that are forming a figure-of-eight pattern around the urethra that has been sutured closed with 4/0 PDS. The cranial ends of the slings will be secured to the ventral bladder neck area with 4/0 PDS simple interrupted sutures.

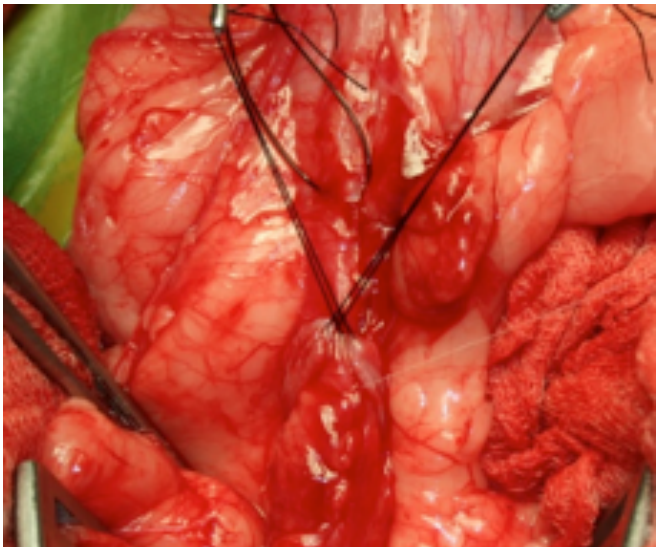

Figure 8. The cranial ends of the slings are secured to the ventral bladder neck area with 4/0 PDS simple interrupted sutures.

sleeve to assist with its passing through the two obturator foramina to form a sling across the dorsal aspect of the distal urethra when the animal is standing. The plastic sleeve is removed before closure of the episiotomy.

The article by Claeys *et al*. (2010) describes the results in female dogs with a short-term cure in 6 of the 7 cases[13]. This polypropylene tape that can now be used as an artificial urethral sling, and can be obtained from Price Medica (orders@pricemedica.co.za). Animals treated with the TVT-O by Dr Hans van der Zee in our Small Animal Surgery Section performed well over the first 12 months.

5. Artificial sphincters

Hydraulic urethral occluders are being used with increasing frequency as artificial sphincters for the treatment of USMI in female dogs[14-16]. The artificial canine urethral sphincter (DOCXS Biomedical Products & Accessories, Ukiah, California) consists of a medical grade silicon cuff and tubing and a subcutaneous access port. Cuff sizes are available in lumen diameters ranging from 6 mm to 16 mm and width from 11 mm and 14 mm. The size selection is dependent on the measurement of the urethral circumference at the location of placement (which should be at least 2 cm caudal to the neck of the bladder. Before placement, the cuff should be tested to verify that it is functional. The cuff, tubing and port should be flushed with sterile saline to remove any air bubbles. The urethra is dissected circumferentially at the placement site and its circumference is measured with a Penrose drain.

The un-inflated sphincter cuff is placed around the urethra and secured to itself with non-absorbable monofilament nylon sutures. The associated tubing is tunnelled through the body wall and exited into the subcutaneous space of the caudal abdomen or inguinal region, and secured to the port with a boot fastener[16]. The port is then secured to the external abdominal wall fascia with monofilament nylon sutures. The bladder should be manually expressed before abdominal closure to verify that the urethra is not obstructed with the occluder in place[16].

Cuff inflation is delayed for up to 6 weeks to allow resolution of inflammation and to permit revascularization of the dissected portion of the urethra. If the patient remains incontinent after artificial sphincter placement, then the cuff is typically inflated about 25% by percutaneous injection into the port with sterile saline through a 22G Huber needle. In an initial report, three out of four dogs required inflation of the cuff one or more times after sphincter placement[15]. The fourth dog required port removal because of superficial ulceration, but all four dogs were incontinent without medical management 26 to 30 months after artificial sphincter placement[15].

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By Lilian Cornejo, DVM, DACVIM
CVC IN SAN DIEGO PROCEEDINGS

Hepatic Lipidosis
Maximising a Successful Outcome

Feline hepatic lipidosis is the most commonly encountered liver disease in cats, and results from accumulation of fat within the majority of hepatocytes.

