INTRODUCTION

Dramatic changes have occurred in the past 20 years regarding the way veterinarians view vaccines and vaccination practices. The concepts of core and non-core vaccines, disease risk assessment, extended inter-vaccination intervals, and using products that minimize vaccine-associated inflammation for cats are currently mainstream veterinary medicine. There are now a number of well-referenced publications that document the long duration of immunity provided by most feline viral vaccines. The fourth revision of the American Association of Feline Practitioners (AAFP) Vaccination Guidelines for cats was published in late 2013. The World Small Animal Veterinary Association Guidelines were published in 2010 and updated in 2015. The European Advisory Board on Cat Diseases (ABCD) guidelines were published guidelines in 2010 and a companion document on Injection Site Sarcomas was published in 2015. These are thorough documents that are exhaustively referenced and are an outstanding resource for veterinary practitioners as they change vaccination practices.


VACCINE-ASSOCIATED SARCOMAS - HISTORICAL BACKGROUND

In addition to recognizing the long duration of immunity of feline vaccines, the occurrence of vaccine-associated sarcomas (VAS) is another reason why the AAFP developed their first feline vaccination guidelines in 1986. The underlying cause for all injection-site tumors is chronic (months, years) injection site inflammation. Vaccine-associated sarcomas are connective tissue tumors occurring at vaccine injection sites. The link between chronic inflammation and the induction of neoplasia has been recognized for many years in human medicine and has been reported in association with many causes of chronic inflammation, not just injections. (Okada 2014) Similarly in veterinary medicine, neoplasia has been previously reported in association with, but not limited to, ocular trauma in cats because of inflammation caused by leakage of lens proteins, metallic implants used in fracture repair which cause low grade electrolysis and chronic site inflammation, metallic implants used in TPLO surgeries, and chronic parasitic infections (S. lupi).

The occurrence of neoplastic transformation at the site of vaccine injections is of relatively recent origin in cats. Since publication of Dr. Mattie Hendrick’s original review recognizing this problem in 1991 there have been many articles documenting the association between vaccine administration and neoplasia at vaccine injection sites. As early as 1999, the World Health Organization classified veterinary vaccine adjuvant as a class III/IV carcinogen on the basis of their review of the literature on VAS in cats. Vaccine-associated neoplasia is not limited to cats and has also been reported in dogs, ferrets, rabbits, and most recently, in horses.
Why is injection site neoplasia a relatively new problem for our cats? Weren’t we administering vaccines and giving many other injections to cats prior to the mid-1980’s? The answer is that there was a major change in the types of vaccines being given to cats in the mid-1980’s. Prior to that time, the only vaccines routinely given to cats were modified live (non-adjuvanted) FVRCP and MLV (non-adjuvanted) Rabies vaccines. In the mid-1980’s all MLV rabies vaccines were removed from the market because there was a 1:500,000 incidence of vaccine-induced rabies and this was deadly for pets and also a human health hazard. MLV rabies vaccines were replaced with killed, adjuvanted rabies vaccines. At the same time, killed, adjuvanted FeLV vaccines were introduced for cats as was killed, adjuvanted FVRCP vaccine. In addition, at about the same time many states initiated mandatory rabies vaccination for cats so many more cats received adjuvanted rabies vaccines because these were the only type available at that time.

The recent trend of referring to these tumors as “injection-site sarcoma” (ISS) rather than “vaccine-associated sarcomas” (VAS) has purposely or inadvertently led to the misperception that any injectable agent given to a cat is equally likely to induce neoplasia at the injection site. (Yeh 2016). There is no question that the overwhelming majority of injection-site tumors are related to vaccine administration at the site. A recent review by Wilcock et al (2012) states: “The only proven cause for injection site sarcomas in cats is prior administration of a killed, adjuvanted vaccine. Claims implicating other agents such as lufenuron or microchips are unsubstantiated because previous vaccination in that same location could not be ruled out.” They also note that the interval between vaccine administration and tumor development may be as short as 4 months or as long as 13 to 15 years. The long lag period between vaccine administration and neoplastic transformation has hampered our ability to perform good epidemiological studies about this problem. It took many years to collect data from enough cats with a sufficiently long known vaccination history to compare the VAS incidence rates among cats that were administered different types of vaccine products.

A primary role of an adjuvant in a killed virus vaccine is to induce chronic site inflammation to recruit the antigen presenting cells of the macrophage/monocyte system to the site of injection to process the killed vaccine antigen and present it to the lymphocytes of the immune surveillance system. Vaccine adjuvants can also cause tissue necrosis, granuloma formation, retention of antigen at the injection site, and fibrosis. Adjuvant material and the associated inflammatory response may persist at the site of administration for many months to many years post-injection. It is this tissue damage combined with the chronic injection site inflammation that is suspected to be the cause of neoplastic transformation at the site of vaccine administration. A recent review by Okada (2014) states: “Inflammation, especially chronic one, is the definite cause for tumor development and progression, and it is well referred to as “inflammation-related carcinogenesis”.

It has been proposed by some that it is only the genetics of the cat that make it so susceptible to injection site neoplasia. Cats as a species are peculiarly susceptible to cell damage from oxidizing agents, hence their inability to tolerate some drugs and other chemical agents compared to other species. Inflammation results in the elaboration of many free radicals and reactive oxygen species (ROS – superoxides, peroxides, etc.). Kang et al (2014) report detection of endogenous DNA damage that neoplastic cells in injection-site sarcomas experience from replication stress, reactive oxygen species formation, and oncogene activation. While some cats may indeed be genetically more susceptible to inflammation-induced neoplasia than others, this alone does not explain why the problem of injection-site neoplasia associated with vaccines did not appear and become widespread throughout the world prior to the mid-1980’s. Cats breed locally, not nationally and internationally. For VAS to become a problem worldwide at approximately the same time, we cannot blame cat genetics alone. In any case, we cannot control the genetics of the cat. We can only control and limit the number and amount of chronic inflammatory agents that we inject into the cat.
For these reasons, the AAFP guidelines (2006) say to use “less inflammatory products”. The World Small Animal Veterinary Association (WSAVA - 2015) vaccination guidelines for cats state: “Non-adjuvanted vaccines should be administered to cats whenever possible.” The European Advisory Board on Cat Diseases (ABCD) Guidelines on prevention and management of ISS (2015) state: “Non-adjuvanted vaccines should be selected in preference to adjuvanted vaccines.”

Finally, the most recent epidemiologic review of injection-site sarcomas (Srivastav 2012) demonstrated a mean ten times higher risk of neoplasia in cats vaccinated with killed adjuvanted vaccines compared to recombinant (non-adjuvanted) vaccines. The range at a 95% confidence interval went from a 2.5 to an infinitely times greater risk for any individual cat. Because of this and for all the reasons discussed above, I personally recommend that inactivated, adjuvanted vaccines NEVER be used in cats.

**CORE FELINE VACCINES**

**PANLEUKOPENIA (FELINE PARVOVIRUS), FELINE HERPESVIRUS, FELINE CALICIVIRUS (FVRCP)**

There is little new to discuss regarding the core FVRCP vaccine. I recommend only modified live virus parenteral FVRCP vaccines because they are not adjuvanted. All of the major biologics manufacturers MLV FVRCP products are essentially created equal and work equally effectively.

Intranasal (IN) Herpesvirus/Calicivirus vaccine is NON-CORE but may be useful in high density cat environments such as shelters, breeding colonies, catteries, and cat rescue facilities where good husbandry may be lacking and kittens and older cats of unknown respiratory viral carrier status may be comingled. The IN vaccine may provide early onset local mucosal protection that is not blocked by maternal antibody. The objective is to try to prevent early age upper respiratory viral infection and the occurrence of nasal turbinate damage that may result in a cat with lifelong chronic secondary bacterial rhinosinusitis. There is no advantage to using a trivalent IN vaccine containing feline parvovirus because the IN route is not the natural route of infection for this virus. Parenteral feline parvovirus vaccination is preferred.

**FELINE LEUKEMIA VIRUS**

Feline leukemia virus (FeLV) is a type-C oncornavirus of the retrovirus group. Both horizontal and vertical transmission occur; virus is excreted in many secretions and excretions with saliva being the most important for transmission. FeLV is very unstable in the environment and is viable for an extremely short time under even the most optimal conditions. Therefore close, intimate contact between cats is required for transmission. The reported incidence of FeLV infection in cat populations varies depending on the source of the information but averages <1-3% in suburban cats to up to 30% in endemic multiple cat households and colonies. In our practices, we are seeing fewer FeLV cats because catteries (previously the largest source of infected cats) are now FeLV-free, most shelters and animal rescue groups are not adopting out (are keeping separate or euthanizing) FeLV positive cats, and most individual cat owners are having kittens or cats tested before allowing them into their households and tend to keep cats more confined. Nevertheless, the risk to pet cats from FeLV is still very real.

**THE TESTING CONUNDRUM - WHICH CATS SHOULD BE FeLV TESTED AND HOW ?**

I recommend using the more sensitive peripheral membrane blood/serum ELISA tests for routine screening (or prevaccination testing) of all sick or healthy cats. These tests are easily run using a few drops of whole blood so are suitable for testing even very small patients. However, red blood cell debris may produce false positive results in some test systems, and this may produce in a “weak” positive result. If you find this “weak positive” in a low risk cat, repeat the test on serum. These test systems also have built in positive
and negative controls so that any technical problem or cross reactivity should be immediately evident. Some test systems are also available as a combined FeLV/FIV test so that an adult cat can be tested for both diseases simultaneously. Two recent studies were reported in 2016 that showed that of the in-house ELISA tests, the IDEXX SNAP test has the best sensitivity and specificity (Liu 2016, Levy 2016).

Even with the high quality of the test systems available, the incidence of disease in the population of cats you are testing must be taken into account when you are interpreting FeLV test results. If the incidence of FeLV in the test population is only 1% as might be expected in a typical healthy suburban cat population, and you are using a test with a sensitivity and specificity of 98%, the reliability of a positive test is only 33%. That is, two of the three positive results you get from testing 100 healthy cats are false positive. Therefore, an apparently healthy cat or kitten should never be condemned on the basis of a single positive ELISA test result. Alternatively, using the same test on a sick cat population with an incidence of FeLV of 30%, the reliability of a positive test result is 99.7% (Christley – 2008).

If the peripheral blood ELISA test is positive, an immunoflorescent antibody (IFA) test can be performed to confirm infection and help determine the stage of the infection. The IFA test on peripheral blood is considered by many to be the gold standard for FeLV testing. A positive IFA test confirms well established FeLV infection in the bone marrow and correlates well with persistent infection for life. Drawbacks to this test are slightly reduced sensitivity compared to peripheral blood ELISA tests, it must be run by an outside commercial laboratory resulting in a time delay in obtaining test results, and higher cost than ELISA FeLV tests. Alternatively, a serum ELISA can be repeated in 3-4 months. If the cat is in an early stage of FeLV infection, regression of the infection and termination of viremia should occur within that period of time and the ELISA test will be negative when repeated. Cats remaining persistently ELISA FeLV antigen positive after 3-4 months will usually remain antigenemic for life. Table 1 shows the timeline for progression of FeLV infection in the cat and the expected results of testing during the course of infection.

**PCR TESTING FOR FeLV**

The polymerase chain reaction (PCR) is a methodology used to detect minute amounts of viral protein in a sample. In a study performed at the University of Florida, blood samples samples from 205 cats were evaluated with both ELISA and a polymerase chain reaction (PCR) test for FeLV antigen. The PCR test was unsuccessful in amplifying the sample in 39 cases and these were not evaluated. Both ELISA and PCR tests for FeLV antigen were positive in 17 cats (100% correlation). One hundred forty nine samples were ELISA negative and 148 of these were PCR negative (99.33% correlation). One sample was ELISA negative and PCR positive. It was suggested that this may represent a latently infected cat. Alternatively, this may represent a false positive PCR test. In this study, PCR FeLV results correlated nearly 100% with ELISA results. There seems to be no diagnostic advantage to using PCR as a screening test; it cannot be performed in-house, and is more than twice as expensive as ELISA testing. In addition, PCR could not be successfully performed on 20% of the samples submitted in this study. PCR testing may have an advantage in a symptomatic FeLV suspect that is repeatedly negative on routine FeLV testing (ELISA, IFA). If PCR can truly detect latent virus (which we don’t know for sure), it may be possible to confirm infection in such a patient. Bone marrow would be a better target tissue than peripheral blood for attempting to identify latent infection in these cats.

**FeLV VACCINATION**

As of 2017, there are a number of contenders in the FeLV vaccine market. The reported efficacy of these vaccines varies depending on the conditions under which they were studied and how the studies were performed. The most recent comparative study of three commercial FeLV vaccines (two inactivated adjuvanted vaccines and one recombinant non-adjuvanted FeLV vaccine) using kittens tested under
identical laboratory conditions closely mimicking natural infection shows no statistically significant
differences among the vaccines’ ability to protect against persistent viremia. (Grosenbaugh et al, 2016)

Even without vaccination, adult cats have a great deal of natural resistance to FeLV infection. That is why
vaccine studies using adult cats as the experimental subjects have had to use massive, immunosuppressive
doses of corticosteroids to make the unvaccinated cats (controls) susceptible to FeLV challenge and show
any significant difference in protection upon FeLV challenge compared with the vaccinated group. Clearly
this is a very artificial infection system and not a natural challenge model that is comparable to conditions
of FeLV exposure that a pet cat would experience in nature.

I strongly recommend that ALL cats/kittens be tested with a serum ELISA test before vaccination. The
client is wasting his/her money if the cat is already infected. There is no scientific evidence to show that
vaccination of FeLV positive cats significantly improves their condition or longevity. And it is difficult to
explain to a client why their cat is later diagnosed with FeLV disease after it has supposedly been
"protected by vaccination".

WHICH CATS SHOULD BE VACCINATED AGAINST FeLV?

FeLV is a disease of friendly cats which mutually groom and interact closely on a continuing basis. In
nature, most kittens are infected either in utero or during the nursing period via milk or saliva from the
infected queen as she is grooming the kittens. A kitten infected at less than 6-8 weeks of age has a nearly
100% chance of remaining persistently infected for life. Susceptibility to FeLV infection slowly decreases
incrementally over the first year of life. (Hoover 1976) Because protecting against FeLV during this most
susceptible first year is so important, the AAFP and other groups that have developed feline vaccination
guidelines (World Small Animal VMA, European Advisory Board on Cat Diseases) all recommend
universal kittenhood vaccination against FeLV.

Alternatively, it is very difficult to naturally infect a healthy adult cat (over one year of age) with FeLV and
this vaccine is considered NON-CORE for adult cats. (Wilson 2012) A single adult cat confined strictly
indoors has no chance of exposure or infection. Adult cats in FeLV-negative catteries that use good
screening procedures and maintain an FeLV-negative status are at the lowest risk and as long as closed
status and test procedures are maintained, FeLV vaccination is not necessary for this group. For adult pet
cats, I usually ask the owners about environmental history, tell the owner about the vaccine and that while
there is natural resistance in adults, vaccination will provide additional protection. The cats most at risk for
FeLV infection are those in multiple cat households in which FeLV is endemic. Casual contact will not
result in infection. The risk to suburban indoor/outdoor cats is generally quite low. I recommend testing for
FeLV prior to vaccination and let the owner make the decision about whether to vaccinate for this disease
or not in adult cats.

RABIES

It is surprising to me that some states do not currently require rabies virus vaccination for cats. In the
United States, cats have the highest incidence of rabies of all domestic animals. (Monroe 2016) The
incidence of rabies in cats is more than three times that of dogs. This high rate of rabies infection in cats
probably relates to the fact that rabies vaccination of cats is not required in some locales and because cats
are more likely than dogs to interact non-fatally with potential wildlife reservoirs such as skunks, raccoons,
and foxes. Even cats kept strictly indoors are not protected from potential rabies exposure. Bats are the
second most common wildlife rabies carrier species in the United States. Bats live in or can enter homes
and are often hunted down and captured/ killed by domestic cats putting them at risk for rabies exposure.
Therefore, for the protection of both cats and their owners, rabies vaccination is recommended for all cats regardless of their housing/husbandry status. As with other feline vaccines, I recommend only non-adjuvanted rabies vaccine for cats. There are now both 1 year and 3 year duration of immunity USDA approved non-adjuvanted recombinant feline rabies vaccines available and practitioners have a choice as to the type of product they would prefer for their patients.

An excellent online resource for information about rabies law, reported cases of rabies, and management of potentially rabies exposed animals for each state, is the RabiesAware website: http://www.rabiesaware.org. This site is an excellent resource developed by Dr. Richard B. Ford working with the cooperation of and input from the Public Health veterinarians in each state. Data on the site is updated regularly as rabies incidence and state rabies regulations may change over time.

OTHER NON-CORE FELINE VACCINES

Chlamyphila and Bordetella are on the AAFP non-core list but I do not recommend giving either of these vaccines to household pet cats. Both of these diseases are very uncommon causes of upper respiratory disease and these vaccines are reactive and have a short duration of immunity. Chlamyphila or Bordetella bacterins may be helpful for short-term use in shelter or cattery situations where an outbreak of upper respiratory disease has occurred if these agents are cultured from a number of cats and confirmed as a major component of the respiratory syndrome.

VACCINES NOT RECOMMENDED

Feline Coronavirus (FIP), Feline Immunodeficiency Virus (off market in 2016), Virulent Systemic Calicivirus

PEDiatric Vaccination:

The goal of pediatric vaccination is to stimulate active and solid immunity before the susceptible kitten is exposed to pathogenic organisms. This means that we must start a vaccination program early enough to prevent active disease as maternal antibody wanes.

The pediatric core FVRCP vaccine series should be started when the kitten is seen for its first pediatric examination at 6-8 weeks of age. Core vaccines should be repeated at 3-4 week intervals until the kitten is 16 weeks of age. Although some biologics manufacturers have experimental studies that demonstrate good protection by 12 weeks of age, recent research using conventional kittens indicates that maternal antibody interference with vaccination may persist in some kittens beyond 13 weeks of age. Therefore, the vaccination guidelines recommend administering the final pediatric vaccination at 16 weeks of age or older. Rabies vaccine should be given at 12 weeks of age or older as per the Rabies Compendium and state/local ordinances. I recommend using only a non-adjuvanted rabies vaccine for cats.

As previously described, kittens should be tested negative for FeLV prior to vaccination. In addition to the other serious consequences of infection, there is no demonstrated benefit of giving an FeLV vaccine to an FeLV-infected cat. I recommend using only non-adjuvanted FeLV vaccine according to the manufacturer’s instructions.

RE-VACCINATION INTERVALS:
The CORE vaccines: FVRCP, FeLV, and RV should be repeated at one year of age. As per AAFP, WSAVA, and ABCD recommendations, FVRCP is recommended no more frequently than every 3 years after that time. RV should be re-administered according to the manufacturer’s licensing approval (1 year or 3 years) and according to national/state/local ordinance. FeLV vaccination may be continued if the cat is at risk of exposure after 1 year of age.

**VACCINE SAFETY: ADVERSE EVENTS**

Adverse events associated with vaccine administration to small animals are relatively rare given the frequency with which vaccines are given to patients and the complexity of these biological agents. The one large published study reviewed patient records from the database of a large corporate veterinary clinic network. The incidence of adverse events associated with vaccination was reported to be approximately 1:200 for cats within 30 days of vaccination. In these studies, young animals and cats receiving multiple vaccines/antigens per visit were at higher risk for an adverse event. (Moore 2010) Unfortunately, the true incidence of adverse events associated with feline vaccination is still largely unknown because adverse events may not be recognized as vaccine-related, may not be reported by owners to veterinarians, may not be reported by veterinarians to manufacturers, and because we have no unbiased national database for adverse event reporting to which veterinarians or researchers have access.

**ADVERSE EVENT REPORTING**

In order to properly report a suspected adverse vaccine event, veterinarians must keep accurate patient records that include the product name, manufacturer, lot and serial number, and the location of vaccine administration. All suspected events should be reported immediately to the professional services hotline for the vaccine manufacturer. If the patient is simultaneously given several vaccines produced by different manufacturers, it may be impossible to determine which one may have caused the adverse event. If a serious adverse event is confirmed, it should also be reported to the USDA adverse event reporting site:

https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/veterinary-biologics/adverse-event-reporting/ct_vb_adverse_event (Google: USDA adverse event)
TABLE 1

PATHOGENESIS OF FeLV

Following infection, FeLV has a specific pattern of replication that affects the results of FeLV testing and the clinical signs that may be seen in an individual cat.

STAGE I: Days 2-4
Replication: In local lymphoid tissue (retropharyngeal, tonsil, GI mucosa.
Clinical signs: None to mild viral (fever)
FeLV status: All tests negative at this time
Prognosis: Other than kittens, the majority recover

STAGE II: Days 4-21
Replication: Few circulating lymphocytes and mononuclear cells (primary viremia).
Clinical signs: None, or mild viral signs
FeLV status: Serum ELISA becomes positive, PCR may be positive, (IFA, saliva, tears negative)
Prognosis: Most recover, possible latency (6-30 months)

STAGE III: Day 21+
Replication: Systemic lymphoid centers (germinal centers)
Clinical signs: None, or mild to moderate viral signs
FeLV status: Serum ELISA positive, PCR positive, (IFA, saliva, tears negative)
Prognosis: Recovery for many, possible future LSA

STAGE IV: Day 21-42
Replication: Bone marrow stem cells, epithelial cells
Clinical signs: Peripheral blood alterations, viral signs
FeLV status: Serum ELISA, PCR, BM IFA positive, (peripheral blood IFA +/-, saliva, tears negative)
Prognosis: Likely to progress to persistent infection

STAGE V: Days 42+
Replication: Marrow origin, generalized viremia
Clinical signs: All associated hematologic and systemic FeLV signs possible
FeLV status: Serum ELISA, PCR, BM and peripheral blood IFA positive (saliva, tears negative)
Prognosis: Persistent viremia, recovery from this stage of infection is rare

STAGE VI: Days 42+
Replication: Marrow viremia, widespread epithelial and lymphoid replication
Clinical signs: Any associated with FeLV
FeLV status: Serum ELISA, PCR, BM and peripheral blood IFA, ELISA on saliva and tears may be positive
Prognosis: Long term prognosis is grave, 85% of cats die within 3 years in multitude households, longevity is increased for single cats with good veterinary care
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Bartonellosis

Introduction

_Bartonella_ spp. cause cat scratch disease (CSD) and other clinical syndromes in human beings, and are an important cause of endocarditis in dogs. On the other hand, there is scant documented scientific evidence that _Bartonella_ infection causes overt clinical disease in naturally infected cats, in spite of a high prevalence of bacteremia and seropositivity in areas of the United States with warm temperatures and high humidity.

Microbiology

_Bartonella_ spp. are facultatively intracellular gram-negative rods that are related closely to _Brucella_ spp and the rickettsiae. Their intra-erythrocytic location precludes easy blood culture and a reliable response to antimicrobial therapy.

Four species of _Bartonella_ have been shown to infect pet cats. _B. henselae_ infection is most common, and is the most important cause of CSD. _B. clarridgeiae_ may be responsible for a small number of cases of CSD. Rare infections of cats with _B. koehlerae_ and _B. bovis_ also have been reported. Two main genotypes of _Bartonella henselae_ have been identified worldwide – Houston and Marseille. A third genotype, Berlin, has only been identified from one cat in Germany. Exotic cats have also been found to carry _Bartonella_ sp. including: mountain lions, cheetahs, African lions, Florida panthers, pumas, and bobcats.

Transmission

_Bartonella_ spp. are transmitted between cats by _Ctenocephalides felis_ – the cat flea. Fleas ingest the organism during a blood meal from a bacteremic cat, and infect a naïve cat through regurgitation of infected saliva during a subsequent blood meal. Ticks may transmit the organism rarely between cats, and are the primary mode of transmission of _Bartonella_ spp. between dogs. The organisms are not transmitted between cats by fighting, grooming, mating, or in-utero.

Human beings become infected with _Bartonella_ spp. when flea feces from a bacteremic cat are inoculated into a cat scratch. Although not confirmed, rarely, infection may possibly be acquired directly through the bite of an infected flea.

Cats at Risk

Although _Bartonella_ infections in cats have only been reported in the modern veterinary literature since 1992, the organism has apparently been infecting and adapting to cats for hundreds of years. A recent paper reports the isolation of _Bartonella_ antigen from dental pulp by PCR assay in 800 year-old cat teeth from France. The prevalence of bacteremia and seropositivity in cats in the United States is highest in regions that favor the reproduction and persistence of fleas. Rates are highest in the southeastern United States (up to 40%), and lowest in the northern tier of states. The incidence in the EU, UK, and other countries also mirrors this pattern with higher seroprevalence rates in warm, moist locales, and much lower rates in colder climes.
Bacteremia is more likely to occur in cats with fleas, free-roaming cats, young cats, and those from multiple-cat populations.

Pathogenesis

After experimental inoculation into Bartonella-naïve cats, the organisms infect erythrocytes and the initial bacteremia lasts 2 to 32 weeks. After this, infected cats undergo bouts of cyclic bacteremia. Between bacteremic phases, when blood cultures (and blood PCR tests) are negative, the organisms may persist in endothelial cells, lymph nodes, or the central nervous system.

Following initial production of anti-Bartonella IgM antibody, IgG antibody is produced and remains detectable for months to years. There is no evidence that the height of the IgG antibody titer correlates with the presence of bacteremia – this observation is important when considering the diagnostic utility of antibody titers.

Clinical Signs – Experimental Infections

Part of the confusion about the clinical importance of Bartonella infections in cats arises from the observation that clinical signs are more likely to occur in cats after experimental inoculation that in naturally infected cats. Inappropriate extrapolation of data from experimental studies is one of many factors that have led to an overdiagnosis of clinical bartonellosis in the general cat population.

Following experimental inoculation, some infected cats have developed an inflammatory lesion at the injection site, mild generalized lymphadenopathy, splenomegaly, fever, lethargy, anorexia, myalgia, behavioral or neurologic changes, and reproductive abnormalities. In a number of other studies, no clinical signs were seen following infection. This probably relates to variable pathogenicity among the strains of B. henselae used in these studies.

Clinical Signs – Natural Infections

With rare exception, Bartonella spp. cause prolonged asymptomatic infections in naturally infected cats. Well-documented clinical signs arising directly from infection are very unusual and mostly anecdotal.

Bartonella spp. have been linked directly with endocarditis in one cat. Based on anecdotal reports, the organism may be a rare cause of lymphoplasmacytic gingivostomatitis (LPG) and uveitis. Clinicians should remember that feline calicivirus and plaque intolerance are very common causes of chronic LPG, and that many affected cats coincidentally will be Bartonella-seropositive given the high prevalence of infection with the organism.

A similar situation arises with uveitis, which usually is caused by viral or fungal infections. Because uveitis often is accompanied by intra-ocular bleeding, the isolation of Bartonella organisms or antibody from within the eye does not confirm infection. The diagnosis of Bartonella-induced uveitis is supported by the exclusion of all other more common causes, the demonstration of higher antibody levels in the aqueous humor than in serum, and a specific response to selective antimicrobial treatment.

Recent studies by Dr. Mike Lappin and the infectious disease group at Colorado State University have shown no statistical differences in Bartonella seropositivity between cats with and without uveitis, oral cavity disease, and central nervous system disease.
**Bartonella Infections in Human Beings**

The CDC estimates that there are 24,000 cases of CSD/year in the U.S. This is an incidence of 9.3/10,000 ambulatory patients/year. The seropositivity rate for *Bartonella* in humans is between 3.6% to 15%; with the latter value occurring in a survey of veterinary professionals. Most persons inoculated accidentally with infected fleas feces through a cat scratch probably show no clinical signs or suffer from a vague, mild self-resolving, flu-like illness that does not prompt a visit to the physician. On the other hand, some immunocompetent people will develop typical CSD, with or without systemic complications. Persons with impaired immune systems are at risk for more severe complications of infection.

Typical signs of CSD include the development of a pustule (primary inoculation lesion) in the infected scratch within 7 to 10 days of the injury. Regional lymphadenitis, usually non-painful, occurs within 1 to 3 weeks of the injury. Lymph node enlargement may persist for weeks to months. Antimicrobial treatment does not shorten the duration of disease reliably.

Atypical signs of CSD include Parinaud’s oculoglandular syndrome (associated with a primary inoculation lesion on the conjunctiva and regional lymphadenopathy following infection of the conjunctiva with flea feces from a bacteremic cat), relapsing bacteremias and fevers, encephalitis, endocarditis, hepatitis, pneumonia, and osteomyelitis.

Angioproliferative lesions are more common in immunocompromised persons and include cutaneous lesions of bacillary angiomatosis, and cystic hepatic lesions of bacillary peliosis. Systemic complications of zoonotic *Bartonella* infections are more likely to be severe in immunocompromised human patients. Paradoxically, the response of these patients to antimicrobial treatment is better than that of immunocompetent patients with typical CSD.

**Diagnosis**

Laboratory tests to detect or exclude infection with *Bartonella* spp in cats include the detection of anti- *Bartonella* antibodies through immunofluorescent antibody (IFA) and Western Blot (WB) tests, blood cultures, and the amplification of *Bartonella* DNA by polymerase chain reaction (PCR) tests. These tests are used to place cats into one of the following categories:

1. Healthy cats that are not infected with *Bartonella* spp., and therefore are safe companion animals for immunocompromised persons.
2. Healthy cats that currently are infected with *Bartonella* spp., or that have been infected previously with *Bartonella* spp.
3. Sick cats (for example, cats with uveitis or stomatitis) with concurrent *Bartonella* infection that is not the cause of their clinical illness.
4. Cats with *Bartonella*-induced illness (an unusual occurrence in clinical practice).

Because of the high prevalence of seropositivity to *Bartonella* in the general cat population and the low incidence of *Bartonella*-induced disease, the detection of serum
antibodies has a poor predictive value (42 to 46 per cent) for the confirmation of disease caused by the organism. Similarly, using the IFA test, there is a poor correlation between the height of the antibody titer and the ability to detect bacteremia. Because titers in infected cats vary greatly over time, increases in titers associated with vague clinical signs should be interpreted with caution.

Conversely, a negative IFA titer has a high negative predictive value (>90 per cent), making it a useful screening test to exclude bacteremia in an asymptomatic or symptomatic cat. A small number of cats may be seronegative between cycles of bacteremia, and blood cultures and PCR tests may be needed to confirm the status of these cats, especially if they are being considered as companion animals for immunocompromised persons.

With the possible exception of endocarditis, the clinical diseases attributed anceotally to infection with *Bartonella* spp. usually are caused by more common infections. Therefore, tests for these other diseases (for example, FeLV/FIV, toxoplasmosis, cryptococcosis, histoplasmosis, and the aforementioned causes of stomatitis) should be performed and interpreted before tests for *Bartonella* infection are ordered.

Based on our present scientific knowledge of the epidemiology of *Bartonella* infections in cats and human beings, there are no valid indications for the routine testing and subsequent treatment of healthy pet cats that live with healthy owners. This recommendation is also that of the Centers for Disease Control and this information is available on their website: www.cdc.org

**Treatment**

Treatment should be reserved for that small group of sick cats with apparent *Bartonella*-induced disease, based on careful interpretation of serological and culture/PCR results and an exhaustive exclusion of other more common diseases. At the present time, there is no evidence that antimicrobial therapy eradicates *Bartonella* organisms completely from infected cats. Although the level of bacteremia may be reduced temporarily, recurrence of bacteremia usually occurs due to the intra-erythrocytic location of the organism.

Enrofloxacin and doxycycline have been used to reduce the level of bacteremia. Unfortunately, the recommended dose of enrofloxacin has a high risk of inducing retinotoxicity, precluding its safe use in cats. Azithromycin (5-10 mg/kg PO q24h for 5d, then q48h for 40d) has been recommended as more effective. Unfortunately, because bacteremia is cyclic, and because organisms are rarely cleared with antibiotic therapy, there is no good endpoint on which to base apparent success of treatment. PCR testing may be negative at some point, and then return to positive weeks to months later. Antibodies will persist for years, often at high levels even after organisms are gone.

**Prevention**

Prevention of CSD in human beings sharing a house with cats depends primarily on scrupulous and effective flea control. Even if the cat is bacteremic, human infection from cat scratches, etc. will not occur unless the injuries are contaminated with flea feces. Children, who are at most risk for the development of CSD if infected, should be taught to play gently with their pet cats, especially new kittens, to avoid scratches.

Cats being considered as companion animals for immunocompromised persons should be selected from a flea-free background if possible. The cats should be screened
initially with an IFA test. If the test is positive, it would be wise to consider the cat no further as a safe companion. If the IFA test is negative, blood cultures and PCR tests should be performed. If these latter tests are negative, the cat can be considered safe, as long as it is kept indoors exclusively, not exposed to cats with fleas, and is treated diligently with a year-round flea-control program.

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Bacterial L-form Infections

One of the more interesting causes of chronic abscesses is L-form bacterial infection. L-forms are cell wall-free bacteria that are usually a common species that has undergone alteration due to some environmental pressure. Lesions occur most often on the limbs and systemic signs of illness such as fever and anorexia accompany the abscesses. Joints may be involved and can collapse due to cartilage destruction. This discharge appears purulent and is granulomatous with many PMNs and macrophages. Organisms cannot be identified on cytologic examination and culture on routine bacteriologic media is unsuccessful. L-form infections do not respond to commonly used antibacterial agents but respond promptly to tetracycline drugs (tetracycline and doxycycline [most recommended for the cat due to fewer side effects]). The clinical appearance of Mycoplasma infections is similar to that described for L-forms and tetracycline drugs are also effective against these organisms.
Methicillin-Resistant Staphylococcus Aureus (MRSA)
Methicillin-Resistant Staphylococcus Aureus

What is MRSA and where does it come from?

MRSA is a Staphylococcus aureus that has acquired the mecA gene, which encodes a protein (PBP2A) that reduces the binding affinity of beta-lactams, including penicillins, cephalosporins and carbapenems (e.g. meropenem) (Weese 2005, Duquette & Nutall 2004, Weese 2005Proc). Therefore, MRSA strains are resistant to a wide range of drugs, and frequently also possess genes or mutations conferring resistance to a variety of other antimicrobials (Rich et al 2005). While MRSA is not more inherently virulent than S aureus susceptible to these antibiotics, the sometimes limited treatment options and likelihood of failure of initial or empirical therapy can complicate treatment of infections.

Initially in humans, most cases MRSA were hospital-associated, occurring in patients that are or have recently been hospitalized. In the late 1990s, “community-associated MRSA”, or CA-MRSA, was increasingly identified as a problem, with infections occurring in people with little or no hospital exposure. As MRSA increased in people in the community, the potential for exposure of companion animals increased, and there was a parallel increase in identification of MRSA in various animal species. MRSA strains found in household pets are almost always strains that are common in people in the region, supporting the human source of MRSA. Most often, USA100 (CMRSA-2) is identified in pets in North America, while in other countries strains identified in pets can differ, depending on the prevalent human strains (e.g. eMRSA-15 in people and pets in the UK). In horses, MRSA strains can be more variable, with a combination of a human strain that seems to be horse adapted (USA500/CMRSA-5), common human strains (USA100) and livestock associated MRSA strains (ST398). In some regions, livestock-associated MRSA is a concern. This is most common in some European countries but can be found almost worldwide. The livestock-associated MRSA strain ST398 is most common and is a human strain that moved to pigs and became methicillin-resistant. This strain is widespread in livestock (especially pigs) in many regions and can cause human infections. Pet infections are uncommon but can occur.

Is MRSA a concern for my patients?

Morris et al 2006a,b, O’Mahoney et al 2005, Owen et al 2004, Rich 2005, Rich & Roberts 2004, Rich et al 2005, Strommenger et al 2006, , van Duijkeren et al 2004a,b, van Duijkeren et al 2005, van Duijkeren et al 2003, Weese 2005, Weese 2006a,b,c). The risk of MRSA infection is thought to increase in animals with close contact with people at higher risk of MRSA infection or colonization. This would include animals that visit hospitals, animals that live with people with MRSA infections or animals that live with people that are higher risk for MRSA exposure (e.g. healthcare workers). Antibiotic exposure and veterinary clinic hospitalization are important risk factors for pets.

**Do all animals with MRSA get sick?**

No. Only a small percentage of animals (or humans) that acquire MRSA develop illness (Tomlin et al 1999, Owen et al 2004). The actual percentage is unknown but it is likely a very small number compared to the number of animals that are “colonized”. MRSA colonization is the presence of MRSA on or in the body (e.g. skin, pharynx, nose, gastrointestinal tract) in the absence of disease. Just like S. aureus rarely causes infections in otherwise healthy animals, most animals that are exposed to MRSA either are not receptive to the organism or become transiently colonized, whereby the bacterium can be isolated but disease is not present. While the likelihood of a colonized animal developing disease is low overall, the risks are higher in patients that are more likely to develop an opportunistic infection, such as those with allergic skin disease, immunocompromised or undergoing surgery. These “colonized” animals are also a concern because of the potential for transmission of MRSA to human contacts (Boost et al 2007, Cefai et al 1994, Cuny et al 2006, Enoch et al 2005, Huijsdens et al 2006, Juhász-Kaszanyitzky et al 2007, Leonard et al 2006, Loeffler et al 2005, Manian 2003, O’Mahoney et al 2005, Strommenger et al 2006, van Duijkeren et al 2005, Weese et al 2006a,c).

The prevalence of MRSA colonization is low overall, with most studies reporting rates <2%.

**My client or their physician claims that their MRSA infection was transmitted from their pets. What do I tell them? What should I do?**

Interspecies transmission of MRSA occurs. Humans can infect dogs or cats, and pets can infect their owners (Boost et al 2007, Cefai et al 1994, Cuny et al 2006, Enoch et al 2005, Huijsdens et al 2006, Juhász-Kaszanyitzky et al 2007, Leonard et al 2006, Loeffler et al 2005, Manian 2003, O’Mahoney et al 2005, Strommenger et al 2006, van Duijkeren et al 2005, Weese et al 2006a,c). Thus, it is often difficult to determine which way MRSA was transmitted, although human-to-pet transmission is likely much more common than pet-to-human transmission. It is plausible that a community-associated MRSA infection originated from a pet, but this is probably rare. Since MRSA is a human-associated bacterium, pets that are MRSA carriers probably acquired it from
household contacts, who would be more likely sources of infection. The likelihood of a pet being the source is probably greatest when a pet acquired MRSA at a veterinary hospital and takes it home to a household that was MRSA free.

Determining whether a pet is the source of infection is difficult to impossible. Isolation of MRSA from the pet of a person with MRSA does not say whether the pet infected the person, the person infected the pet, or both were infected by the same unknown source. It is rarely, if ever, indicated to investigate a pet as a source of a sporadic MRSA infection in an owner.

MRSA can cause recurring infections in households, where there are serial infections of different individuals. Sometimes, whole household eradication is attempted, with treated of all people to break the transmission cycle. In that type of situation, where the “whole” household is being treated, it is reasonable to consider the pet as part of the “whole” household. This might include discussion of improved infection control and hygiene practices for pet contact or consideration of temporary removal of the pet from the household while decolonization therapy of the people is underway. Since MRSA colonization in dogs and cats is transient, the pets can be re-introduced to the household after testing. This is a rare situation.

Concern has been raised about dogs involved in hospital visitation programs, both in terms of an increased risk of developing MRSA infection and a risk of transmitting MRSA to hospitalized patients. Dogs that visit human hospitals are at increased risk of MRSA infection (Lefebvre et al 2009). Dogs that are allowed to lick patients or are fed treats by patients are at increased risk, likely from ingestion of MRSA from the patient’s skin. Contamination of the dog’s haircoat with MRSA has also been identified after visitation. Guidelines for pet therapy programs have been developed to reduce the risk of MRSA transmission (Lefebvre et al 2008, Murthy et al 2015). These guidelines do not recommend screening of dogs for MRSA colonization.

**When should I consider screening pets?**

Routine screening is not advised. Rarely is there any indication to screen any pet for MRSA colonization, apart from research studies. One main reason is that screening results rarely change management of the pet. Negative results are not a guarantee of MRSA negative status, because screening is not 100% sensitive. Further, pets that live with infected or colonized individuals, or have contact with high risk individuals (e.g. hospital therapy animals) are at continual risk of exposure. Screening may provide an indication of the MRSA status on the day or screening, but exposure could happen any day after. The recommendations would be to continue to use good hygiene and infection control practices to reduce the risk of MRSA exposure. MRSA positive status would not change that
recommendation, since the focus for prevention of MRSA transmission from pets to people is hygiene (e.g. hand washing, avoiding contact with the nose, mouth and feces, avoiding licking). These are the same recommendations that would be reasonable for any pet that may have contact with high risk individuals. Another reason not to screen is a lack of evidence that decolonization therapy is effective or indicated in dogs or cats. MRSA colonization is almost always transient in those species, so MRSA will be eradicated naturally if re-exposure is prevented (i.e. through good hygiene practices). Active decolonization using topical (intranasal) antibiotics, systemic antibiotics and chlorhexidine bathing is used in humans but is impractical in dogs and there is no evidence that it could be effective. Therefore, neither routine testing nor treatment of healthy carriers are indicated.

**Are my staff at risk?**

Several studies have identified an increased risk of MRSA colonization in veterinary personnel compared to the general public. Reasons for this have not been specifically investigated but it likely relates to exposure to patients with MRSA colonization, probably along with deficiencies in standard infection control practices such as hand hygiene.

Most people that carry MRSA do not develop disease, but MRSA carriage can lead to an increased risk of infection in certain situations (e.g. after undergoing surgery). There are a limited number of reports of MRSA infections in veterinary personnel, but more anecdotal information. It is likely that the incidence of occupational MRSA infections is very low in veterinary personnel, but some risk is present.

While veterinary personnel are at increased risk of exposure, testing is not indicated. Testing is rarely of use for people in the general population (as opposed to people being admitted to hospitals). Further, test results would not likely lead to any treatment, since decolonization of MRSA carriers is not a routine or recommended practice in most countries. If MRSA infections are occurring in patients in a clinic, it is reasonable to consider that there might be a human source. However, testing is fraught with potential problems. Identifying MRSA in a veterinary healthcare provided would not confirm whether the person was the source of infection or whether they were infected by a patient. Testing also involves many privacy and liability issues that are best avoided (e.g. if a person is positive, does that represent an occupational infection?).