The end result is severe intrahepatic cholestasis which leads to a rapid decline in liver function. Many affected cats are middle-aged or older. Overweight cats that become anorectic are particularly at risk. Stressful events or another systemic disease that causes the cat to stop eating are common precipitating factors. Studies have documented a concurrent or inciting disease in 50-95% of cases. Common concurrent illnesses include pancreatitis, cholangiohepatitis, inflammatory bowel disease, extrahepatic bile duct obstruction, and neoplasia. However, any illness or event that causes a prolonged lack of food intake can put cats at risk for developing HL, even those that are not overweight.

The exact cause of HL is unknown. Catabolism due to inadequate caloric intake appears to play a central role in the development of the disease. Factors theorised

to be involved in the pathogenesis include:
1) increased breakdown of peripheral fat stores with resultant increased fatty acid delivery to the liver. As a result, oxidation of fatty acids in the liver by mitochondria is overwhelmed (i.e., decreased oxidation of fatty acids; leads to lipid accumulation),
2) impaired removal of triglycerides (as VLDL) from the liver.

History and physical exam

Patients typically present with prolonged anorexia, gastrointestinal signs, weight loss (often > 25% of body weight), and lethargy. Icterus is frequently present on initial examination (> 70% of cases). Other findings include hepatomegaly, dehydration, and muscle wasting.

Preliminary diagnostics

Initial CBC may be normal or reveal a mild non-regenerative anemia (22% of cases) and poikilocytosis. A declining haematocrit is a common finding during treatment and is influenced by a number of factors including repeated blood draws, haemolysis (due to Heinz bodies or severe hypophosphaetemia), and/or blood loss. Care should be taken to limit the amount drawn during daily blood sampling to the absolute minimal amount needed to run the desired test, to avoid exacerbation of anaemia. Approximately 25 to 40% of HL cats will require blood products during hospitalisation.

Biochemical abnormalities include moderate increases in ALP, mild to moderate increases in ALT, and increases in AST. In contrast to other feline hepatobiliary diseases where GGT is typically increased, GGT is often normal or only mildly elevated in patients with hepatic lipidosis. Hypokalaemia (30%), hypophosphataemia (17%), and hypomagnesaemia (28%) may develop during the course of the disease, particularly during the refeeding period. When present, hypoalbuminaemia, hyperammonaemia, and hypoglycaemia, are indicators of severe hepatic dysfunction.

In a study of 22 cats with naturally occurring liver disease, coagulation abnormalities were found in 82% of cats. Vitamin K deficiency was the most common abnormality detected. Coagulopathy in HL cats can result from deficiency of active vitamin K (due to cholestasis) and from reduced production of clotting factors. Routine coagulation testing such as PT and PTT are appropriate, but proteins-induced-by-vitamin K absence (PIVKA) may be a more sensitive test to detect vitamin K dependent coagulopathies.

On abdominal ultrasonography the liver is diffusely hyperechoic and may also be enlarged. Ultrasound eamination also assists with documentation of concurrent diseases such as pancreatitis or other intra-abdominal conditions that may have led to the anorexia. All HL patients should be screened for the presence of concurrent or underlying diseases. One study found a decreased recovery rate in HL cats that had concurrent pancreatitis compared to those in whom HL was the only diagnosis.

Diagnosis

Definitive diagnosis of HL is established by histologic examination of the liver, which reveals severe intrahepatic lipid accumulation. Hepatic lipidosis may be suggested on fine-needle aspiration of the liver, and many clinicians prefer this test during the initial phase of treatment to support a presumptive diagnosis of HL, and may postpone biopsy of the liver until the hospitalised patient is more stable.

Fine-needle aspiration of the liver can easily reach a diagnosis of HL by demonstration of a predominance of heavily vacuolated hepatocytes. It has the advantage of being much less invasive and may be a more appropriate means of arriving at a preliminary diagnosis during the early phase of treatment in the fragile liver cat. However, it has significant drawbacks, in that it may miss concurrent hepatic disorders and may on occasion incorrectly classified as primary.

EDITORS NOTE: Liver FNA may show lipid accumulation in the hepatocytes of normal cats.

Liver biopsy is required for definitive diagnosis and to rule out other concurrent hepatic conditions, and is usually obtained with ultrasound-guidance. Because of the high lipid content, biopsy specimens of HL cats may float when placed in formalin. Obtaining a biopsy is particularly important if suspicion for another primary hepatopathy exists. Aerobic and anaerobic bile or liver cultures are also obtained at this time.

Histologic examination of biopsy specimens demonstrates severe vacuolisation in the majority of hepatocytes. Inflammation and necrosis are absent unless another concurrent hepatic disorder is present. Vitamin^{K1} (0.5 mg/kg SC q 12h) is administered in an attempt to improve coagulation abnormalities prior to liver biopsy. This may resolve abnormalities resulting from vitamin K_1 deficiency.