It is important for veterinary personnel to be aware of their increased MRSA risk. They should ensure that their physician understands that as well. In situations where high risk personnel are screened (e.g. people from long-term care facilities or who have been recently hospitalized are often screened for MRSA at hospital admission or prior to selected surgical procedures), it is reasonable to include
veterinary personnel in that high risk group. That allows for implementation of specific infection control practices (e.g. decolonization prior to surgery, altered peri-operative antimicrobial prophylaxis). Otherwise, screening is not recommended.

**Should I treat an animal that is a carrier of MRSA?**

As discussed above, routine decolonization therapy is not recommended in humans or animals that are colonized with MRSA. There is currently no evidence that it is effective, and most (if not all) pets will clear MRSA colonization spontaneously if re-infection is prevented. Nasal mupirocin is not practical because nasal passages cannot be adequately covered. Additionally, intranasal therapy does not address pharyngeal, GI or cutaneous colonization. Chlorhexidine baths can be used but they do not address the primary colonization sites.

The key to minimizing colonization is stringent household infection control practices, in particular avoiding high risk contact and frequent hand hygiene. If re-infection is prevented, culturing colonized animals every few weeks generally demonstrates that they have eradicated the organism within a few weeks. In rare situations where MRSA infections are rampant in people in the household and the entire household is undergoing eradication therapy, kenneling the pet for a couple weeks is a reasonable option, to allow it to clear the colonization and avoid re-infection.

**Are there other methicillin-resistant staphylococci? Are they similar to MRSA?**

Any Staphylococcus species can be methicillin-resistant. Methicillin-resistance rates are high amongst coagulase negative staphylococci (CoNS). However, this group of staphylococci rarely causes disease (with the exception of S. schleiferi schleiferi in skin infections). While methicillin-resistant CoNS can be isolated, they are often contaminants and treatment is infrequently required. These are thought to pose little to no zoonotic risk.

Coagulase positive staphylococci are the main causes of infection. This includes S. aureus, S. pseudintermedius (previously often referred to as S. intermedii) and S. schleiferi coagulans. These are important pathogens in animals and all can be methicillin-resistant. Methicillin-resistant S. pseudintermedius (MRSP) is the most common cause of MR-staphylococcal infection in dogs and cats, and has become very common in recent years. MRSP is often highly resistant, with few viable treatment options.

**How do I treat MRSP and MRSA?**
Treatment is guided by disease factors (e.g. location, type of disease), patient (e.g. age, comorbidities) and bacterium (antimicrobial susceptibility) factors. MRSP is no more inherently virulent than susceptible S. pseudintermedius so the only thing that differs between treating MRSP and susceptible S. pseudintermedius infections is the drug choice. Early identification of the MRSP infection is important to ensure prompt treatment with an appropriate antimicrobial. However, the presence of methicillin-resistance does no mean that patients need to be treated more aggressively, longer or any differently than an animal with a similar infection caused by methicillin-susceptible S. pseudintermedius (apart from the drug choice). The same applies for MRSA.

MR-staphylococci should be considered resistant to all beta-lactams (including meropenem), regardless of laboratory results. Laboratories are supposed to report MRSP as resistant to all beta-lactams, regardless of results, but this is not universally applied. There is a limited number of anti-MRSA cephalosporins (e.g. ceftobiprole) but these have not been studied in animals and their impact on MRSP is unknown.

Clindamycin can be an excellent drug for staphylococcal infections but inducible resistance is a concern. This is a phenomenon whereby the bacterium has resistance that is not detected using regular testing methods, but is induced in response to the drug. This occurs most often in MRSA but can be present in a small percentage of MRSP isolates. Inducible resistance is only a concern is isolates that are reported as erythromycin-resistant but clindamycin-susceptible (or where erythromycin susceptibility is not known). Those should be testing with methods to detect inducible resistance (e.g. D-test). It is important to know if the laboratory providing results performs such testing if clindamycin is to be considered.

Doxycycline can also be an effective drug. However, breakpoints that are widely used by laboratories will indicate some S. pseudintermedius as susceptible when they are truly resistant. Lower breakpoints have been recommended but these are not widely used. An MIC less than 0.125 ug/ml should be present to consider an isolate susceptible (Maaland et al 2014). Reports that indicate S/I/R or MICs where the lowest concentration that is testing is above 0.125 ug/ml cannot provide confidence of true susceptibility.

Fluoroquinolones may be effective in vitro for MR-staphylococci but they are not typically recommended. In humans, they are generally contraindicated for MRSA infections because of poor clinical response. Data are not available for animals but clinical observation suggests that the same applies for MRSA and MRSP infections in animals. These drugs should not be routinely used for MR-staphylococcal infections. However, there are recent reports from some regions of fluoroquinolone-susceptible MR-staphylococci that are different than the most common genetic lineages but that clinically do appear to respond to
fluoroquinolones. It is reasonable to continue to recommend avoiding this drug class, particularly for life-threatening infections, unless regional evidence suggests a likelihood of efficacy.

While MRSP is often resistant to most antimicrobials, there are typically a few viable (although not always desirable) options. Amikacin and chloramphenicol are often effective both in vitro and in vivo. The use of drugs that are used for treatment of resistant pathogens in humans (e.g. vancomycin, linezolid) should be avoided as much as possible. Use of drugs such as those in animals is banned in some regions, as is the use of chloramphenicol in some countries. Consultation with an expert in infectious diseases is warranted when use of drugs such as those is being considered.

Abscesses caused by MR-staph can often be managed with incision and drainage, plus or minor local therapy (e.g. chlorhexidine). If a patient has a discrete abscess with no signs of systemic infection (e.g. fever) and no evidence of cellulitis extending beyond the abscess, antimicrobials are not likely indicated. Similarly, topical treatment of superficial pyoderma (e.g. chlorhexidine bathing) has been recommended as a first-line treatment, without antimicrobials (Hillier et al 2014).

**Do dogs and cats persistently carry MRSP?**

As opposed to MRSA, MRSP is a host-adapted species for dogs. There is evidence that dogs can carry MRSP for potentially long periods of time after infection. This complicates MRSP control because dogs may be a short-term, long-term or even persistent source of exposure of other animals and humans. Currently, there is no known way to eliminate MRSP colonization.

**Is MRSP zoonotic?**

Like many bacteria found in and on dogs and cats, there is the potential for MRSP to cause human infection. However, the risk appears to be very low. MRSP is no more likely to cause an infection that meticillin-susceptible S. pseudintermedius (MSSP). Most dogs carry MSSP so there is abundant exposure of people to MSSP. Yet, human MSSP infections are very rare demonstrating the poor affinity that this bacterial species has for humans. MRSP infections have been reported in people (Stegmann et al 2010, Starlander et al 2014), so the potential must not be dismissed; however, care must be taken to approach the risk in a balanced manner. Basic hygiene and infection control practices (e.g. hand washing, avoiding contact with infected sites or common colonization sites) is recommended for people that are in contact with MRSP infected dogs. More intensive measures are not recommended, even in households with high risk personnel. Realistically, most healthy dogs carry pathogens that are more likely to cause disease in people than MRSP, so efforts directed at high risk households
need not focus on MRSP, but on reducing exposure to the myriad potential pathogens that might be present in or on pets.

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**Virulent Systemic Calicivirus –**

**Signs of this strain of calicivirus may include:**
High fever
Facial and limb edema
Ulceration, crusting and focal hair loss, especially on the face, muzzle and pinnae
Icterus
Dyspnea, DIC and death in severe cases
Death may occur in some cats with minimal preceding signs
Findings on blood chemistry panel may include hyperbilirubinemia, hyperglucosemia and increased CK.
Other signs seen with more typical feline URI may also occur, including nasal and ocular discharge, oral ulceration, anorexia and depression. Unless accompanied by the signs described above, cats showing these typical URI signs should not be considered suspect cases.

**Course of disease:**
The incubation period is between 1-5 days. Cats of all ages, including fully vaccinated cats, have been affected. No other species is known to be affected by this strain of calici virus. There is no known risk to human health. Treatment, as for any virus, is supportive care. It is likely that a significant percentage of cats will continue to shed virus for some time after recovery from clinical signs, as occurs with other strains of feline calici virus. Therefore cats may still be infectious to others following apparent recovery. Confirmed cases should have negative viral cultures before being reintroduced to other cats.

**Transmission:**
Virus is present systemically, and may be shed in feces and in nasal, ocular and oral secretions. The virus can be readily spread by fomites as well as direct transmission. It can be carried for at least several hours on contaminated hands, clothing, instruments, shoes, etc. Droplet transmission is possible over 1-2 meters. Although calici virus may be carried through ventilation systems on dust and hair, airborne transmission over distances greater than a few feet has not been documented in this outbreak.

**Prevention:**
Calicivirus is moderately hardy in the environment, but bleach (5% diluted at 1:32) is effective as a disinfectant. Suspect cases should be housed in strict isolation, with separate equipment, gowns, gloves, caps, and protective footwear used. Possibly contaminated surfaces should be thoroughly cleaned with bleach. Contaminated exam rooms should be cleaned with bleach, held empty for 24 hours, and cleaned again with bleach prior to reuse. If contamination of a home or clinic is suspected, all surfaces should be thoroughly cleaned and disinfected. If surfaces can’t be bleached, the facility or home should be quarantined for 1-2 weeks following cleaning, prior to allowing entry of naïve cats. Heavily contaminated objects such as bedding should be discarded or thoroughly washed. Veterinary staff and others who handle sick cats should change clothing prior to handling healthy cats and at the end of a shift. There has been no further spread of disease documented in clinics that have taken these precautions.

**The VS-FCV vaccine is NOT recommended.** VS-FCV is NOT a disease of household pets. The virus arises anew in each population in which it appears as a mutation from caliciviruses already carried in that group of cats. There is no single genetic mutation that accounts for virulence. No two strains are alike. The vaccines is adjuvanted and may lead to the development of injection-site sarcoma due to chronic site inflammation.

Key points to remember:

1. Neither VS-FCV nor field strain FCV can be diagnosed on clinical signs alone.
2. Diagnosis of calicivirus is further complicated by the fact that calicivirus can be isolated from the oral cavity of as many as 1 in 4 healthy cats, so simply detecting the virus in saliva does not provide a definitive diagnosis - its presence could be completely coincidental. Finding calicivirus in other samples such as serum or tissue is more suggestive that an acute infection is present, but does not rule out the possibility that a co-pathogen such as Bordetella bronchiseptica or panleukopenia is responsible for severe manifestations of disease.

3. So far, no relationship has been discovered between the genetic sequence of a particular strain of calicivirus and the level of virulence. Virulent systemic strains are not particularly closely related to one another, although within each outbreak isolates from individual cats have been similar. Specialized laboratories can only distinguish between strains that are closely related to one another or to the vaccine. Therefore, within a given outbreak it is possible to identify which cats have been infected with that particular strain, but there is no way to say what the virulence may be of any particular strain from an individual cat based on genetic sequence.

4. If you have a single suspect case in your clinic the cat should be carefully isolated and all surfaces should be cleaned with soap and water, followed by application of freshly made bleach solution diluted at 1/2 cup per gallon or potassium peroxymonosulfate (Trifectant®). The area should be allowed to dry thoroughly and the process repeated before any other animals are placed on the surface or in the cage. All suspect cases should be treated symptomatically, the clinic should treat all suspects as being potentially contagious and staff should increase bio-security vigilance.
GENERAL APPROACH TO THE PATIENT WITH CHRONIC NASAL DISEASE

SNEEZING
Sneezing is acute, explosive retrograde expulsion of air and debris from the nasal passages. Occasional sneezing with no other accompanying clinical signs may be a normal finding associated with clearing of the nasal passages of routinely accumulated debris. More frequent or paroxysmal sneezing efforts and those accompanied by significant nasal discharge, or other evidence for intranasal disease, indicate a need for further diagnostic evaluation including nasal cavity examination, radiography, and biopsy.

NASAL DISCHARGE
Viral upper respiratory diseases, allergic rhinitis, and early parasitic infections typically produce serous discharge early in the course of disease but this changes to mucopurulent when secondary bacterial invasion occurs. Mucoid or mucopurulent discharge is more common with chronic disorders such as bacterial rhinosinusitis, lymphoplasmacytic rhinitis, neoplasia, or fungal infection. Fresh blood may intermittently be mixed with the discharge, particularly in the latter two disorders. Frank blood discharging from the nasal cavity (epistaxis) will be discussed below. Unilateral discharge from the nasal cavity suggests a focal problem such as a nasal tumor, fungal infection, chronic foreign body, or tooth root abscess. Bilateral discharge may also occur with fungal and neoplastic rhinitis as extension of these diseases occurs over time. Allergic rhinitis, viral and bacterial rhinosinusitis, lymphoplasmacytic rhinitis, and parasitic rhinitis usually cause bilateral discharge.

EPISTAXIS
Frank hemorrhage (epistaxis) from the nasal cavity is usually the result of trauma (e.g., direct trauma, acute foreign body inhalation) or a bleeding or vascular disorder (e.g., coagulation factor deficiency, DIC, thrombocytopenia, vasculitis).

DISTORTION OR DESTRUCTION OF THE NASAL CAVITY
Internal distortion or destruction of the turbinates occurs because of lysis due to a chronic inflammatory or infectious process (e.g., herpesvirus rhinitis, chronic bacterial rhinosinusitis) and results from the effects of lysosomal enzymes and swelling of affected tissues. Expansion of a mass lesion (e.g., fungal granuloma, neoplasm, nasal polyp) will also cause internal nasal destruction that may involve deviation or destruction of portions of the nasal septum. Lysis of the bones of the skull (e.g., maxillary, facial, nasal bones) is almost always the result of an aggressive expansile disease such as a fungal infection or tumor. Damage to the internal or external bones of the nasal cavity may also result from trauma.

STERTOR/STRIDOR
Stertor or stridor is defined as increased noise associated with respiratory efforts. In addition to the noise noted on respiration, nasal obstruction may prolong the inspiratory phase of respiration if the mouth is closed. Bilateral nasal cavity obstruction may result in open mouth breathing or panting during which time increased nasal sounds will not occur. Stertor/stridor originating in the nasal cavity may be accompanied by nasal discharge, sneezing, snorting, gagging, or reverse sneezing. All of the above are indications for further diagnostic evaluation of the nasal cavity.

OTHER SIGNS
Central nervous system (CNS) signs may rarely occur secondary to an intranasal disease because of direct extension of the process through the cribriform plate into the brain. Occasionally, the signs of brain derangement (e.g., seizures, behavioral changes, ataxia, depression, gait abnormalities) may occur without obvious evidence of intranasal disease. Pain originating in or noticed on palpation of the nasal cavity is usually associated with erosive or expanding mass lesions. Foreign bodies or allergic rhinitis may cause a patient to paw at its face or rub its muzzle. Exophthalmos suggests extension of the problem into the retrobulbar space; ocular discharge may be associated with the primary disease (e.g., viral rhinitis) or be caused by obstruction of the nasolacrimal system. Exudate draining into the nasopharynx or nasopharyngeal foreign bodies or polyps may cause gagging. Finally, lesions that erode or disrupt the hard or soft palate may result in expulsion of food or water from the nose and difficulty eating or drinking. The diagnostic approach previously described should lead to a diagnosis in these patients.

DIAGNOSTIC PROCEDURES
The diagnostic evaluation of the nasal cavity includes radiography or special imaging, rhinoscopy/examination of the nasopharynx, nasal cavity cytology/biopsy, and possibly cultures and/or exploratory rhinotomy.

CHRONIC BACTERIAL RHINOSINUSITIS (Chronic Snufflers)

Introduction
Chronic secondary bacterial rhinosinusitis in cats is a common sequel to infection with feline herpesvirus or calicivirus early in life. Damage to the turbinates and nasal mucosa produces an environment conducive to secondary bacterial colonization and makes effective treatment difficult.

Clinical signs
Clinical signs include a previous history of upper respiratory viral infection (often at an early age), chronic sneezing, stertor, and mucopurulent to occasionally mucohemorrhagic nasal discharge. Signs may be unilateral but are usually bilateral. Epiphora may be present due to blockage of the nasolacrimal system due to mucosal swelling or mucus obstruction. Systemic signs of illness are usually absent in this chronic stage and these cats usually eat well, are active, and are otherwise normal in appearance.

Diagnosis
The diagnosis is assisted by a history of previous viral respiratory disease and is made by
ruling out other more specific and serious disorders. Routine laboratory studies are usually normal. FeLV and FIV testing should be performed to rule out immunosuppression as an underlying cause for chronic infection. Radiographs reveal variable degrees of increased density in the nasal passages and sinuses. Radiographic evidence of turbinate destruction may be present in severe cases but destruction of the overlying bones of the nasal cavity should not occur. The frontal sinuses may appear opaque due to inflamed mucosa and/or accumulation of mucus. Examination of the nasal cavity reveals inflamed, swollen nasal mucosa; cytology and biopsies demonstrate purulent inflammation with bacteria present and thickening and distortion of the nasal turbinates. Therapeutic nasal flushes at the time of this procedure may result in temporary relief of signs but they will recur over time.

**Pathophysiology**

Chronic bacterial rhinosinusitis is usually a consequence of turbinate and respiratory mucosal distortion and damage caused by viral upper respiratory infection that occurs early in life. Occasionally, an older cat will also develop chronic rhinitis following acute respiratory viral infection. Respiratory clearance mechanisms do not function well in this abnormal environment. Mucus and debris accumulate and the normal bacterial flora of the nasal cavity proliferates causing inflammation, additional mucosal swelling, and mucus secretion. The epithelium of the frontal sinuses may be similarly affected or the frontal sinuses may be secondarily involved because swelling within the nasal cavity blocks drainage from the sinuses.

**Treatment**

Treatment is palliative and supportive. The owners should be advised that cure is unlikely and intermittent therapy will be required for many cats. Antibiotic therapy may help reduce bacterial numbers. For most diseases, antibiotic therapy should ideally be based on culture and sensitivity results, however, with these nasal diseases, a swab culture frequently reveals a mixed population which may or may not be representative of the most significant organism(s). Therefore, I find nasal cultures a waste of time and the owner’s money. Azithromycin seems to be a useful drug for some cats. Doxycycline is effective against Mycoplasma (if that is present) as well as other bacteria and has some anti-inflammatory effects that may be useful. Patients with Pseudomonas infection may benefit from systemic carbencillin or nebulized amikacin.

Topical decongestants (e.g. pediatric strength Neosynephrine, Afrin) may provide temporary relief of nasal congestion but can be irritating and will dry nasal passages. Topical antibiotic therapy will not penetrate the congested passages and is generally not beneficial unless delivered with a nebulizer. Humidification with a vaporizer or nebulizer may help the cat expel inspissated secretions from the nasal passages. Surgical exploration with drainage of the frontal sinuses may be useful in some patients to improve respiration.

Recently, maropitant (Cerenia) has been used with some success in some of these cats. This drug has some anti-inflammatory activity that seems to be the basis of its efficacy. It is used at the antiemetic dose and has recently been approved for long term use.

There is also recurring interest in using intranasal Herpes/Calici vaccine as therapy in these chronic sniffer cats. There is one study showing shortening of the duration of clinical signs of acute URI with IN vaccine. I have not found IN vaccine to be helpful in most chronic sniffer cats but it won’t be harmful and may be worth trying because we have little to offer these patients. If it does help, that’s a plus. **Prognosis**

Cats with chronic bacterial rhinosinusitis often have a normal lifespan if the owners do not become discouraged and remove the cat from the household because of contamination of the
environment with debris from the chronic nasal discharge. The prognosis for controlling the condition is guarded because the underlying turbinate and mucosal damage is permanent and the abnormal intranasal environment which lends itself to persistent bacterial recolonization will be a persistent problem.

Fungal Rhinitis: Cryptococcosis and Aspergillosis

Introduction

Cryptococcus neoformans is a saprophytic yeast-like fungus with a worldwide distribution. Infection of the nasal cavity probably occurs by inhalation of the organism from the environment. However, preferential localization in the nasal cavity following intravenous inoculation has been demonstrated for some strains of the Cryptococcus. Aspergillus can also affect the nasal cavity of cats but is very rare. There is no apparent age, breed, or sex predilection. Concurrent retrovirus infection (FeLV or FIV) is not predisposing for acquiring the infection but cats with either of these two immunosuppressive diseases are likely to have more disseminated disease and will be more resistant to treatment.

Clinical signs

Clinical signs of nasal fungal infection include unilateral or bilateral, mucopurulent to hemorrhagic nasal discharge and sneezing. Fever is usually absent, however systemic signs of illness may include anorexia, weight loss, and lethargy. With Cryptococcus, Regional lymphadenopathy and skin granulomas are present in about 30% of affected patients. Ocular involvement including chorioretinitis, optic neuritis, retinal detachment, and anterior uveitis occurs in some animals. Central nervous system (CNS) spread of the organism can result in seizures, ataxia, circling, paresis, paralysis, or amaurotic blindness. Disseminated disease with involvement of the lung, bone, and kidney is found in a few patients.

Diagnosis

Routine hematologic studies and urinalyses are usually normal. Biochemistry profiles may reveal hyperglobulinemia consistent with chronic antigenic stimulation but are usually otherwise normal. In patients with CNS involvement, cerebrospinal fluid analysis will demonstrate increased cellularity, neutrophilia, and an elevated protein content. FeLV antigen and FIV antibody tests are usually negative in cats with only nasal cavity involvement; cats with disseminated cryptococcosis are more likely to have concurrent retrovirus infection. Radiographic studies usually reveal evidence of unilateral or bilateral soft tissue density in the nasal cavity early in the course of infection. Destruction of the nasal turbinates and overlying nasal bones may occur by expansion of the fungal granulomas as the disease progresses. The diagnosis can be confirmed by identifying the Cryptococcus organisms on cytologic or biopsy specimens. Organisms are usually numerous in cytologic specimens and are easily detected with routine Wright–s Geimsa hematologic stains. Special fungal stains may be required to find organisms in histologic specimens. The latex cryptococcal antigen test (LCAT) is a sensitive method to detect the polysaccharide cryptococcal capsular antigen in serum and other body fluids. The test is 95% sensitive and 100% specific and can be used to confirm the diagnosis as well as monitor the efficacy of therapy. Because the organism is ubiquitous, culturing and serologic testing for Aspergillus is prone to false positive results. The diagnosis of aspergillosis is made by identification of the fungal hyphae on cytologic or histopathologic specimens.

Pathophysiology
The expansion of the pyogranulomatous reaction to the fungal organisms causes both soft tissue and bony destruction within the nasal cavity. Secondary bacterial infection is often superimposed on the fungal disease. The development of subretinal cryptococcal granulomas causes elevation of the retinal epithelium (serous retinal detachments) and chorioretinitis. Further systemic dissemination of the organism is more common in cats with concurrent retrovirus infection or those who have been treated with corticosteroids. Fungal granulomas can be found in the lymph nodes, skin, lung, CNS, or other organs in these patients.

**Treatment**

Itraconazole (10 mg/kg PO q24h) or fluconazole (5.0-10 mg/kg PO q12-24h) are very effective in eliminating nasal cryptococcal infection in most affected patients. Treatment may be required for 6-12 months depending on the severity and extent of disease. Using terbinafine with either ITZ or FLU may enhance efficacy and shorten treatment time. Cryptococcal antigen titers can be followed to monitor the progress of treatment. A progressively decreasing titer after 1-2 months of treatment is considered good evidence of improvement. Cryptococcal antigen titers may remain positive at a low level for long periods of time despite successful treatment. Good clinical resolution of disease should be a factor in determining when antifungal treatment can and should be discontinued. Fluconazole is the drug of choice for patients with CNS involvement because of its excellent penetration into the CSF. However, the prognosis for recovery from this form of infection is guarded. Amphotericin B (systemic IV liposomal formulation or SQ deoxycholate) can be used in resistant cases. *Aspergillus* may also respond to the azole antifungals (ITZ), however, topical infusion with clotrimazole (only in animals with an intact cribriform plate) is more likely to give beneficial results.

**Prognosis**

The prognosis for nasal cryptococcosis and aspergillosis in patients without retrovirus infection is good with appropriate treatment. The prognosis for patients with concurrent retrovirus infection or CNS cryptococcosis is guarded to grave.

**NASOPHARYNGEAL POLYPS**

**Introduction**

Nasopharyngeal inflammatory polyps are an unusual feline disorder. These masses originate in the middle ear and extend through the auditory tube into the nasopharynx or external ear canal. The initiating cause of the polyps is unknown, however, calicivirus has been isolated from some cats. Affected cats are usually less than 6 years of age; there is no breed or sex predilection.

**Clinical signs**

Clinical signs include stertor, stridor, occasional sneezing, and occasional serous or mucoid nasal discharge. Difficulty swallowing and a change in voice occur in some patients. Anorexia, weight loss, and malaise may occur because of difficulty eating and drinking.

**Diagnosis**

Laboratory parameters and examination of the nasal cavity are usually normal. A nasopharyngeal mass can be seen on radiographs or during examination of the nasopharynx. Radiographic or CT/MRI examination of the tympanic bullae may reveal increased soft tissue density on the side associated with the polyp.

**Pathophysiology**
Nasal stertor and stridor are caused by obstruction to airflow caused by the presence of the polyp in the nasopharynx. In some patients, the polyp may be sufficiently large or hang ventrally far enough to partially occlude the esophageal opening or larynx. This results in gagging, difficulty in swallowing, or forceful and severely compromised inspiratory efforts.

**Treatment**
Surgical removal of the polyp from the nasopharynx by blunt dissection and gentle traction will alleviate signs of respiratory obstruction and may be curative. Because the inflammatory process originates from the middle ear, exploration and curettage of the tympanic bulla may be necessary to prevent recurrence in some patients.

**LYMPHOPLASMACYTIC RHINITIS**

**Introduction**
Although this condition has not been well defined, the lymphoid and plasma cell infiltrates that characterize lymphoplasmacytic rhinitis suggest that there is an immune-mediated basis for this disorder.

**Clinical signs**
Clinical signs include sneezing, stertor, nasal congestion, and unilateral or bilateral nasal discharge. The nasal discharge is usually mucopurulent in nature, however, serous, mucoid, and hemorrhagic discharge has been reported in patients with this condition.

**Diagnosis**
Routine laboratory studies are unremarkable. Radiographs demonstrate increased fluid density, usually confined to the rostral half of the nasal cavity. Lysis of the turbinates and/or the vomer is present in some patients. Examination of the nasal cavity may be apparently normal or may reveal mucosal edema, erythema, or ulceration. Cytologic examination usually demonstrates secondary bacterial infection. Biopsies reveal lymphoplasmacytic infiltration into the nasal mucosa and submucosa. It is important that adequate and representative biopsy specimens are collected because a lymphoplasmacytic infiltration has also been reported in association with nasal aspergillosis and nasal tumors.

**Pathophysiology**
Mucosal swelling due to lymphocyte and plasma cell accumulation results in obstruction to airflow and stimulation of mucus secretion. Secondary bacterial infection is often superimposed on the immunologic disorder because of altered clearance mechanisms within the nasal cavity producing the typical mucopurulent discharge seen in this disease. The mucosal infiltrates also render the tissue more friable than usual so that spontaneous hemorrhage can occur with mild trauma or sneezing.

**Treatment**
Immunosuppressive doses of corticosteroids (e.g., oral prednisone at a dose of 2.2 - 4.4 mg/kg/day) are the initial treatment of choice for idiopathic lymphoplasmacytic rhinitis. High dose corticosteroid therapy should be maintained for at least 2-4 weeks until significant improvement is obtained. The dose can then be tapered slowly by halving the dose every 2-3 weeks to maintain remission of clinical signs. Ultimately, the corticosteroid dose should be reduced to an alternate day schedule to minimize side effects. Inhaled steroids (Aerokat or Nebulair chamber) may also be effective in some patients.

**Prognosis**
The prognosis for improvement of patients with lymphocytic plasmacytic rhinitis is fair; treatment may be needed for months to years.

**ALLERGIC RHINITIS**

**Introduction**

Allergic rhinitis is a poorly described entity in cats. Allergy usually develops in middle-aged to older animals, however allergic rhinitis may occur in a patient of any age. The onset of clinical signs may be acute if there is an abrupt environmental change (e.g. new rugs, household cleaners, change of location). More commonly signs develop slowly over time. Intermittent, seasonal occurrence is present in some affected patients.

**Clinical signs**

Clinical signs are usually similar to bilateral chronic bacterial rhinosinusitis, however, epistaxis without chronic discharge has been described. Concurrent dermatologic signs of allergy including pruritus and face rubbing or pawing occur in some individuals.

**Diagnosis**

Peripheral eosinophilia has been observed occasionally, however, laboratory studies are usually unremarkable. Radiographic changes include variable degrees of increased fluid density in the nasal cavities. There is usually no evidence of nasal turbinate destruction unless the condition is chronic. Cytologic and biopsy findings are usually similar to chronic rhinosinusitis, however, eosinophils may be the predominant cell type.

**Pathophysiology**

Inhalant allergens are the most common underlying cause for allergic rhinitis although food allergens have also been implicated in this disease. Mucosal swelling and eosinophilic cellular infiltration produce upper respiratory obstruction, sneezing, and serous nasal discharge. If secondary bacterial infection is present, clinical signs are usually more severe and the nasal discharge will be mucopurulent in character.

**Treatment**

An attempt should be made to discover the sensitizing environmental allergen(s). If there is a history of an environmental change, it may be possible to remove the potentially offending object or chemical from the environment to observe whether the patient=s clinical signs improve. The accuracy of feline intradermal skin tests or serum ELISA tests for identifying sensitizing inhalant allergens is controversial. Skin or blood testing for dietary allergens is not reliable and the diagnosis of food allergy requires a response to limited antigen diet trials. Depending on the allergens suspected of causing hypersensitivity, treatment for allergic rhinitis might include an elimination diet trial, skin testing with possible hyposensitization, or corticosteroid therapy with prednisone at a tapering dose as previously described or inhalant steroids as for lymphoplasmacytic rhinitis.

**NEOPLASTIC DISEASE**

**Introduction**

Intranasal tumors occur most commonly in middle aged to older animals. Epithelial tumors (carcinoma, adenocarcinoma) are the most common neoplasms of the nasal cavity of cats. Mesenchymal tumors (sarcomas) are uncommon with the exception of lymphosarcoma. This form of lymphoma may be restricted to the nasal cavity but may also involve regional lymphoid
tissue or distant organs or lymph nodes. Rare tumors of the nasal cavity include plasmacytoma and mast cell tumor.

**Clinical signs**

The clinical signs associated with nasal neoplasms are similar to chronic rhinosinusitis. Signs are often initially unilateral but progress to involve both sides of the nasal cavity over time. The nasal discharge is often mucopurulent due to secondary bacterial infection and occasionally contains fresh blood. There may be erosion of the tumor through the facial bones, distortion of the face, exophthalmos, or pain on opening the mouth depending on the duration, location, and extent of tumor invasion.

**Diagnosis**

Routine laboratory studies are often unremarkable. FeLV antigen or FIV antibody tests may be positive in cats with lymphoma. Radiographs usually demonstrate varying degrees of increased intranasal density accompanied by bone destruction, distortion, or erosion. Computed tomography or MRI, if available, are very sensitive tools for evaluating the true extent of tumor involvement. Examination of the anterior nasal cavity may demonstrate a tissue mass obstructing the nasal passage(s). Posterior rhinoscopy as previously described may demonstrate a mass lesion in the posterior nasopharyngeal region. Exfoliative cytology and biopsy are often diagnostic for neoplastic lesions. However, because there is often inflammation associated with the neoplasm, it is critical that a sufficiently large and representative sample of tissue be examined before a diagnosis of neoplasia can be confirmed or excluded. Exploratory rhinotomy and biopsy may be required to make a definitive diagnosis of neoplasia in some patients. Although metastasis from nasal neoplasms is usually slow, aspiration cytology or biopsy should be performed on lymph nodes draining the affected area and thoracic radiographs are recommended prior to initiating therapy.

**Pathophysiology**

Neoplastic diseases exert their destructive effects on the nasal cavity by direct involvement/invasion of or compression of tissues of the nasal cavity and surrounding anatomic structures. Extranasal signs and deformation of the face may also result from compression by the expanding tumor mass or extension of the tumor into extranasal structures.

**Treatment**

The efficacy of treatment for nasal neoplasms is variable and depends on the tumor type, extent of involvement in the nasal cavity, sinuses, retrobulbar space, and overlying bone, the presence of regional or distant metastases, and the presence of concurrent diseases (e.g., retroviral infection in cats). Unfortunately, because of the slow development of clinical signs and the limitations of early recognition, nasal neoplasms are usually in an advanced stage of locally invasive disease by the time of diagnosis. Generally, surgical excision alone is unlikely to provide a substantial increase in survival time. Surgical debulking followed by orthovoltage or megavoltage radiation therapy, or $^{192}$ Iridium brachytherapy with or without surgery cannot be expected to cure disease but may provide improved survival and quality of life with substantial temporary relief of clinical signs. Radiotherapy appears to provide a longer disease-free interval for mesenchymal tumors than epithelial tumors. Lymphoma is very radiosensitive and excellent long term control of disease may be achieved by radiation therapy alone. Complications of radiotherapy include radiation induced ocular, dermal, and CNS damage. These effects are usually not severe and are easily managed. Brachytherapy may be associated with necrosis of the soft tissues, hard palate, or nasal bones. Chemotherapy has not been uniformly successful for the
treatment of nonhematopoietic neoplasms of the nasal cavity but may be effective for nasal lymphoma. Permanent tracheostomy has been used as temporary palliative therapy to improve respiration and quality of life in some patients with untreated or untreatable nasal tumors. Piroxicam at 0.3 mg/kg PO q48h may provide some pain relief and this agent also has anti-neoplastic effects against some of the epithelial tumors.

**Prognosis**
Survival times for patients with nasal tumors that are not treated or receive only surgical debulking, cryosurgery, or immunotherapy average from 3-5 months. The approximate median survival time for cats with nasal tumors that receive radiation therapy is 20 months.

**NASAL FOREIGN BODY**

**Introduction**
A variety of plant and foreign materials have been found lodged in the nasal cavity. These most commonly include plant awns (Afoxtails®), seeds, twigs, and grass blades.

**Clinical signs**
Clinical signs are caused by trauma and irritation from the foreign material and include acute paroxysms of sneezing, snorting, face rubbing, and unilateral nasal discharge or epistaxis. Grass blades extending into the nasopharynx may also cause sporadic gagging or choking. As the foreign body becomes lodged deeper in the nasal cavity, signs become more intermittent and secondary bacterial infection occurs due to mucosal damage and inflammation.

**Diagnosis**
Routine laboratory parameters are usually normal. Radiographs may be normal, demonstrate a metallic foreign body, or demonstrate unilateral increased soft tissue density. The diagnosis is made by ruling out other disorders and by locating and removing the nasal foreign body. The posterior nasopharynx should be examined thoroughly because grass blades may be discovered above the soft palate in this area. Occasionally exploratory surgery is required to diagnose and remove deeply lodged foreign bodies.

**Pathophysiology**
Acutely inhaled foreign bodies cause direct trauma to and irritation of the nasal mucosa. If not expelled by sneezing or removed, the foreign body usually becomes lodged deeper in the nasal chambers. Depending on its size and location, pressure necrosis of soft tissue or bony structures may occur. Chronic irritation and inflammation causes continued intermittent episodes of sneezing, discomfort, and provides an environment conducive to secondary bacterial infection.

**Prognosis**
Removal of the foreign body is curative, however, secondary infections due to chronic intranasal damage may persist.

**NASAL TRAUMA**

**Introduction**
Nasal trauma is most commonly associated with vehicular accidents or falling from a height (Ahigh rise syndrome®).

**Clinical signs**
The primary clinical sign is epistaxis that may be associated with obvious facial damage
and maxillary or hard palate fractures. Signs of traumatic injury to other organs or systems may be present in some patients. Concurrent pneumothorax is a common finding in animals that have fallen from a height. Fractures of the nasal or frontal bones may produce local subcutaneous emphysema.

**Diagnosis**
Routine laboratory studies and nasal radiography are not usually necessary because the cause of the nasal hemorrhage is obvious.

**Pathophysiology**
Blunt trauma usually causes superficial tearing of the nasal mucosa or the minor disruption of turbinates. The small superficial mucosal vessels bleed profusely but clots form quickly if coagulation function is normal. Local subcutaneous emphysema is produced by air dissecting into the subcutaneous tissues from the nasal cavity through fractures in the overlying bones.

**Treatment**
Epistaxis from trauma is usually self-limiting and treatment is not usually necessary. In patients with severe or uncontrollable hemorrhage, cotton swabs dipped in dilute epinephrine solution (see previously for nasal biopsy) can be applied to the nasal mucosa or the nasal cavity can be packed with sterile gauze. Acepromazine (0.05-0.01 mg/kg IV) can also be used to assist in reducing acute hemorrhage. However, because the mechanism of action of this drug is to reduce blood pressure, it should not be used in trauma patients that may already have or may develop significant hypotension.

**Prognosis**
The prognosis for patients with traumatic nasal injury is excellent. Hard palate fractures usually seal without intervention but surgical correction may be required if an oronasal fistula persists after healing.

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**DENTAL DISEASE**

**Introduction**
Oronasal fistulae, severe periodontitis, and apical abscesses may cause chronic nasal discharge. Older animals are more commonly affected.

**Clinical signs**
Clinical signs include chronic unilateral or bilateral nasal discharge that may be serous, mucopurulent, or hemorrhagic. Patients with large oronasal fistulae may have food particles mixed with the nasal exudate. A fetid odor may be noticed around the mouth or nasal cavity. Epiphora and swelling below the eye is occasionally present in cats with apical abscessation of the fourth upper premolar. Oral examination may reveal an obvious dental or periodontal problem. However, small oronasal fistulae, apical abscesses, or retained tooth roots may not be identified on gross inspection of the oral cavity.

**Diagnosis**
There are no specific laboratory findings for this disorder and results are usually consistent with the patient's age. Routine nasal radiographs may demonstrate rhinitis but oblique views or dental radiographs may be needed to thoroughly evaluate the dental arcades for apical abscesses, retained tooth roots, or bone lysis. Nonscreen, #4 intraoral film will provide exquisite detail for these studies. Nasal biopsy will help rule out more serious diseases if radiographs and careful oral cavity examination are equivocal for dental disease.
Pathophysiology
Disruption of the periodontal attachments allows bacteria to colonize along the tooth root and create an abscess pocket at the tooth root apex. Pressure necrosis and lysosomal enzymes in the expanding abscess cause bone lysis and drainage of exudate into the nasal cavity. Naturally-occurring loss or veterinary extraction of affected teeth may leave a permanent stoma between the oral cavity and nasal chambers (oronasal fistula) creating a pathway for food debris and bacteria to enter the nasal cavity.

Treatment
Treatment involves dental prophylaxis and specific treatment for apical abscesses. Apical abscesses may be treated by extraction of the affected tooth, or periapical curettage or standard endodontic drainage followed by packing of the root canal with gutta percha. Broad spectrum antibiotic therapy should be administered for several weeks following these procedures. Preexisting oronasal fistulae or those created by dental extraction should be surgically closed by primary closure or sliding flap techniques to eliminate communication of the oral cavity with the nasal chambers.

Prognosis
The prognosis for most patients with nasal disease secondary to dental disease is very good with appropriate treatment. Patients with large or chronic oronasal fistulae have a more guarded prognosis and it may be very difficult to completely eliminate signs of nasal disease.

ANATOMIC ABNORMALITIES
Stenotic nares are common in brachycephalic breeds of animals and the condition is often associated with an elongated soft palate. This problem is seen occasionally in Persian and related cats. The stenosis results in noisy respiration and mouth breathing. Surgical correction by removal of the dorsolateral nasal cartilages is curative.

Anatomic abnormalities of the hard palate (e.g., cleft palate) or soft palate (e.g., unilateral or bilateral hypoplasia) can result in nasal discharge and food or water being expelled from the nares. These defects are uncommon in cats and signs are usually present from an early age. Careful examination of the oral cavity usually reveals the abnormality. Laboratory and radiographic studies are not usually performed in these patients. Treatment consists of surgical correction of the anatomic abnormality.
Causes of Anemia

The basic pathophysiologic causes of anemia in the cat are not significantly different than those in other species, although specific disorders may be unique to the cat. Mechanisms resulting in anemia primarily include: Failure of production and excessive loss (bleeding disorders, excessive destruction). Sequestration in the spleen rarely, if ever, occurs in cats except with Mycoplasmosis (hemobartonellosis). Excessive utilization is not a feature of anemia but can be a cause of other cytopenias (thrombocytopenia, leukopenia).

Clinical and Physical Findings

Historical signs of anemia include lethargy, depression, and anorexia (this is usually a late sign). Dyspnea and tachypnea may result from low oxygen carrying capacity with hypoxemic stimulus of the respiratory control center. Pica, most often manifested as licking bricks, cement, eating kitty litter, and the like is a surprisingly common historical complaint from owners or may be observed by finding “gravel” in the stomach or intestinal tract on radiographs. Physical findings include pale mucous membranes and tachycardia. Rapid breathing or apparent dyspnea reflects hypoxemia. Other significant physical findings might include: icterus, petechia, or overt evidence of clinical hemorrhage depending on the underlying cause of the anemia.

Laboratory Evaluation

A complete blood count should be performed and the reticulocyte count should be measured. Clinics using in-house hematology equipment for hematologic analysis should always at least make a blood film for cytologic examination. Special staining techniques may be needed to examine films for hemoparasites. Additional internal organ system evaluation should include a serum biochemistry profile, urinalysis, and other studies as indicated by the signalment and history. All anemic cats, regardless of vaccination status should be tested for the presence of feline leukemia virus antigen and feline immunodeficiency virus antibody. Additional laboratory evaluation may be needed on a case by case basis. Fungal serology for histoplasmosis and blastomycosis is usually falsely negative, is not of diagnostic benefit, and may lead the clinician to incorrectly rule out these diseases. Urine antigen testing is much more accurate (MiraVista Labs). Feline coronavirus serology (this is NOT an FIP test) is of little use as a screening or diagnostic test. Many causes of anemia in the cat will have an immune component but true primary immune-mediated hemolytic anemia is rare compared to dogs. Therefore, beware of overinterpreting the results of a positive Coomb’s test in this species.

Developing a Differential Diagnosis for Anemia

In developing a differential diagnosis for the anemia in a specific patient, it is most helpful
to determine first if the anemia is responsive or non-responsive. Then proceed to try to assess whether the problem is most likely a primary hematologic one (uncommon) or whether the anemia is secondary to a more generalized specific disorder and should be addressed as part of the whole rather than as a separate problem.