All clotting factor deficiencies will not correct with this therapy. Therefore if coagulation parameters remain abnormal, fresh frozen plasma or fresh whole blood may be required before liver biopsy to provide necessary clotting factors. Risk of complications is also increased if the platelet count is below 80,000. Haemorrhagic complications from liver biopsy can be reduced to 10% or less by careful patient selection. Most post-biopsy bleeding complications occur within the first 5 hours after biopsy but can be delayed up to 18 hours. Cats should be closely monitored for significant post-biopsy bleeding during this time.

The goals in treatment of the HL cat are to:

1. provide nutritional support with adequate calorie and protein intake,
2. correct fluid and electrolyte imbalances,
3. monitor for and manage complications of liver disease (coagulopathy, nausea, refeeding syndrome, HE, etc.), and
4. identify and treat any underlying conditions.

Treatment

Treatment centers around aggressive nutritional support and the management of concurrent or underlying conditions. Early diagnosis and nutritional intervention improve chances for a successful outcome. Failure to treat concurrent disorders may hinder the clinician's attempts at reversing lipidosis.

Initial treatment involves correcting fluid and electrolyte deficits using an intravenous balanced electrolyte solution supplemented with 20 mEq KC/L. Hypokaelemia is common, and if identified, will require more aggressive supplementation and monitoring. Many clinicians, including the author, avoid lactate-containing fluids (such as LRS) because of the concern for decreased metabolism of lactate that may accompany severe hepatic dysfunction. Further studies are needed to determine if lactate-containing fluids pose a true threat to our feline liver patients. Unless hypoglycaemia is documented, avoid supplementing intravenous fluids with dextrose as this may encourage further hepatic TG accumulation.

Vitamin depletion may occur in HL patients. B-Complex vitamins are therefore often added to fluids at 1-2 ml/L to prevent deficits in thiamine and other water-soluble vitamins. Cobalamin (Vitamin B12) deficiency has been documented in 40% of HL cases, and deficiency can be determined in many with a fasting cobalamin level. A single dose of 250 ug SC cyanocobalamin SC can be administered while awaiting results. If reduced vitamin B 12 concentrations are documented, ongoing supplementation may be required.

In critical patients, anaesthesia can further decompensate the patient and may need to be postponed. A temporary nasogastric tube is an excellent route by which to initiate nutritional support (using liquid enteral diets) in these patients. This can be placed in the awake patient after applying a small amount of topical anaesthetic into the nostril (avoid using large amounts which can drain down to and numb the larynx).

Once the patient is more stable (1-2 days), anaesthesia can be performed to place a more permanent and wider diameter lumen feeding tube (e.g., 12 Fr oesophagostomy tube, 18-20 Fr gastrostomy tube). This can often be accomplished at the time the patient is anaesthetised for an ultrasound-guided liver biopsy. Because of the fragile nature of these patients post anaesthesia, surgery is avoided unless indicated for the management of an underlying condition.

Nutrition

In order to ensure adequate energy and nutrient intake, feeding must occur through a feeding tube. The use of appetite stimulants and palatability enhancements of foods are usually insufficient to promote sufficient intake by the HL cat, and therefore risk delaying appropriate therapy which may in turn decrease chances for recovery.

Relying on force-feeding and appetite stimulants is discouraged.

The nutritional target is to provide the cat with its maintenance energy needs by providing a complete and balanced diet that is protein rich (~30-40%), contains a moderate amount of lipids (~ 50%), and is low in carbohydrate (~20%). The formula 1.1 x (30 x kg body weight) + 70 provides a good initial estimate of total caloric need. Recovery diets are are commonly used and fulfill these criteria and can be easily blenderised with some water for use through a tube.

Protein must not be restricted, unless overt hepatic encephalopathy is noted. Feedings are divided over 4-6 meals per day, and one third of the daily requirement is fed on day one. The amount is gradually increased to the full daily requirement (by another 1/3 each day) over the next 2-3 days. At the same time IV fluid rates are gradually reduced by the amount of fluid now being provided through the feeding tube. At this point the daily feeding amount can be gradually switched to q8h feedings, which is easier for owners to manage at home.