**Responsive anemia**

The most common disorders in this category cause hemolysis and include hemoparasites (mycoplasmosis, cytauxzoonosis), and immune-mediated hemolytic anemia.

Mycoplasmosis was formerly called hemobartonellosis but the organisms have been reclassified: *Mycoplasma haemofelis* (larger, more pathogenic), and *Candidatus Mycoplasma haemominutum* (smaller, less pathogenic). These organisms may be seen on routine hematologic staining but PCR testing is more sensitive. Hemoparasitic Mycoplasmas may be present in asymptomatic cats and cats with other diseases so be careful not to overinterpret a positive test result for these organisms. Mycoplasmosis is treated with doxycycline at 10 mg/kg PO q24h for 3 weeks.

*Cytauxzoon felis* is a tick-borne hemoparasite found in east Texas, Louisiana, Oklahoma and more recently in other southern states. The usual host is the bobcat and domestic cats are only accidentally infected. In domestic cats, cytauxzoonosis is often rapidly progressive and fatal. There is currently no effective treatment for this parasite.

A recent study on immune-mediated hemolytic anemia (IMHA) in cats demonstrated that this tends to be a disease of middle-aged cats with Himalayans overrepresented. Clinical signs are typical for anemia. Prednisone or prednisolone was the cornerstone of therapy with an average dose of 15 mg/cat/day required to induce remission. Additional immunosuppressive therapy was needed for approximately 1/3 of affected cats. Response to therapy was slow in some cats and treatment may be required for months before hematocrit levels return to the normal range. Other causes of hemolysis in cats include toxins, chemotherapeutic agents and other drugs, onions, lily bulbs and other plants, porphyria (rare), and hypophosphatemia. A responsive anemia may also be seen 5–7 days after acute blood loss.

**Non-responsive anemia**

Anemia of chronic systemic inflammation or infection is the most commonly encountered type of non-responsive anemia in the cat. Renal insufficiency producing EPO deficiency is another commonly encountered problem. Myelophthisic disease or myeloproliferative disease is not likely to produce only anemia but generally causes a pancytopenia. Localized bone marrow infection or inflammation may cause very specific cell line arrest or destruction. This is particularly true with sequestered FeLV infection in the marrow which causes maturation arrest in the RBC line. Other diseases such as fungal (e.g. histoplasmosis) or granulomatous disease (e.g. systemic mycobacteriosis) may cause variable cytopenias. Bone marrow suppression may be caused by drugs (chemotherapeutic agents, griseofulvin {especially in FIV+ cats}, methimazole, chronic TMPS, and others). Chronic blood loss with resulting iron deficiency, B12 deficiency due to severe intestinal malabsorptive disease, and folate deficiency (TMPS), can also produce non-
responsive anemia. A response to acute blood loss will not be seen for 5-7 days so this may also appear to be a non-responsive anemia. Pure red cell aplasia (PRCA) is a disorder most commonly seen in younger cats. There is specific immune-mediated attack directed at the RBC precursors in the bone marrow. Other marrow elements are normal. Hematology demonstrates a severe, non-responsive anemia with other cell lines normal. Diagnosis is made by demonstrating absence of RBC pre-cursors in the bone marrow. Because this is an IM disease, the treatment is the same as previously described for IMHA.

Supportive Therapy for Anemic Cats

Obviously, the object of diagnostic evaluation is to determine the cause of the anemia and, if possible, treat that specifically. However, some oxygen carrying support may be needed at least in the short term for many patients. Blood transfusion is generally a very safe procedure for both the donor and the recipient. In a study of blood transfusion in 91 cats, Type A blood was the most common type in both donors (129/134) and recipients (86/91). Type B was found in 5/134 donors and 4/91 recipients. One recipient was type AB and this cat was given type A blood. Blood typing was performed in all cats and cross-match in most prior to transfusion. Transfusion reactions were noted in only 2 cats, the signs of which included fever, tachypnea, and hyperbilirubinemia. Both cats survived. The expected calculated increase in PCV was not seen in some cats post-transfusion. This was believed due to continuing active blood loss, volume of distribution changes, and lysis of transfused cells. Oxyglobin (bovine Hb) now available from Dechra, is another approach to increasing oxygen carrying capacity of the blood without the attendant risk of blood cell incompatibility. However, adverse events to Oxyglobin use are relatively common and include pulmonary edema, pleural effusion, vomiting, and neurologic signs. This is most likely due to volume overload and the rate of infusion.

Bone Marrow Aspiration

Bone marrow aspiration for cytology, IFA FeLV marrow evaluation, and/or marrow core histopathology is a relatively easy, reasonably non-invasive, and often very helpful technique for evaluation of anemic and other cytopenic patients. The proximal humerus is my personal preference as the site for both aspiration and core sampling. This area is readily accessible and has a large amount of cancellous bone containing marrow elements.

Specific bone marrow aspiration needles (16-18 ga Rosenthal type) should be used for aspiration. A Jamshidi biopsy needle (4 inch, 8 ga) is used for core sampling. Feline patients are best anesthetized for this procedure although it can be done with sedation and local anesthesia in debilitated or fragile patients.

The marrow aspiration/biopsy site should be clipped and surgically prepared. A scalpel blade is used to make a nick in the skin at the aspiration needle entry site. The needle is driven into the cancellous bone and the stylet is removed for aspiration. After removal of 1-2 ml of marrow, the sample is placed in a Petri dish containing 1-2 ml of EDTA solution to prevent coagulation.
Marrow spicules are picked up and placed on a glass slide, then compressed with another slide to make smears for cytologic examination. Several unstained marrow slides should be retained for possible submission for FeLV IFA is the slide evaluation is suggestive of retroviral disease. Other special stains for fungi and mycobacteria can also be performed on these unstained slides if necessary.

A marrow core should be collected from all patients with multiple cytopenias or if the marrow aspiration appears non-diagnostic or blood diluted. The core may be collected from the same general location (but not from the same “hole”) from which the aspiration was performed. Following core removal, the specimen can be rolled on a clean glass slide before being placed in formalin for histopathologic evaluation.

Suturing the skin after marrow collection is usually not necessary. Any local bleeding is usually easily controlled with gentle pressure. Surprisingly, most cats have little discomfort after bone marrow collection procedures although 24 hours of analgesic administration is recommended to minimize patient discomfort.

Case Study: – If time permits
Biosecurity should be considered as an essential aspect of patient care. Biosecurity has been defined as work of strategy, efforts and planning to protect human, animal and environmental health against biological threats.\(^1\) Biosecurity practices include the prevention of the introduction of infectious disease, controlling the spread of diseases within the population and or environment and disinfection of surfaces and fomites.\(^2\) One Health is a concept that relates to the intricate relationship between animal, human and environment with regards to health and disease. It also highlights the collaboration of multiple disciplines at different levels in order to attain optimal health.\(^3\) Biosecurity is built on three pillars that is human, animal and environment, these elements are also shared by the One Health concept and biosecurity should be implemented with One Health in mind. Collaboration and cross-learning from different disciplines in order to control the spread of pathogenic microorganisms is paramount and key for innovation and success. The three pillars of biosecurity / One Health have an intricate association among them and the microorganisms, thus when implementing strategies to control infectious diseases the intricate interrelation of the aforementioned elements must be kept in mind.

Infectious pathogens are a serious risk continuously challenging human and animal health. In human medicine the improvements in medicine, public health and social standards have led to a paradoxical increase in exposure and susceptibility to pathogens.\(^4\) In veterinary medicine the increased technological and scientific advances have allowed the veterinary care team to treat sicker patients, whom in turn are more prone to acquire infectious diseases. Therefore as in human medicine, veterinary medicine must see infectious disease prevention as a key component patient care. Furthermore animal caregivers are at a higher risk of acquiring zoonotic diseases, thus protecting personnel should also be included as part of the overall biosecurity plan. In veterinary hospitals nosocomial infections are not just a patient care issue. Outbreaks of infectious pathogens can have a significant impact on normal operations of the hospital by having a negative impact on revenue, client confidence, public image and staff morale.\(^2\) Furthermore, as stated above, some infectious agents are zoonotic.\(^2\) Therefore the goals of a properly implemented biosecurity protocol are to improve quality of animal care that is to reduce infection rates, morbidity and mortality; improve personnel safety and reduce costs.

Health-care associated infections (HAIs) in human hospitals impose significant economic burden to the health care system.\(^5\) In the US over 1.7 million patients per year suffer HAIs and one-third or more are believed to be preventable.\(^6\) The annual direct hospital costs of treating HAIs in the US ranges from $28.4 billion to $45 billion.\(^5\) Prevention of HAI is an example of value based medicine, where the goals of improving quality and decreasing cost is synergistic.\(^6,7\) In the US HAIs are subject to mandatory reporting and are linked to reimbursement, thus reducing payment for HAIs which in turns incentivizes providers to improve infection prevention measures.\(^6\) Furthermore HAI prevention may also contribute to the reduction of antimicrobial resistant infections.\(^6\)

The prevalence of hospital associated infections in privately owned veterinary hospitals is unknown.\(^8\) A recent survey of veterinary teaching hospitals revealed that 82 % of the institutions had reported an outbreak of an infectious disease. *Salmonella enterica* was the leading cause of
outbreaks (65 %) followed by methicillin resistant *Staphylococcus aureus* (MRSA) (42 %). Fifty-eight percent of these institutions had to restrict patient admissions while 32 % had to close in order to control the outbreak.  

Outbreaks in veterinary teaching hospitals reported loses $250,000 to $4.12 million per outbreak, these costs were likely conservative and much greater in the long term since it does not account for intangible loses.  

In a recent equine consensus the authors concluded that veterinarians and managers need to be aware that there is a recognizable standard of care with regards to infection control. Meaning that measures geared towards minimizing the spread of contagious infectious diseases and education must be part of the care provided by veterinarians to their patients and clients. Not meeting these standards would constitute malpractice and represents a failure to meet the ethical responsibilities to patients and clients. Even though the focus of this report was on equine, its relevance is also applicable to small animal practices and the veterinary care team. I would highlight the point of biosecurity education as paramount not just for the animal care team but for animal owners also.

The implementation of biosecurity strategies in order to challenge the *status quo* requires an organizational cultural change for it to be successfully embraced by the animal care team. However change in the organizational culture is always difficult, but not impossible. Leadership, teamwork and education are key concepts that if implemented correctly will lead to smoother implementation, thus higher chances of success.

When implementing a biosecurity program it is important to understand that it must be tailored to the particular situation, disease risk and level of risk aversion that is consider appropriate for each veterinary practice.
References

Understanding disinfectants use in the workplace  
Lucas Pantaleon DVM MS DACVIM MBA

Biosecurity can be defined as the strategies, efforts and planning to protect human, animal and environmental health against biological threats. Biosecurity is a broad term and encompasses the interrelation of human, animal and pathogens with the environment. Thus environmental disinfection is an important element to help maintain the health of human and animals by keeping the environmental bioburden in check. However important, disinfectants are only one part of a broad biosecurity protocol implementation.

The Center for Disease Control guidelines describes disinfection as a process that eliminates many or all pathogenic microorganism, except spores, on inanimate objects. Objects and surfaces are normally disinfected with liquid chemicals (disinfectants). It is important to keep in mind that there are factors that could influence the efficacy of disinfectants. Some of these factors are: prior cleaning, organic and inorganic load, type and level of microbial contamination, concentration and contact time of the disinfectant, physical nature of the object, presence of biofilm and temperature and pH of the disinfection process.

When developing an infection control program, both veterinarians and the animal care team need to understand the important role that disinfectants play. It would be inadequate to base the selection of disinfectants solely on cost or tradition. Disinfectants should primarily be selected on safety and efficacy, since the use of the wrong formulation for an inappropriate contact time generates a false sense of security risking the spread of pathogenic organisms over a wider area during the disinfection process. Selecting the optimal disinfectant should be based on five key criteria as outlined on the table below.

<table>
<thead>
<tr>
<th>Properties</th>
<th>Questions to ask</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kill Claims</td>
<td>Is the product effective against the most prevalent pathogens, including those that</td>
</tr>
<tr>
<td></td>
<td>Cause hospital infections?</td>
</tr>
<tr>
<td></td>
<td>Cause outbreaks?</td>
</tr>
<tr>
<td></td>
<td>Are of concern in your facility?</td>
</tr>
<tr>
<td>Kill and wet contact times</td>
<td>How fast the product kills the pathogens of concern?</td>
</tr>
<tr>
<td></td>
<td>Does the product keep surfaces wet for the required kill time?</td>
</tr>
<tr>
<td>Safety</td>
<td>Does the product have an acceptable toxicity rating?</td>
</tr>
<tr>
<td></td>
<td>Does the product has an acceptable flammability rating?</td>
</tr>
<tr>
<td></td>
<td>What is the personal protection equipment required?</td>
</tr>
<tr>
<td></td>
<td>Is the product compatible with surfaces at your facility?</td>
</tr>
<tr>
<td>Ease of use</td>
<td>Is its odor acceptable?</td>
</tr>
<tr>
<td></td>
<td>Does it have an acceptable shelf life?</td>
</tr>
<tr>
<td></td>
<td>Does the product comes in formats to meet your facility’s needs (i.e. liquids, concentrate, wipes, and multiple sizes)?</td>
</tr>
<tr>
<td></td>
<td>Does the product work in the presence of organic matter?</td>
</tr>
<tr>
<td></td>
<td>Does the product clean and disinfect in a single step?</td>
</tr>
<tr>
<td></td>
<td>Are directions for use simple and easy to understand?</td>
</tr>
<tr>
<td>Other factors</td>
<td>Does the supplier offer training and education?</td>
</tr>
<tr>
<td></td>
<td>What type of customer support is offered?</td>
</tr>
<tr>
<td></td>
<td>Is the overall cost acceptable?</td>
</tr>
<tr>
<td></td>
<td>Can the product be the standardized disinfectant use in your facility?</td>
</tr>
</tbody>
</table>
Selecting a disinfectant is one component for effective disinfection. The second component is the practice, which encompasses correct product application covering all surfaces to be disinfected, training personnel and following manufacturer’s label instructions.  

The importance of cleaning and disinfection stems from the fact that pathogenic microorganisms survive on environmental surfaces and fomites, some for prolonged periods of time. The major sources of environmental contamination are the patient’s endogenous flora and the hands from healthcare workers. Studies have shown that contact with contaminated environmental surfaces was just as likely to contaminate hands as direct contact with a patient. Thus improving cleaning and disinfection practices, including the use of a daily cleaning disinfectant product, reduces hospital acquired infections in the healthcare setting, improper cleaning and disinfecting can play a major role in spreading pathogenic microorganisms. 

Not only will properly implemented cleaning and disinfection protocols lead to a decrease in the environmental bioburden, but also keep the environment cleaner, free of odor and pathogens making it a better place for animal caregivers to work and animals to reside in a comfortable safe place.

Using newer generation disinfectant products that are compatible and gentle to surfaces and equipment would prevent deterioration thus prolonging their lifespan and saving money in the long term. Safe disinfectant technologies would also prevent adverse reactions to animals and humans. The table below summarizes the most important characteristic of the some commonly used disinfectant classes in veterinary medicine.
<table>
<thead>
<tr>
<th>Class – Disinfectant</th>
<th>Advantages</th>
<th>Disadvantages</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Alcohol</strong></td>
<td>Hand sanitizers shown to decrease bacterial count and some viruses on skin</td>
<td>Poor activity in organic material (OM)</td>
<td>Does not leave residues after evaporation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Damaging to equipment</td>
<td>Inability to penetrate protein-rich materials 6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Flammable</td>
<td></td>
</tr>
<tr>
<td><strong>Biguanides – Chlorexidine</strong></td>
<td>Topical antiseptic</td>
<td>Very poor activity in OM</td>
<td>Inactivated by anionic detergents</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Poor environmental safety</td>
<td>Toxic byproducts to aquatic fauna</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Not active against spores, non-enveloped viruses or mycobacteria</td>
<td></td>
</tr>
<tr>
<td><strong>Chlorine – Sodium Hypochlorite (bleach)</strong></td>
<td>Inexpensive</td>
<td>Very poor activity in OM</td>
<td>Lung irritation can occur if chlorine gas is produced by mixing bleach with ammonia or acids</td>
</tr>
<tr>
<td></td>
<td>Broad spectrum</td>
<td>Ineffective cleaner</td>
<td>Can produce ocular irritation or oropharyngeal, esophageal, and gastric burns 6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Damages fabrics and corrosive</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Poor stability in solution</td>
<td></td>
</tr>
<tr>
<td><strong>Peroxigens – Peroxymonosulfate (Virkon®)</strong></td>
<td>High activity in OM</td>
<td>No sporocidal, fungicidal or mycobactericidal activity 7</td>
<td>Potentially corrosive</td>
</tr>
<tr>
<td></td>
<td>Broad spectrum</td>
<td></td>
<td>Stable in solution for 7 days</td>
</tr>
<tr>
<td><strong>Peroxigens – Accelerated Hydrogen Peroxide (Rescue®)</strong></td>
<td>High activity in OM</td>
<td>No sporocidal claim in the veterinary market</td>
<td>Good cleaner efficacy</td>
</tr>
<tr>
<td></td>
<td>Broad spectrum</td>
<td></td>
<td>High safety</td>
</tr>
<tr>
<td></td>
<td>1 to 5 minutes contact time</td>
<td></td>
<td>Non corrosive</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Stable in solution for 90 days</td>
</tr>
<tr>
<td><strong>Phenols</strong></td>
<td>High activity in OM</td>
<td>Ineffective cleaner</td>
<td>Skin and mucosa irritation 6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Variable activity against non-enveloped viruses</td>
<td>Variable environmental safety</td>
</tr>
<tr>
<td></td>
<td></td>
<td>No sporocidal activity</td>
<td></td>
</tr>
<tr>
<td><strong>Quaternary Ammonium</strong></td>
<td>Non irritating to skin</td>
<td>Ineffective cleaner</td>
<td>Occupational asthma 8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Not effective against non-enveloped virus, spores or mycobacterium</td>
<td>Evidence leading to development of bacterial antibiotic cross-resistance 8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Inactivated by anionic detergents</td>
<td></td>
</tr>
</tbody>
</table>
References

The environment and fomites (i.e. equipment, thermometers, stethoscopes, etc.) play important roles in the transmission of pathogenic microorganisms, since microbes are able to survive on surfaces for variable periods of time; this is true for human and veterinary medicine. The table below portrays pathogen survival time on human and veterinary hospital surfaces.

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Environmental Survival Time</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Staphylococcus aureus</em> (including MRSA)</td>
<td>7 days to &gt;12 months</td>
</tr>
<tr>
<td><em>Enterococcus spp.</em> (including VRE)</td>
<td>5 days to &gt;46 months</td>
</tr>
<tr>
<td><em>Acinetobacter spp.</em></td>
<td>3 days to 11 months</td>
</tr>
<tr>
<td><em>Clostridium difficile</em> spores</td>
<td>&gt;5 months</td>
</tr>
<tr>
<td><em>Pseudomonas aeruginosa</em></td>
<td>6 hours to 16 months</td>
</tr>
<tr>
<td><em>Klebsiella</em> spp.</td>
<td>2 hours to &gt;30 months</td>
</tr>
<tr>
<td><em>Salmonella</em> spp.</td>
<td>Months to years</td>
</tr>
</tbody>
</table>

Not all microorganisms are the same, since microbiologic factors that aid with the surface environment mediated transmission are variable. Some of the microbiologic factors that need to be considered are:

- Pathogen needs to survive for prolonged time on surfaces
- Ability to remain virulent
- Frequent environmental contamination
- Ability to colonize patients
- Ability to transiently colonize hands
- Transmission via contaminated hands
- Small inoculating dose
- Relative resistance to disinfectants

In human hospitals admitting a patient to a room previously occupied by a patient with methicillin-resistant *Staphylococcus aureus* (MRSA) or vancomycin-resistant *Enterococcus* (VRE) significantly increases the chances for acquiring those pathogens. In large animal hospitals *Salmonella* spp environmental contamination, along with concurrent disease, antibiotic treatment, stress and use of common instruments such as thermometers are risk factors for nosocomial salmonellosis. Viral pathogens such as *feline calicivirus* can survive in the dried state for 21 to 28 days at room temperature, 8 to 12 hours on computer keyboards, 1 or 2 days on computer mouse and up to 3 days on telephone buttons and receivers. This highlights the importance that environmental surfaces play harboring and disseminating microbes.
The colonized or infected patient’s endogenous flora is the major source for nosocomial pathogens.\textsuperscript{1} This is especially true for gastrointestinal and respiratory pathogens, since they are very contagious and indirect transmission via fomites is easy in the hospital environment.\textsuperscript{7}

Enterococci and bacteria of the family \textit{Enterobacteriaceae} are part of the gastrointestinal flora of dogs and cats, hospital surfaces contamination could create an opportunity for nosocomial bacterial infections.\textsuperscript{2,8,9} A study revealed rare isolation of bacteria of the \textit{Enterobacteriaceae} family from sampling surfaces of private veterinary hospitals, likely suggesting that cleaning and disinfection protocols successfully targeted those microorganisms.\textsuperscript{2}

\textit{Enterococcus spp} are resident bacteria of the gastrointestinal tract. These bacteria have virulence factors (gelatinase which contributes to biofilm formation) and can be multi-drug resistant, furthermore they can survive in clean hospital environments resulting in disease and difficult to treat infections.\textsuperscript{2,10} \textit{Enterococcus faecalis} that possess gelatinase can form biofilm on surfaces (cage doors). Biofilms are important since they reduce disinfectant penetration, thus allowing for the persistence of the bacteria on surfaces.\textsuperscript{2}

Hand contamination resulting from either direct patient contact or indirectly from touching contaminated environmental surfaces and or fomites is likely the most frequent route of indirect nosocomial transmission in all species.\textsuperscript{1,7,11}

In the veterinary setting proper shoe wear hygiene is important, especially in high risk and high traffic areas. Thus proper use of a foot bath with a disinfectant solution is common practice in veterinary hospitals.\textsuperscript{7}

Fomites such as contaminated stethoscopes and thermometers that are normally used for examining multiple animals, can become contaminated with microorganisms from skin and feces of an animal that is a carrier of a pathogen or a sick patient.\textsuperscript{2} If these devices are not cleaned and disinfected between examinations, they can spread pathogens to other patients, which in turn can become a carrier or ill.
References

PREVENTING MOSQUITO-BORNE DISEASES:
A MULTI-MODAL APPROACH
Robert A. Wirtz, PhD

The world’s most dangerous animals are not the Big Five from Africa, as many would suppose, but mosquitoes, other arthropods and the many diseases they transmit to human and animals worldwide. The most effective approach to limiting the morbidity and mortality of mosquito-borne diseases has been a multi-modal approach capitalizing on the concurrent use of all available tools, to include focal mosquito control and drugs. The US President’s Malaria Initiative will be described as a model program demonstrating the success of a multi-modal approach for a mosquito-borne disease.

The President’s Malaria Initiative was launched in 2005 as a five year program to reduce malaria-related mortality by 50% across 15 high-burden sub-Saharan African countries. The program relied on the rapid scale-up of four proven and highly effective malaria prevention and treatment measures: insecticide-treated bed nets (later long lasting nets), indoor residual spraying, accurate diagnosis and prompt treatment with artemisinin-based combination therapies and intermittent preventive treatment for pregnant women. From 2005-2015, the PMI and other international programs were responsible for reducing the infection prevalence of *Plasmodium falciparum* in endemic Africa by 50% and reducing the incidence of clinical disease by 40%. An estimated 660 million clinical cases have been averted since 2000, and approximately six million deaths among children under five years of age prevented.

Dengue, chikungunya, Zika and other arthropod-borne viruses are emerging or reemerging worldwide. A brief summary of these diseases will be given, with a focus on the emergence of Zika in the Americas and the need for a multi-modal approach for control.

References:


President’s Malaria Initiative website: https://pmi.gov

CDC Zika website: https://www.cdc.gov.zika
THE DISEASE OF HEARTWORM: BEYOND THE BOOKS

Stephen L. Jones, DVM

Transmitted in every state of the continental United States, Hawaii, and various regions of the world, heartworm, *Dirofilaria immitis*, is one of the most common and debilitating parasitic diseases affecting today’s pets. Over the past fifty or more years, research has produced effective heartworm preventives, developed accurate diagnostic tests, and led to a safer and more effective treatment protocol for heartworm infection. But, what remains poorly researched is the pathogenesis of the disease itself. Without a complete understanding of heartworm disease, the practitioner is continually challenged to prevent, diagnose, and treat a disease not fully understood. Through a series of necropsy images obtained from both normal and clinically ill heartworm infected dogs, an effort will be made to demonstrate gross pathology as it relates to the pathogenesis of heartworm disease.

With the exception of a single laboratory study, all necropsies were performed in clinical practice, on animals naturally infected with heartworm.

Cases represented were: 1) Clinically normal 2) Eosinophilic pneumonitis 3) Right sided heart failure 4) Caval syndrome 5) Hemoptysis 6) Post-adulticide complication 7) Normal post-adulticide 8) Slow kill

Findings based on gross pathology included the following observations: 1) Intimal proliferation of the endothelial surfaces (rugus endarteritis) of the pulmonary arteries 2) Fibrosis and dilatation of pulmonary arteries 3) Complete obstruction of distal pulmonary arteries 4) Dead heartworm emboli 5) Thromboemboli 6) Fibrosis of the pulmonary parenchyma 7) Heartworm remnants 8) Dead heartworms intertwined in the tricuspid valves 9) Clumping of worms in the right heart, tricuspid valve and vena cava 10) Pulmonary artery rupture with adult heartworms in bronchi

Heartworm disease is complicated and results from a combination of factors that involve microfilaria, living adult worms, dead adult worms, antigen-antibody complexes, the host immune system, and a variable host response to the presence of the parasite. In addition, the severity heartworm disease can vary depending on the number of heartworms present and the chronicity of infection.

Once adult heartworms reach the pulmonary arteries, disease can develop quickly. No pet infected with adult heartworms was found to be grossly normal- including those with low numbers of worms in the pulmonary arteries for less than three months. Because disease develops so early during infection, annual testing is imperative to identify and treat infection as quickly as possible. Slow kill methods will allow progression of disease and can lead to more advanced clinical disease and potentially death.

Damage caused by heartworm infection often persists even after adult heartworms are eliminated. Chronic fibrotic changes are unlikely to resolve significantly, and as such, symptoms
of disease may never improve or resolve completely. Because of the potential for permanent disease, prevention of infection utilizing a macrocyclic lactone is most important. Veterinarians, their staff, and their clients must work together to find ways to increase both the owner’s acceptance and the compliant use of macrocyclic lactone preventives. The topical use of products that both repel and kill mosquitoes can also be advantageous in reducing the likelihood of heartworm transmission.

Reference list available upon request for those who provide a self-addressed stamped envelope.
Repellency Against Mosquitoes Reduces Heartworm Disease Transmission

John W. McCall, PhD

For the past three decades, US veterinarians have focused on the use of macrocyclic lactone (ML) drugs administered monthly or twice yearly for prevention of canine heartworm disease (HWD). While effective when used compliantly, this approach ignores the role of mosquitoes as vectors of the heartworm parasite. Our recent research indicates that this limited, uni-modal approach may be enhanced with a multi-modal protocol that targets mosquitoes by blocking their ability to transmit ML-resistant as well as ML-susceptible microfilariae (Mf) from microfilaremic dogs to mosquitoes, or infective third-stage larvae (L3) from infected mosquitoes to dogs, while also killing mosquitoes. Two animal models using a ML-resistant *Dirofilaria immitis* (JYD-34 Strain) were developed and used in two studies.

The first study was designed to assess the ability of DPP (dinotefuran + permethrin + pyroproxyfen, Vectra® 3D®) administered once topically on day 0 to block the transmission of Mf from infected, microfilaremic dogs to uninfected mosquitoes. Two groups of 3 microfilaremic dogs were used: An untreated control group and a group treated with DPP. All dogs were exposed to 80 (±20) *Aedes aegypti* mosquitoes 7, 14, 21 and 28 days posttreatment. For each exposure, a sedated dog was exposed in a mosquito-proof container (29 in x 16 in x 13in) for 1 h. After exposure live and moribund mosquitoes were placed in an insectary and viability was assessed daily until dissection for L3 on day 16. Anti-feeding effect (repellency-1 h) ranged from 95.8%-100%. Insecticidal efficacy (24 h) ranged from 95.5%-100%. A total of 810 mosquitoes fed on untreated dogs, and all 22 mosquitoes that fed on treated dogs were dead within 72 h (anti-transmission effect of 100%), completely blocking L3 development and potential transmission of L3. Thus, the topical formulation of DPP used in this study was more than 95% effective in repelling and killing mosquitoes for 28 days after treatment and was 100% effective in blocking transmission of the infection from microfilaremic dogs to mosquitoes. Repellent and insecticidal properties of ectoparasiticides could contribute substantially to reducing the risk of heartworm transmission, even those heartworm subtypes resistant to ML preventives or any other type of preventive, and slow down the spread of resistance.

The second study was designed to evaluate the effectiveness of orally administered milbemycin oxime (MBO, Interceptor®) when combined with topically administered DPP against experimental infection of dogs exposed to mosquitoes infected with *D. immitis* L3. For this study, 32 Beagles were allocated to 4 groups of 8 dogs each: Group 1 was the untreated control, Group 2 received DPP on Day 0, Group 3 received MBO on Day 51 and Group 4 was treated with both products. The dogs were exposed to infected mosquitoes on Days 21 and 28. Mosquitoes were dissected before and after exposure to estimate the number of L3 transmitted to each dog. After exposure (1h), mosquitoes were counted/categorized and mortality was assessed daily for 3 days. The dogs were necropsied 150-157 days PI. A total of 413 mosquitoes fed on the total of 16 dogs in the 2 groups not treated with DPP and only 6 fed on the total of 16 DPP-treated dogs [overall anti-feeding (repellency) effect of 98.5%]. Anti-feeding effect for the DPP, MBO and DPP + MBO groups was 98.1%, 5.2% and 99.1%, respectively. The estimated average number of L3 transmitted to untreated controls, DPP, MBO and DPP + MBO dogs were 76, 2, 78 and 1, respectively. The average worm burdens for the control, MBO, DPP and MBO + DPP groups were 41.0 (range 21-66), 17.1 (range 7-39), 1.5 (range 0-7) and 0. The heartworm
preventive efficacy was 58.2% for MBO alone, 96.3% for DPP alone and 100% for DPP + MBO.

In conclusion, DPP repelled and killed most mosquitoes that were capable of transmitting Mf from infected dogs to uninfected mosquitoes and most mosquitoes that were capable of transmitting L3 from infected mosquitoes to uninfected dogs. This research supports a “Double Defense” protocol in which DPP can be combined with any heartworm preventive. All dogs exposed to heartworm should benefit from the mosquito repellency and insecticidal efficacy of DPP added to their heartworm preventive protocol, and this benefit is even more obvious when a macrocyclic lactone-resistant strain(s) of heartworm is involved or lack of compliance in the administration of ML preventives is known or suspected.

We propose a practical, multi-modal strategy for heartworm prevention, incorporating systemic macrocyclic lactone anthelmintics, as well as topical mosquito-repellent ectoparasiticides, with other adjunctive measures, (e.g., mosquito control, reducing exposure, and doxycycline) which will enhance the practitioner’s ability to provide a comprehensive and more effective preventive strategy against heartworm disease.

Reference list available upon request for those who provide a self-addressed stamped envelop

1 Vectra 3D®, Ceva Animal Health, Lenexa, KS USA
2 Interceptor®, Virbac, Fort Worth, TX USA
Pets and Kids – Have fun and be safe! Parts I and II
E’Lise M Christensen Bell, DVM DACVB

INTRODUCTION
Animals and children can be like cookies and cream or oil and water. And which version you get can depend on what’s going on for all the individuals (including animals) in the family that day. Thankfully, there are known ways to get more sweet days than sour. Coach your clients with these tips to get immediate improvements in current problem situations and to avoid problems in the future.

PREP THE INGREDIENTS BEFORE MIXING
Contrary to popular belief dogs, cats, and other common pets do not come pre-programmed with a knowledge of how to act around children. And children certainly do not naturally understand how to interact with other animals. They are still learning how to interact with other children of the same species! How could they possibly have a handle on how to interact with a completely different one?

Prep for Non-Human Animals
- Design, set-up, and teach each animal to use a “success zone.” This is a safe place where the animal is taught to feel comfortable. Examples of success zones are exercise pens, cat condos, elevated cat resting areas, hiding houses/crates (all species), and/or rooms that can be closed or baby gated off. This space should be used proactively to keep animals and children physically separated in order to prevent fearful and/or aggressive behavior from developing or intensifying. This space IS NOT a torture chamber! The animal MUST be taught to enjoy this resting space for relaxation and/or play. This can be done in many dogs using a remote activated food dispensing system or with creative use of puzzle toys.
- Exercise properly and daily
- Use desensitization and counter-conditioning to teach the animal to enjoy being handled, hugged, and kissed
- Use positive reinforcement training to teach: TOUCH, TARGET, SIT, BACK UP, GO TO BED (the success zone), FIND IT, and other fun, useful behaviors. Yes, cats can be taught these as well!
- Use positive reinforcement to teach the animal to come briskly when called. This is best done by using food or play rather than human touch or praise in some animals. In order for the animal to come when called, the cue must never mean the end of something the animal enjoys (such as playing with other dogs) or the presentation of something the animal dislikes (such as a bath). If those things must be done, the handler should go get the animal rather than calling him/her.
- Use calming supplements, calming tools (such as body wraps), pheromones, and/or medications to treat ongoing anxiety, agitation, aggression, and/or impulse control disorders.
- All meals delivered in puzzle toys
- Adequate food, water, shelter
- Provide a safe and comfortable overnight sleeping spot where the animals cannot be physically disturbed by children. This is likely to be a comfortable space on the floor, in a crate, or behind a gate. It’s best for dogs and cats not to sleep in the bed with parents or children if there will be people in and out of the bed at night since this is disruptive for their sleep patterns, can cause fear, and can be painful.
- PROACTIVE treatment for pain, itch, or other medical problems
Prep for Parents

-UNDERSTAND species specific body language ASAP. There are many great resources for information on this. Remember, research shows the vast majority of people (veterinarians included!) do NOT understand species-specific body language even if they have worked with dogs for years, unless they have been specifically trained in it. The school of life is not enough. Here is one free and very helpful website option. [http://eileenandddogs.com/dog-body-language/](http://eileenandddogs.com/dog-body-language/)

-KNOW that adult supervision is not enough. Most young children are bitten by dogs they know in the presence of an adult. This can even be an adult who is “actively” attempting to control the situation. Poor prognostic indicators for a situations include: the adult doesn’t understand species-specific body language or is trying to actively encourage the child and animal to interact. Many adults will actually hold fearful or aggressive animals still so children can pet them. This is a terrible idea.

-ACCEPT that animals are not humans and the majority of pets we keep do not enjoy intense physical contact such as hugging, kissing, putting faces close together, or lifting/carrying. In fact, these interactions can be painful, terrifying, and, at best, emotionally uncomfortable. For an excellent illustration of this, send your clients to [www.stopthe77.com](http://www.stopthe77.com) for a wonderfully educational short video. Parents should watch this video before showing it to their children. It’s sad.

-ROUTINE is important for children and animals. Household rules need to be predictable for all species in the house.

-ACTIVELY interact with the animal in a way the animal likes at least once daily without children present in order to help gauge the patient’s well-being as well as provide some one-on-one attention in a way the animal likes.

-PLAN interactions between animals and children rather than letting them happen and “seeing how it goes.”

-“FINE” is a warning thought. Anytime an adult knowledgeable about body language uses this word in his/her mind, one must recognize the animal is likely not comfortable but is only “tolerating” the interaction.

-Interactions MUST be fun for everybody, animals included.

-MODEL appropriate behaviors around animals. Adults should not hug, kiss, play roughly, and disturb animals while eating or resting, etc. when children are around even if the animal likes it. Other animals may not like it and children cannot identify this difference.

Prep for Babies, Toddlers, and Kids of All Ages

-TEACH appropriate petting on stuffed animals not real animals.

-TEACH alternative behaviors when around pets. Instead of “hugging pets, hug friends.” Keep hands on belly button or at sides, etc.

See this video for a cute and educational song-> [https://www.youtube.com/watch?v=36Z9RRjiQMA](https://www.youtube.com/watch?v=36Z9RRjiQMA)

-TEACH kids not to approach the success zone when the animal is in it or, ideally, ever.

-TEACH kids to ignore and give space to all animals who are eating, sleeping, or awake and resting.

-TEACH kids to understand species-specific body language so they can identify an animal who is enjoying an interaction from one who is not.

-HELP children disengage from situations that are too tempting for them to dismiss themselves.
-TEACH kids they should never approach or pet a non-family animal unless they have the owner’s permission AND parental permission. And even then, it’s just better not to interact with unknown animals.

-TEACH kids to play species-appropriate games with family animals—FIND IT, FETCH (for animals who don’t guard toys), POSITIVE REINFORCEMENT TRAINING, and NOSE WORK are great options. All of these, except nose work, are great interactions for cats and children, too. Many cats enjoy laser play, and it is a wonderfully hands-off game. Laser toys or reflection chasing games should be avoided in dogs due to anecdotal concerns about triggering compulsive behaviors in sensitive patients. This problem appears to be much less likely in cats.

-CALL the pet. When a child is getting into a hot zone with an animal, the animals are more likely to get out of the way and come to the adult than the children. This is especially true for animals who have strong reinforcement histories for coming when called.

**WARNINGS AND SAFETY TOOLS**

If an animal already has a history of aggression towards children then strict separation is required to prevent injuries. Unfortunately, separation and avoidance strategies can fail. Therefore, even if the major plan is avoidance, all of the Prep for Parents, Kids, and Animals must still be done.

A properly fitted basket muzzle is a wonderful way to improve safety for families who love dogs with fears, phobias, or aggressive behaviors that might result in biting attempts. HOWEVER, a dog who is wearing a basket muzzle should still not be exposed to his/her triggers. The basket muzzle is a backup safety tool. It is common for people to obtain muzzles for dogs and then allow children to be children with them (for instance, crawling on them, pulling their ears, grabbing their feet, dressing them up). Unfortunately, this can make the aggression worse in the long run and is a very poor quality of life for the animal.

Leashing, even in the home, can be an effective way to manage a dog’s location. However, many parents find this difficult, especially with toddlers, as toddlers are inclined to grab leashes.

**SUMMARY**

Parents often forget that even if the animals in their family tolerate or enjoy typical handling from children, other animals could be frightened and/or aggressive with this type of interaction. Thankfully parents and children can learn the skills they need to understand and enjoy many different species in a way that’s fun for the animals, too.
Two Minutes a Day Keeps the Rodeo Away: Training Puppies and Kittens for Medical Handling
E’Lise M. Christensen DVM, DACVB

Many clients avoid the veterinary clinic because of the distress they feel when they contemplate the fear their animals will face at the veterinary clinic.

The combination of at-home implementation and in-clinic low stress handling early in the pediatric/juvenile period maximizes an animal's chances for appropriate medical care throughout his/her life. This means clients will be more comfortable coming back to see us and complying with recommendations. And in the long term, this means healthier, happier families.

Effective, efficient options for low stress handling in the veterinary clinic are easy to implement once veterinarians and staff decide to do so. These therapies don’t require more time than regular examination plans, and will make your day more fun as well as bond pets and clients to your practice. In addition, these therapies help patients improve from visit to visit, rather than decompensate from visit to visit which is common.

Much has been written and presented on low stress handling and restraint. In this lecture, we are going to focus on options for handling techniques for examinations as well as at-home behavioral therapies to support your patients through their medical care.

We may not get to all of these techniques during the lecture depending on how many questions we have. It’s only around 50 min after all. That goes very quickly! So I have provided links to helpful videos and sites. I strongly encourage you to look at these videos and sites and to share them with your staff. If you have any questions as you work to make changes, don’t hesitate to reach out to your local veterinary behaviorist or to contact my service directly at info@behaviorvets.com.

IN THE HOSPITAL
Principles of low stress handling and restraint should be implemented with all animals, and especially our pediatric and juvenile patients. Negative experiences during this sensitive time can set them up for a lifetime of behavioral pathology in the hospital and in their homes. What you do for even 3 seconds at the veterinary clinic can have life-threatening, long-term consequences for each animal’s behavior.

To that end, remember the following for improved outcomes.

All patients who can be given food and generally like food should get very, very tasty treats for essentially most of the visit. It wouldn’t be wrong to have a patient receive 30-50 treats from the time they come in the door to the time they leave the hospital. Remember, treats should be of appropriate size (1 lick of anything liquid or pasty, 1 piece of food that is less than ¼ in diameter (or smaller if possible). For instance, a tiny sliver of a bonito flake would be appropriate as one treat for a cat. Half of a bonito flake would still be plenty, even for a large breed dog.

Animals who are generally eager to eat, but stop eating in the hospital are stressed. The algorithm for low stress handling is a great tool to keep the practice team on track (http://veterinarymedicine.dvm360.com/low-stress-handling-algorithm-key-happier-visits-and-healthier-pets).
Appropriate placement and use of motivators (food, play, toys, and some types of touch or talking) is imperative for the best experience. For instance, applying peanut butter or squeeze cheese to the exam table, exam room floor, or a wall or cupboard can help even untrained patients remain stationary for exams. Very tasty food should be presented for the duration of handling and especially any injections or other painful procedures.

Remember, all patients need adequate footing, staff and veterinarians who understand body language, quiet, clean rooms with minimal smells of cleaners and other animals, and handlers/owners who are calm and happy rather than anxious and agitating. Examples of agitating owner behaviors include touching repeatedly, speaking in a worried tone with repetitive staccato sounds, scolding the patient, grabbing or hugging the patient, etc.) In addition, 60W lighting, classical music, small amounts of lavender essential oils and species specific pheromone therapy can be helpful. It’s important the veterinary team feel comfortable changing the venue of the examination or procedure to help the patient relax if needed. For instance, some pets will feel more comfortable if examined outside, some in their homes (some not), on the floor, in the owner’s lap, in your lap, in a bowl, bed, carrier, or sink. Judicious use of warming devices can be helpful as young pups and cats of all ages may be eager to rest on a warmed surface.

In case towel restraint may be used to help keep the patient cozy and comfortable for handling, a regular sized, moderately thick bath towel should be pre-placed on the examination site so the options for appropriate towel wraps are kept open. Remember, most towel wraps are done with pre-placed towels, the notable exception would be the towel grab. Note: a pre-placed towel is not adequate for warmth or traction.