Additional supplements

It is unknown whether supplementation with taurine, carnitine or arginine hastens recovery. L-carnitine is a cofactor that permits conversion of fatty acids into energy via of beta oxidation. A 1990 AJVR study concluded that carnitine deficiency does not contribute to the pathogenesis of HL. An anecdotal report of improved survival in another group of cats with severe HL supports the need for further study. A dose of 250 to 500 mg of medical grade carnitine is used when supplementing cats. Taurine is required for bile acid conjugation and levels may be reduced in some HL cats. Supplementation can be provided at 200-250 mg PO q 24h for the first 7 to 10 days of treatment.

Glutathione is an important antioxidant within the liver and depletion is common in HL cats. Stores can be replenished by providing thiol donor substrates (N-acetylcysteine, S-adenosylmethionine [SAMe]). SAMe is enteric-coated and should therefore not be crushed and given through the tube.

***Editor note:** maropitant (Cerenia) is very effective for nausea in cats. Dose at 1 mg/kg OID

Complications

Complications are commonly encountered during the first three days of refeeding and patients must be closely monitored during this time. Vomiting is common in HL cats that are being refed and can result from severe hepatic dysfunction and reduced stomach volume.

Metoclopramide (1-2 mg/kg/day as a CRI) has antiemetic and prokinetic actions and should be used if vomiting develops*. Additional antiemetics may need to be added if nausea persists (e.g., dolasetron 0.6-1.0 mg/kg IV q24h). If vomiting does not subside, consider looking for other contributing conditions and/or switching from bolus to CRI ('trickle') feedings.

With refractory nausea, temporarily switching to total parenteral nutrition may be necessary. Hypokalaemia and hypophosphataemia may exacerbate GI signs and can cause weakness. Additionally, phosphorus levels below 1.5 mg/dl can induce hemolysis. Phosphorus should be supplemented if levels fall below 2.0 mg/dl (potassium phosphate at 0.01-0.03 mmol/kg/hr). Phosphorus levels should be monitored every 6 to 12 hours during supplementation. Reduce KCL in the IV fluids by the amount of potassium provided in the phosphate infusion. Hypokalaemia carries a negative survival risk and decreased levels should therefore not be ignored.

Hepatic encephalopathy (HE) is managed with antibiotics and lactulose to limit ammonia production and NH_3 diffusion across the blood brain barrier. Stuporous patients require more aggressive measures. Protein restriction may be needed in severe cases of HE. Monitor HL patients for risk factors that can worsen or precipitate HE which include hypokalaemia, alkalosis, gastrointestinal bleeding, constipation, infection, hypoglycaemia, and azotaemia. Overtranquilization and stored blood products (increased ammonia content) can also exacerbate HE.

Hypotension may develop in some patients following anaesthesia and blood pressures should be measured in patients who appear more depressed or have a slow recovery. Hepatic encephalopathy is another differential that can cause similar symptoms. Persistent hypotension should trigger aggressive supportive measures (IV crystalloids, +/- colloids, +/- pressors in fluid-replete patients) and a search for other contributing causes (e.g., sepsis).

Vital signs are closely monitored. A PCV/TP, blood glucose, and azostrip are checked every day. Evaluation of electrolytes may be required one or more times each day and should be tailored to the individual patient. A sudden dramatic decline in PCV should prompt investigations for hypophosphatemia and blood loss.

Long-term care

Once patients have reached their full caloric needs, are stable, and vomiting is controlled, care can be transitioned to the owner at home. Owners must be given detailed instructions on feeding tube care, how to detect tube site infections, and trouble-shooting when problems occur (e.g., tube clogging). Cats often require tube feedings for 3-8 weeks. Fresh food is offered each day throughout this period to encourage the patient to resume oral feeding. Once cats start consuming food orally, tube feedings can be gradually reduced each week as long as their weight remains stable during this time. The tube can be removed once the cat is completely off all supplemental feedings for at least one week and consuming a sufficient amount of food to maintain its weight during that 'tube-feeding-free' time.

Outcome

Survival rates in HL cats have been reported to range from 60-88%. Early aggressive treatment combined with diligent monitoring and supportive care offer the best chance at a successful outcome. Higher median potassium and hematocrit levels as well as young age were more favorably associated with survival in one study. Bilirubin levels often decline by 50% in the first 7-10 days in responders. Liver enzymes decline more slowly. Those with a poor prognosis often succumb during the first week of treatment. A lower survival rate has been reported with concurrent pancreatitis. Residual hepatic dysfunction does not persist following complete recovery and recurrence is unlikely unless the precipitating cause was not corrected.

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