When doing the patient’s examination, take a couple of 1-2 second breaks to play or give additional tidbits while you are talking to the owner. Leave temperatures and other generally stressful interventions until the end of the examination if possible. Choose the level of importance for each interaction so you can do the most important ones first.

After the examination is done, especially if the patient is on an examination table, spend at least 20-30 seconds giving more treats or playing with the animal. Examinations and all interactions with staff should start and end on a good note for the patient, since these are the parts of the experience with the staff that are most likely to remain in the memory of the patient.

Well-managed species-specific socialization classes can help owners learn how to teach their pets that medical handling can be fun. They are especially good because these classes provide a safe avenue for animals to visit the hospital at a time when no medical handling is needed. Indeed, a well-run class should be fun for all participants and bond them more strongly to the hospital.

AT-HOME TRAINING
Important medical handling behaviors include (but aren’t limited to):
- Mat Training
- Chin Rest (in hand, on lap, or on object)
- Taking pills
- Ear Handling
- Nail Trimming/Foot Handling
- Allowing Injections
- Tooth Brushing
Each of these behaviors can be trained through desensitization and counter-conditioning, classical conditioning, and/or through operant (teaching the patient to do something specific in order to obtain a reward) therapy.

When these techniques are combined, patients can learn quickly to not only tolerate, but enjoy most medical handling.

Basic tips to keep in mind:
- Don’t increase the difficulty of a game/training activity until the patient has had AT LEAST THREE successful repetitions at the current level.
- Back up at least 2 steps if species-specific signs of agitation are noted.
- Intersperse easy, fun, and previously learned cues throughout training in order to keep the patient relaxed and feeling successful.
- Keep training sessions short enough that the patient is always interested in doing more when you stop. Nothing good comes from training too long.
- If an animal elects to stop training, allow this choice 100% of the time. Start easier next time and stay at the easier steps longer. Forcing an animal to participate will backfire.
- Go SLOWLY. It doesn’t take long to train these behaviors in most patients. But it will take forever if you go too quickly through a training game plan because there will be too many mistakes. This will cause frustration and even fear.

For more information on body language and teaching animals to tolerate medical handling see the following resources:
www.fearfreepets.com
www.drsophiayin.com
http://abtconcepts.com/training-videos/

Special Mat Training: 5 reps/day
- Get out special mat (mat should be comfortable/easy to stand on and have ample traction on both sides—slippery is the enemy here)
- Keep grooming and medical tools within the animal’s sight during these sessions, but do not manipulate them during these sessions.
- Feed meals from puzzle toys on it
- Reward pet for going to mat
- Over time reward pet for staying on the mat
- This behavior can be easily trained using an automated food dispenser with a training program, such as a Treat and Train.
- These 5 reps are separate from any other behavioral therapies done using the mat as “base.”

Chin Rest: 5 reps/day
- Choose where the pet will rest his/her chin (the TARGET), options include a towel on a lap, an object, or a hand.
- Keep grooming and medical tools within the animal’s sight during these sessions, but do not manipulate them during these sessions.
- Present target
- Mark exactly when the behavior happens (say “yes” or use a clicker) and reinforce any approaches to the target
-Eventually, only mark when the chin hits the target, then when the chin rests on the target, and then when the animal will hold his/her head on the target for gradually increasing durations.
-At this point, additional handling can be added intermittently (pill taking, ear handling, foot handling, voluntary injections, etc.).

Taking Pills 5 reps EOD:
-After training the special mat behavior this can be started.
-Give pet treats in pill gun on the special mat.
-Give pet tasty liquid on the special mat.
-Practice “Three Treat” game: Take 3 soft treats, the first treat is a blank, the second treat is filled with a piece of crunchy kibble or an empty pill capsule and quickly follows the first, the third treat is a blank and quickly follows the second. In this way, the pet gets used to getting treats in quick succession. The goal of this activity is for the pet to be very excited about getting the treats as quickly as possible so that the next treat is always being anticipated and the treat in the mouth is ignored in favor of the new one. Every day the treat containing the crunchy should vary.
-Pilling and giving liquid medications can be helped by pairing them with a previously learned and enjoyed chin rest behavior (see above).

Ear Handling 5 reps EOD
-Teach the chin rest (see above).
-Have all medical items in sight of the patient.
-Lift the ear, give a treat within 1-2 seconds.
-When animal is comfortable with 3 reps of this in a row, go to the next step.
-Put a finger inside, give a treat within 1-2 seconds.
-When animal is comfortable with 3 reps of this in a row, go to the next step.
-Move the finger around inside the ear, give a treat within 1-2 seconds.
-When animal is comfortable with 3 reps of this in a row, go to the next step.
-Lift other ear, repeat.
-When this is going well, add shaking a bottle of ear wash just before ear handling.
-Eventually, you can add actually putting a small amount of ear wash on a cotton ball or squirting it in the ear, rubbing it around, and giving a treat with 1-2 seconds. Each of these is a separate step.

Foot Handling: 5 reps EOD
The following video demonstrates basic desensitization and counterconditioning to nail trims in an aggressive dog. This plan should be proactively followed for all puppies, even if they are not growling, snarling, snapping, biting, stiffening, or showing other signs of agitation.
https://drsophiayin.com/videos/entry/training_a_dog_to_enjoy_toenail_trims/

The following picture and write up demonstrates basic desensitization and counterconditioning for nail trims in a cat.

Voluntary Injections: 5 reps EOD
-Send pet to the special mat.
-Present chin rest target.
-When patient is resting chin on target, rub/tent injection areas. Pick a different area for each rep. Some options include femoral regions, paraspinal regions, shoulder areas, and “scruff” area. Give a treat within 1-2 seconds of each rep. 
-When the animal is comfortable with rubbing/tenting of these areas, the protocol can be escalated to poking a finger gently in the tented skin and eventually to poking the skin with different items (pen, pencil, chop stick, and eventually a capped syringe, and then a syringe with a needle). The ultimate step up would be injection of saline into the tented area.
-The goal here is to help the animal be comfortable with a variety of sensations along with the skin tent.

Tooth Brushing: 5 reps EOD
-Obtain a brushing implement
-Classically condition the brush by showing the brush, then giving a treat within 1-2 seconds, remove the brush immediately from sight.
-Repeat until patient is eagerly looking from brush to the owner expectantly.
-Then continue the process by gradually placing the brush closer and closer to the pet’s teeth.
-When that’s going well, you can teach the chin rest behavior, if not done already and progress from there.

Owners do not need to work on each of these activities every day. They can split the work on these behaviors into MWF behaviors (do mat training, chin rest, pillling, foot handling, and tooth brush conditioning MWF) and TR (do mat training, chin rest, vaccination prep, and ear handling behaviors TR). They can take the weekend off, and they will still get great results.

SUMMARY
When both family members and veterinary teams work together to prepare puppies and kittens for examinations as well as to make trips to the veterinary clinic fun and enjoyable, these patients will stay in your practice longer and make their families happier. And importantly, the patients will be happier and that’s better for everyone.

REFERENCES

Available upon request
SEDATION FOR THE DIFFICULT TO HANDLE PATIENT
Lisa S. Ebner, DVM, MS, DACVAA, CVA

Not all patients are happy to see their veterinary health team and readily allow restraint for simple diagnostic procedures or even a physical exam. It is important to distinguish between the patient that is aggressive vs. nervous vs. just excited. Obviously, cats are not small dogs so we have to approach them differently both from a pharmacologic and environmental approach. Non-pharmacologic approaches such as Dr. Sophia Yin’s Low Stress Handling™ technique can be implemented in both dogs and cats in the veterinary health care setting. This may help avert a situation where a patient is unnecessarily stressed out and therefore drugs must be given to complete an examination or diagnostic procedure.

The veterinary health team understands that any patient has the potential to inflict harm upon them with little to no warning. Therefore, taking a minute to slow down and assess each patient from a distance to read their body language is worthwhile. A calm dog will be socially interactive and easily approached. The wiggling body will be a positive cue that you can proceed with greeting the patient. The nervous dog may be hiding behind the owner or retreats when you enter the room. The body language is tense and you may observe the patient yawning or licking. If the patient is able to gently take an offered treat, then it is probably safe to proceed with the exam or procedure but continue to assess body language for any changes. The uncooperative patient may warrant administration of a sedative drug in order to pursue examination or diagnostic procedures. The owner should be warned about the potential increased risk associated with administering drugs to a patient prior to a thorough examination and pre-anesthetic blood work.

The nervous or fearful patient can benefit from a neuroleptanalgesic approach to sedation, utilizing a sedative/tranquilizing drug combined with an opioid. This combination of drugs will lead to a synergistic effect, therefore lowering the amount of each drug needed to produce the desired effect. Dexmedetomidine (2-5 µg/kg) combined with butorphanol (0.2-0.4 mg/kg) and given IV is quite effective in most patients. If IV access is not possible to obtain, then a slightly higher dexmedetomidine dose (5-10 µg/kg) and similar butorphanol dose can be given IM. If a more painful diagnostic procedure is planned, switching out the butorphanol for hydromorphone (0.05-0.1 mg/kg) will be beneficial and also create a nicely sedated patient. Benzodiazapines, such as diazepam or midazolam, are not the best recommendation for sedation and could actually lead to more excitement in an otherwise healthy (not sick) patient. Acepromazine is not the author’s first choice in this type of case due to the long onset and duration of action, cardiovascular side effects, no reversal agent, and possibility that the patient will not sedate as reliably with this drug if already anxious.

Owners of aggressive dogs should place a muzzle on them at home prior to transport to the veterinary clinic or in the parking lot of the clinic prior to entering the building. If the aggressive dog arrives to the clinic without a muzzle and does not allow the veterinary health team members to place one, another option besides the “leash through the door” trick would be a squeeze cage. Some squeeze cages can be bought for less than $100 online. But Shor-Line also makes a
squeeze restraint in various size cages\(^1\). An aggressive dog will need a stronger “cocktail” of drugs in order to properly sedate it for safe examination. The author typically adds ketamine (3 mg/kg) to the dexmedetomidine and hydromorphone combination mentioned above. The drugs will likely have to go by the IM route instead of the IV route. The absorption rate can be affected by administration of the drugs incorrectly in the subcutaneous region or in a patient with a substantial amount of body fat. After administration of the drugs, the patient should be allowed to remain in a quiet, darkened room with intermittent checks by the health care team. Once it is safe to approach the patient, an IV catheter can be placed if the planned procedure is anticipated to be prolonged or painful so that more drugs can be quickly given to the patient. The effects of the ketamine begin to wear off in about 20 minutes, so this should be taken into consideration. It is important not to reverse the dexmedetomidine with atipamezole until the effects of the ketamine have worn off (or at least 20 minutes after ketamine administration) due to the potential for excitement in recovery. The author typically does not reverse dexmedetomidine in aggressive dogs unless the patient is experiencing problems from the drug or the owner does not wish to wait for the drugs to wear off so the patient can go home. Alfaxalone is a newer drug on the market that is typically used as an intravenous anesthetic induction agent in dogs and cats, but can also be given by the IM route. The volume needed for larger patients could be problematic, but the amount of alfaxalone can be reduced by combining with dexmedetomidine and an opioid. The level of sedation from alphaxalone in dogs is dose dependent, but the author typically uses about 2 mg/kg IM if combined with other drugs (such as dexmedetomidine and an opioid) for sedation.

For the aggressive cat, often getting it out of the clinic cage can be problematic. One product that the author has success in using is called the EZ-Nabber\(^2\). It consists of a metal frame and a mesh netting that opens up to “catch” the cat in, then can be secured and medications can be administered through the mesh netting. This product cost about $135 and can help prevent unnecessary scratches and bites from fractious feline patients. One protocol the author recommends for these difficult to handle cats is a combination of ketamine (5 mg/kg) + dexmedetomidine (10 µg/kg) + butorphanol (0.2 mg/kg) IM. If a painful procedure is planned, the butorphanol can be switched out for morphine or hydromorphone. Buprenorphine does not seem to produce reliable sedation. Telazol\(^3\) (9-12 mg/kg) given IM is another option for sedating difficult to handle cats, but recoveries tend to be rougher. Alfaxalone given IM in cats can produce sedation, but seems to be very dose dependent. The author has noted excitement and myoclonus at lower doses, so to avoid this the author typically uses it in cats for IV induction of anesthesia. Box (or chamber) induction with inhalant anesthetics should be avoided for several reasons.

Another approach is prescribing medications owners can give at home to reduce stress for the patient and optimize the visit to the veterinary hospital. Gabapentin (50-100 mg PO) given to cats

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\(^1\) Shor-Line. 511 Osage Avenue, Kansas City, Kansas 66015.
\(^2\) Campbell Pet Company. P.O. Box 122, Brush Prairie, WA 98606.
\(^3\) Zoetis. 10 Sylvan Way, Parsippany, New Jersey, 07054.
\(^4\) Zoetis Inc. Kalamazoo, MI.
to sedate them several hours prior to transport to the veterinary hospital is one technique gaining popularity. Dexmedetomidine oral gel (Selio®)⁴ is a newer FDA approved product on the market and is indicated for sedating dogs with a noise aversion. Trazodone (3.5-7 mg/kg, PO) has been evaluated for use for facilitating post-surgical confinement in dogs (Gruen et al 2014). Oral acepromazine has a wide dose range (0.55 to 2.2 mg/kg according to Plumb’s), but does not have a reputation for reliable sedation in dogs. Therefore, oral acepromazine is not recommended by the author for the purpose of sedating difficult to handle dogs and cats.

References available upon request.
Any updates in equipment, techniques or drugs in the field of veterinary anesthesia and analgesia are exciting for the general practitioner. Some of the updates are well publicized and others are not. So the purpose of this lecture is to inform the general practitioner about newer additions to knowledge in the field of veterinary anesthesia and analgesia so that they can make informed decisions about what they would like to incorporate in to their own practice.

**Drugs**

Alfaxalone (Alfaxan®)\(^1\) is FDA approved for use as an intravenous injectable anesthetic in cats and dogs. It is a clear, 1% solution with no antimicrobial preservative (so should be thrown out 6 hours after initial use). Alfaxalone is a neuroactive steroid molecule that binds to GABA\(_A\) receptors to increase chloride conduction into the cell. This leads to hyperpolarization of the postsynaptic membrane, ultimately causing CNS depression. It causes hemodynamic stability at clinically relevant doses, but can cause dose-dependent hypotension due to vasodilation. It also causes dose-dependent respiratory depression and/or apnea. It has no analgesic properties and should not cause pain on IV injection. It can be used in patients undergoing cesarean section with similar survival rates in puppies compared to propofol (Doebeli *et al.* 2013). It is an acceptable induction agent in dogs that are a poor anesthetic risk. It can cause paddling and excitement in cats and vocalization in dogs, so recovery in a quiet and dark room is recommended. The cost is around $30 for a 10 mL vial and it is a Class IV controlled substance, so should be kept locked up and usage recorded. In a patient that has achieved moderate sedation from premedication, the typical induction dose is ~ 2 mg/kg IV. A higher dose (3-4 mg/kg, IV) will be needed for a patient that has not received premedication. It can be given IM and also used as a CRI to maintain anesthesia.

Grapiprant tablets (Galliprant®)\(^2\) is an FDA approved new drug for treatment of pain and inflammation in dogs with osteoarthritis. It is a non-COX inhibiting NSAID that works by antagonizing the prostaglandin E\(_2\) EP4 receptor. It should not be used with other NSAIDs or corticosteroids and it should not be given to cats. The dose is 2 mg/kg PO SID (dogs less than 8 lbs cannot be accurately dosed). The most common side effects are gastrointestinal tract upset. A 7-day wash out period should be followed for any patient switching from another NSAID to Galliprant®.

Simbadol™\(^3\) is a high concentration preparation of injectable buprenorphine that is FDA approved for SQ administration once a day for up to 3 days to manage postoperative pain in cats. It takes approximately one hour for onset, so it should be given prior to surgery. The cost for a 10mL vial is around $200, but this can be cost-effective in some patients that would be given additional doses of buprenorphine every 8 hours. Buprenorphine is not associated with hyperthermia in cats. It is not the best opioid analgesic for patients with severe pain, and it is generally recommended to combine it with an NSAID if the patient can tolerate this class of drugs. The Simbadol™ dose is 0.24 mg/kg SQ SID and the concentration is 1.8 mg/mL.
Robenacoxib injection and tablets (Onsior®)\textsuperscript{4} is a newer NSAID on the market approved for use in managing pain and inflammation from soft tissue surgery (dogs) and orthopedic surgery, ovariohysterectomy, and castration (cats) in patients $\geq 4$ months of age (and $\geq 2.5$ kg) for up to 3 days. The injection is only approved for SQ use. The vial should be stored in the refrigerator and discarded 28 days after punctured. The dose for injectable Onsior\textsuperscript{®} is 2 mg/kg SQ SID and the concentration is 20 mg/mL. The dose for oral Onsior\textsuperscript{®} in cats is 1 mg/kg PO SID (dogs are dosed at 2 mg/kg PO SID).

Dexmedetomidine oromucosal gel (Sileo®)\textsuperscript{5} is a FDA-approved medication for the treatment of noise aversion in canines. This formulation is only meant to calm and not sedate dogs. It comes in a 3ml syringe and the concentration is 0.1 mg/ml for administration in to the cheek pouch. The bioavailability of Sileo\textsuperscript{®} is 28%, therefore the cardiovascular side effects are not as profound as that seen with injectable dexmedetomidine. Clients should be instructed to wear disposable gloves when administering Sileo\textsuperscript{®}. It should not be used in debilitated dogs, particularly those with cardiovascular disease.

Fentanyl transdermal solution (Recuvyra®)\textsuperscript{6} has been on the market for several years. It is designed to provide continuous delivery of fentanyl for the control of postoperative pain for four days in dogs without the hassle of the fentanyl patches – which have a tendency to fall off or the possibility of miscalculating the dose of patches to apply for a particular patient. There is a concern for secondary exposure to both the veterinary health team and the owner, so contact with the area of the skin where the solution is applied should be avoided for 3 days. Close attention should be paid to the dose based on body weight due to the potential for adverse effects associated with this potent opioid analgesic. It should be avoided in dogs with systemic disease and should never be applied to cats.

Maropitant (Cerenia®)\textsuperscript{7} is a neurokinin-1 receptor antagonist that inhibits the binding of substance P in the emetic center and chemoreceptor trigger zone to prevent vomiting in dogs and cats. But since substance P is also involved in pain pathways, Cerenia\textsuperscript{®} has also been shown to produce visceral analgesia demonstrated by inhalant-sparing effects when given to dogs (Boscan \textit{et al} 2011) and cats (Nlyom \textit{et al} 2013). Other studies have shown that injectable and oral Cerenia\textsuperscript{®} given one to two hours, respectively, prior to anesthesia premedication can prevent hydromorphone induced vomiting and nausea (Kraus 2013 and 2014). Similar results were found when Cerenia\textsuperscript{®} was given 45 minutes prior to premedication with morphine. Cerenia\textsuperscript{®} was also associated with an improved quality of recovery and a significantly faster return to feeding (Ramsey \textit{et al} 2014). It is of clinical interest that maropitant does not prevent gastroesophageal reflux in anesthetized dogs even if vomiting is prevented (Johnson 2014).

Tapentadol is a mu-opioid receptor agonist and a norepinephrine reuptake inhibitor that may have a use for treating both nociceptive and neuropathic pain. Due to a difference in metabolism in dogs, tapentadol may theoretically be a better choice than tramadol for acute nociceptive pain. In a study comparing tramadol, morphine, tapentadol in Beagle dogs, the latter two drugs were found to induce antinoception while tramadol did not produce this effect (Kogel 2014). More pharmacokinetic studies are needed in veterinary medicine before recommendations can be made.
**Equipment**

EMD Safety Valve\(^8\) is a pressure relief valve that can be placed anywhere on the breathing circuit to prevent barotrauma due to a pop-off valve inadvertently being left closed. The cost is $95.

Pleth variability index (PVI\(^9\)) is a new technology that can be used to show changes that reflect physiologic factors such as vascular tone, circulating blood volume, and intrathoracic pressure excursions. PVI\(^9\) measures the dynamic changes in the amplitude of the pulse oximeter waveform during one or more complete respiratory cycles, then expressed as a percentage (0.02-20\%). This monitor can help clinicians decide whether or not to administer fluids to a patient during surgery. Studies in the literature concluded that PVI successfully detects hypovolemia and return to normovolemia, but was not able to detect hypervolemia (Ricco *et al*. 2012).

AG Cuffill\(^10\) is a syringe-like device used for measuring cuff pressure and controlling the volume of airway cuffs in endotracheal, tracheotomy tubes, and laryngeal masks. It is easy to use and allows the measurement to be done without losing pressure already in the cuff. This device is designed to be accurate (± 2 mmHg/cmH\(_2\)O) for 100 uses, then should be disposed. It should be turned on while disconnected from the airway.

**Techniques**

Feeding a small amount of canned food (half of daily rate) 3 hours prior to anesthesia can reduce the incidence of gastroesophageal reflux in dogs (Savvas *et al* 2016 and 2009).

Assessing pain in cats continues to be a challenge for the practitioner. One currently validated assessment tool is the UNESP-Botucatu multidimensional composite pain scale\(^11\). It encompasses miscellaneous behaviors, reaction to palpation, vocalization, posture, activity level, attitude, appetite, comfort level, and arterial blood pressure. This system is used to assess patients with acute, postoperative pain. Another available pain scale that is simple to use is the Colorado State University Feline Acute Pain Scale\(^12\). The author routinely uses this scale in third year veterinary student live animal surgery laboratories. In feline patients with chronic pain, the Feline Musculoskeletal Pain Index (FMPI)\(^13\) from NC State Veterinary Medicine Comparative Pain Research Laboratory is available for veterinarians to give to the owner to fill out. This tool has been validated for detecting therapeutic efficacy in cats with degenerative joint disease and impaired mobility being given low-dose daily meloxicam (Gruen *et al* 2015).

Ultrasound guided locoregional nerve blocks are increasingly being used in small animal practice, but also gaining popularity in food animal practice. Advantages of using the ultrasound include anatomical visualization, decreased amount of local anesthetic used, improved quality of the local anesthetic block, and decreased incidence of toxicity due to inadvertent administration in to a blood vessel. Successful integration of this technique involves the use of an ultrasound machine with a high-quality image, knowledge of anatomy, and practice finding the various structures. For the latter, the author recommends investing in a hands-on training session with an
expert in this area of anesthesiology. Instructional wetlabs are available at various universities\textsuperscript{14}.\textsuperscript{15} A more affordable alternative is Cornell University CVM Peripheral Nerve Blocks in the Dog DVD course\textsuperscript{16}. Two recent books published on this topic are \textit{Small Animal Regional Anesthesia and Analgesia}\textsuperscript{17} by Luis Campoy and Matt Read and the \textit{Handbook of Small Animal Regional Anesthesia and Analgesia Techniques} by Lerche \textit{et al.}

References available upon request.

Footnotes
\textsuperscript{1} Jurox, Inc. 4520 Main Street Kansas City, MO 64111
\textsuperscript{2} Aratana Therapeutics, Inc. Leawood, KS 66211.
\textsuperscript{3} Zoetis. 10 Sylvan Way. Parsippany, NJ 07054.
\textsuperscript{4} Elanco Animal Health. 2500 Innovation Way. Greenfield, IN 46140. USA.
\textsuperscript{5} Zoetis Inc. Kalamazoo, MI 49007.
\textsuperscript{6} Elanco Animal Health. Indianapolis, IN 46285.
\textsuperscript{7} Zoetis Inc. Kalamazoo, MI 49007.
\textsuperscript{8} Essential Medical Devices, LLC. 770-841-4666.
\textsuperscript{9} Masimo Corporation. 40 Parker, Irvine, CA 92618.
\textsuperscript{10} Mercury Medical. 11300 49th Street North, Clearwater, FL 33762.
\textsuperscript{11} \url{http://www.animalpain.com.br/en-us/avaliacao-da-dor-em-gatos.php}
\textsuperscript{12} \url{https://www.csuanimalcancercenter.org/assets/files/csu_acute_pain_scale_feline.pdf}
\textsuperscript{13} \url{http://carrboroplazavet.com/clients/15178/documents/feline_pain.pdf}
\textsuperscript{14} University of Florida Gator RAP Workshop: \url{http://www.gatorrapworkshop.com/}
\textsuperscript{15} Cornell University College of Veterinary Medicine Veterinary Regional Anesthesia Boot Camp: \url{http://www.event.com/events/2016-veterinary-regional-anesthesia-boot-camp/event-summary-148d50e2b5a448e78cde7d6d5f6c6b1c.aspx}
\textsuperscript{16} \url{http://partnersah.vet.cornell.edu/peripheral-nerve-blocks-dvd}
HOW TO INCORPORATE INTEGRATIVE MEDICINE IN TO YOUR CURRENT PRACTICE
Lisa Sams Ebner, DVM, MS, DACVAA, CVA

Veterinary medicine has become much more supportive in the last decade of the integrative approach to treating a patient. There are many terms floating around the vocabulary of both health care team members and owners. So it helps to be on the same page with what these terms actually mean. Integrative medicine is an approach to health care of the pet that combines conventional (or Western) medicine with complementary and alternative therapies available to the veterinarian. The focus of this approach to veterinary practice is treating the animal as a whole, so attention is given to the mind and spirit as well as the body. The term “holistic medicine” may be used interchangeably with integrative medicine. Complementary and alternative veterinary medicine therapies can include acupuncture, herbal medicine, massage, chiropractic, nutraceuticals, and energy therapy. The term “complementary medicine” implies these therapies are used along with conventional medicine. The term “alternative medicine” implies these therapies are used in place of conventional medicine. The focus of this presentation will be the modalities and therapies available to general practitioners.

Acupuncture is an excellent option for many veterinary patients, not just arthritic dogs. There are many conditions that can respond to treatment with acupuncture such as neurological, musculoskeletal, behavioral disorders, gastrointestinal, respiratory tract diseases, and other various internal medicine conditions. Some conditions will respond better than others, therefore, it is important to keep this in mind when selecting a case to use this modality in. Functional disease is going to respond better than structural disease. The owner should also be given realistic expectations for this therapy. One treatment will not cure a chronic disease. The author asks her clients to be willing to have their pet undergo three treatments before they decide whether or not the acupuncture is right for them to continue. There are several options for the general practitioner that would like to become certified in veterinary acupuncture, each certifying program having a slightly different approach but the same goal in mind for the patient. Even if a practitioner does not wish to become certified in acupuncture, there are still a few acupuncture points that can be very useful to incorporate in to everyday practice. The location of these points requires knowledge of anatomy and the use of acupuncture needles (a box of 100 needles cost around $7). Referral to a certified veterinary acupuncturist may be the best choice for certain clients.

Rehabilitation therapy is probably the most main stream integrative modality used in veterinary medicine. Two program exist in the United States to become certified in rehabilitation therapy for pets. However, even without completion of a certification program and access to expensive equipment a general practitioner with proper training can incorporate basic rehabilitation therapy techniques for patients as needed. Some inexpensive techniques include range of motion and stretching exercises, thermotherapy, cryotherapy, massage therapy, proprioception and balance exercises. With more investment in equipment and training modalities such as aquatic therapy, low-level laser, therapeutic ultrasound, and transcutaneous electrical nerve stimulation can be incorporated for select patients.
A plethora of nutraceutical or dietary supplements are found on the market. It can be slightly overwhelming for a general practitioner to have a working knowledge of what each one claims to do for a patient and the safety of each substance. Studies supporting the use of these substances are lacking in the literature. So it often comes down to anecdotal evidence for their use. Some of the more mainstream supplements include omega-3 fatty acids, avocado/soybean unsaponifiables, polysulfated glycosaminoglycans, glucosamine and chondroitin. The use of herbal therapy for a veterinary patient can only be recommended if the practitioner has formal training in this area, as more harm than good can be done to the patient if an incorrect prescription is made.

Literature supporting the use of aromatherapy in veterinary patients is sparse. One study (Wells 2006) evaluated the use of diffused lavender in canines with travel-induced excitement and the results indicated that it could be a reasonable alternative to traditional treatments. The use of aromatherapy, particularly undiluted essential oils, in pets should be very carefully considered due to the potential for toxicity or irritation. One retrospective study (Khan 2014) evaluated the incidence of toxicosis in dogs and cats that were exposed to 100% tea tree oil. Use of this oil in both species is associated with salivation, central nervous system depression, and tremors. Cats are particularly sensitive to certain essential oils, including cinnamon, oregano, clove, wintergreen, thyme and birch.

Animal chiropractic is a modality that focuses on the preservation and health of the neurological and musculoskeletal systems. A chiropractic adjustment involves short lever, high velocity controlled thrusts to correct vertebral subluxations. A certification program is available through the American Veterinary Chiropractic Association.

Energy medicine is an adjunctive therapy and can include methods such as Healing Touch for Animals® or Reiki. Patients with acute or chronic pain or other chronic illnesses may benefit from energy therapy.

References available upon request.
THE INS AND OUTS OF PERIOPERATIVE FLUID, ELECTROLYTE, AND BLOOD PRODUCT THERAPY
Lisa S. Ebner, DVM, MS, DACVAA, CVA

There have been some recent changes or shifts in the approach to perioperative fluid therapy. This presentation will discuss both crystalloid and colloid fluid therapy. A review of treatment of electrolyte imbalances and blood product therapy will also be covered. In 2013, AAHA and AAFP developed the Fluid Therapy Guidelines for Dogs and Cats from a panel of experts. Although these are not considered the “standard of care” they are practical recommendations for the veterinary health care team. Both maintenance and replacement fluids are discussed. Their recommendations for anesthetic fluids rates include: administration of IV fluids should be < 10 ml/kg/hr, the rate should be lower in cats (3 ml/kg/hr, initially) than in dogs (5 ml/kg/hr, initially), and lower in patients with cardiovascular or renal disease. Also, fluid administration rate should be reduced after one hour of anesthesia, typically by 25% every hour until maintenance rates are reached. For a patient with relative hypovolemia due to peripheral vasodilation, the guidelines suggest starting with an IV bolus of isotonic crystalloids at 3-10 ml/kg and this can be repeated once. If a patient does not respond adequately, then a slow IV bolus of a colloid can be given to dogs (5-10 ml/kg) and cats (1-5 ml/kg). These guidelines reflect the shift away from higher IV fluid administration rates that was typical in the past. The literature supports that higher fluid rates can actually do more harm than good, specifically decreasing pulmonary function, slowing GI tract motility, and development of dilutional coagulopathies.

Crystalloids replace interstitial fluid losses because 75% of isotonic fluids administered intravenously wind up in the interstitial space within about an hour. So generally, the volume of fluids to replace a deficit in the intravascular space is multiplied by about 3. Isotonic fluids (meaning the sodium content is similar to a cell) include Lactated Ringer’s solution (LRS), Plasmalyte-A®, Plasmalyte148®, Normosol-R®, normal saline (0.9%), and 2.5% dextrose in 0.45% NaCl. Physiologic saline (0.9% NaCl) is not buffered and is not considered a balanced solution (meaning that it does not contain electrolytes found in the plasma). Physiologic saline will dilute the plasma bicarbonate level, therefore it is an acidifying solution and with the high concentration of chloride it can also lead to hyperchloremic metabolic acidosis. An advantage of 0.9% NaCl is that it is the least likely to worsen brain edema in patients with head trauma due to the higher level of sodium compared to a more hypotonic fluid such as LRS, which has been associated with increased intracranial pressure in studies of traumatic brain injury (Pinto et al. 2006). LRS, which is almost identical to Hartmann’s solution, contains lactate which is metabolized by the liver. This consumes hydrogen ions and generates bicarbonate, which produces an alkalinizing effect in the body and therefore makes this fluid a good choice for patients with metabolic acidosis. LRS (or Hartmann’s solution) would not be the best choice for treatment of patients with lactic acidosis or potentially for a patient with hepatic dysfunction.

1Baxter Healthcare Corporation. Deerfield, IL 60015.
3 Hospira, Inc. Lake Forest, IL 60045.
4 Zoetis, Inc. Kalamazoo, MI 49007.
Normosol-R® may not be the best choice for fluid resuscitation in a patient with shock due to the buffer being acetate, which is mostly metabolized in the muscles and causes release of adenosine which causes vasodilation and possibly hypotension. Also, the magnesium may lead to vasodilation and worsening of hypotension.

Hypertonic saline (usually 7.5% NaCl) at 4-6 ml/kg can be used to draw fluid from the cells and interstitial space into the intravascular space for patients that acutely have a need for volume resuscitation. It also helps increase cardiac output and has beneficial immunomodulatory effects for the patient. Recent evidence in the literature suggests that hypertonic saline is a superior choice to mannitol for managing intracranial hypertension (Kamel et al. 2011; Mortazavi et al. 2012). However, appropriately selected fluid administration should immediately follow the hypertonic saline to replace total body water deficits. Another technique is combining hypertonic saline with a colloid for administration (aka “turbostarch”). A typical dose for this combination would be 4 ml/kg of each. Hypertonic saline administration should not be given to a patient with uncontrolled hemorrhage as this can lead to increased bleeding and a worse outcome.

Hypotonic fluids, such as D5W (5% dextrose in water) should not be used for fluid resuscitation because the fluid will shift to the intracellular space and cause swelling of cells.

Colloids replace volume in the intravascular space due to the larger molecule size preventing leakage from the capillaries. Hydroxyethyl starch (HES) solutions have fallen out of favor due to safety concerns for critically ill (human) patients such as increased incidence of renal replacement therapy and risk of bleeding following their use in certain populations of patients. In 2013, the FDA added a Boxed Warning label about the adverse effects of HES products. VetStarch™ is rapidly degradable tetrastarch product currently available to veterinarians. It has an advantage over other HES products in that up to 50 ml/kg/day can be used in a patient due to lower levels of tissue accumulation. When higher dosages of HES are given, coagulation may be impaired and the patient may have more bleeding during surgery due to dilutional coagulopathy effects of HES solutions. However, veterinary studies have failed to demonstrate this clinical bleeding effect. There are also no veterinary studies showing a link between renal failure and use of HES solutions. Therefore, the author routinely uses a tetrastarch solution to manage hypotension in anesthetized patients. But it would be prudent to avoid their use in a critically ill patient, particularly one with renal compromise. A typical bolus dose is 5 ml/kg (dogs) or 3 ml/kg (cats) IV over about 15 minutes.

Another trend in fluid resuscitation for veterinary patients is “early goal-directed therapy” with various endpoints being established in the literature. Simple to measure endpoints include heart rate, blood pressure, mucous membrane color, CRT, and pulse pressure. Other endpoints that can be measured with more diagnostic tools include blood lactate levels, central venous pressure, central venous oxygen saturation, and urine output.

Crystalloids or synthetic colloids can be used to replace intravascular volume in mild to moderate cases of blood loss. But in a severe case of blood loss, blood products are indicated. Blood products utilized in the perioperative period may include packed RBCs, whole blood, plasma or cryoprecipitate. Species differences exist in regards to blood volumes, with dogs having a total
blood volume of 88 ml/kg and cats having 68 ml/kg. Blood loss should be estimated during surgery by evaluating gauze squares, lap sponges, and the suction canister. These items can also be weighed to determine amount of blood loss as every gram equals 1 ml of blood. A patient who has lost one third of their red cell mass acutely will require increased oxygen carrying capacity. A transfusion with blood to meet oxygen transport needs should be considered when hemoglobin concentration is below 7 grams per deciliter (or a hematocrit of 21%). When considering transfusion in specific patients, the clinician should consider age, etiology and duration of anemia, presence of coexisting cardiac, pulmonary, or vascular conditions, and hemodynamic stability. There are formulas to determine the mLs of blood required for a dog or cat, but it works out to be about 10-40 ml/kg in dogs and 5-20 ml/kg in cats. Whole blood or packed RBCs can be given at a rate of 5-10 ml/kg/hr. The first 30 minutes should be slower (0.25 ml/hr) to monitor closely for any sign of a reaction. It is important to note that general anesthesia may mask some of the signs of a transfusion reaction. So when in doubt, the transfusion should be stopped. The transfusion should be completed in 4 hours to prevent bacterial contamination and loss of function of the blood product. A transfusion can go faster in a critical situation.

Electrolyte imbalances to consider treating prior to anesthesia include potassium, sodium and calcium imbalances. Acute hyperkalemia, especially if it causes changes in the ECG, should be treated prior to anesthesia. Treatment involves administration of calcium to protect the myocardium (but calcium does not lower the potassium concentration), fluid diuresis, and giving dextrose with or without insulin to move the potassium to the intracellular space. Bicarbonate can be considered in a patient depending on the acid-base status. Hypokalemia may warrant treatment prior to anesthesia if the patient’s potassium concentration is < 2.5 mEq/L. However, potassium supplementation should not exceed 0.5 mEq/kg/hr and fluids containing potassium supplementation should never be used for giving a bolus of fluids to a patient.

Acute hypernatremia (< 24 hours duration) can be more rapidly corrected. If the patient has been chronically hypernatremic, then a slower correction should take place over 2 to 3 days (Na⁺ should not decrease more than 0.5 mEq/L/hr). Calcium levels should be confirmed by an ionized calcium concentration. Hypocalcemia is frequently seen in sick animals presenting for emergency anesthesia and could lead to problems such as hypotension, tachycardia, hyperthermia, and cardiopulmonary arrest. Treatment involves administration of calcium gluconate (10%) at 0.5-1.5 ml/kg IV slowly (over 20 minutes) while continuously monitoring the ECG.
There is no one “recipe” that works for every patient being anesthetized. So the importance of a good physical exam and a minimum database should be emphasized to detect abnormalities that can be corrected prior to anesthesia.

References available upon request.
Including local anesthesia as part of a balanced anesthesia plan is simple to do and does not require expensive equipment. Their addition to general anesthesia is helpful because it reduces the amount of other systemic drugs required, leads to a smoother recovery, and post-operative pain is easier to manage. There are many local anesthesia blocks that can readily be incorporated by any general practitioner. These will be discussed in more detail during this lecture. But there are a few other local anesthetic techniques that will be covered for the practitioner that is willing to practice them and/or use more advanced equipment to be certain the block is successfully performed.

A brief review of the mechanism of action of local anesthetics is beneficial for understanding how they add to your analgesic plan. Local anesthetics block the initiation and conduction of action potentials in nerve fibers. So transmission of nociceptive signals are prevented and the patient does not sense pain. Small diameter (C and Aδ) nerve fibers are blocked preferentially prior to large myelinated fibers (Aβ), so a loss of sensation occurs with varying degrees of loss of motor function. Analgesia is a direct result of sodium ion channel blockade and membrane stabilization. The lipophilic portion of a local anesthetic is unionized, allowing it to cross the cell membrane. Once inside the cell it become ionized and then blocks the sodium channel. This prevents membrane depolarization and propagation of the action potential in the neural tissue. The patient has a dose-dependent loss of sensory, motor, and autonomic function. This effect is transient as the local anesthetic is metabolized or hydrolyzed (see figure 1).

Some of the commonly used local anesthetics in veterinary medicine are compared by their onset, duration, and dose which can cause toxicity (see Table 1). The signs of local anesthetic toxicity can be agent dependent. Generally, CNS signs occur prior to cardiovascular signs. But with bupivacaine, cardiac toxicity signs occur simultaneously with CNS signs. So with bupivacaine, arrhythmias could occur at the same or lower dose that produces seizures. Lidocaine toxicity typically results in CNS excitation and convulsions, but bradycardia and hypotension can result with higher doses. Treatment of CNS toxicity can include support of ventilation, oxygen supplementation, and seizure control. Treatment of cardiovascular toxicity may include blood pressure support, monitoring of ECG, administration of Intralipid1, and cardiopulmonary resuscitation if needed.

There are four main dental nerve blocks that can be utilized depending on the type and site of surgical procedure being performed on the dog or cat. These include the mental, mandibular, infraorbital, and maxillary nerve blocks. The nerve block only has a unilateral effect. It is important to carefully aspirate prior to injection of drug due to the close proximity of blood vessels. A typical volume per site is 0.25 mL in cats and up to 0.75 mL depending on the size of the dog. A 25-27 gauge, one inch needle should be used.

The radial/ulnar/median nerve block, also known as a ring block, is typically used in felines presenting for onychectomy. While the performance of this surgery is a controversial matter,
what is not controversial is the recognized need for adequate analgesia for the patient. Performing this local block prior to surgery will decrease the general anesthetic requirements and improve the recovery period. There are several suggested protocols, but most important is remembering that cats are more sensitive local anesthetic and can develop toxicity at a lower dose than a similar sized dog. A suggested technique is to combine 0.5 mg/kg of both lidocaine and bupivacaine. An opioid such as buprenorphine or morphine could also be added to the block.

Intra-articular analgesia can be used in any joint surgery, for chronic pain from OA, and as a diagnostic tool for confirming joint pain. The treatment is usually inserted steriley after joint exploration and closure. Local anesthetic can be used, although there is some concern for toxic effects on the chondrocytes. This seems to be a dose and time dependent effect, so a one-time use would not be harmful. Morphine (0.1 mg/kg) has also been used in chronically inflamed joints.

Intravenous regional anesthesia, or a Bier block, can be useful for procedures on the distal limbs such as a mass removal. Some simple equipment that will be needed are a tourniquet, esmarch bandage to desanguinate the leg, and an intravenous catheter. It is important to note that only lidocaine should be injected intravenously!

Intercostal nerve blocks are useful for patients having a thoracotomy or presenting for fractured ribs that are experiencing pain during ventilation. Due to the anatomy of the nerve supply, at least two adjacent intercostal spaces cranial and caudal to the origin of the pain must be blocked. The practitioner must be careful not to enter the chest and risk creating a pneumothorax.

Infiltrative blocks, also known as field blocks, are used in superficial areas of the skin. They are useful for biopsies and prior to or after a surgical incision. One study showed that pre-operative administration of bupivacaine to dogs resulted in significantly lowered pain scores and a decrease in administration of additional analgesics in the post-operative period (Savvas et al. 2008). There is a concern about impairment of wound healing due to the local anesthetic, but this has also been seen with infiltration of saline. Other options for topical and infiltration anesthesia include the use of EMLA cream\(^2\), Lidoderm\(^3\) patches, proparacaine\(^4\) ophthalmic solution, and performing a “splash block” in to the surgical wound. Another option for continued infiltration of local anesthetic in a surgical site is a wound soaker catheter. Placing a wound catheter is simple to perform and a patient can be sent home sooner to be managed by the owner at home. Lidocaine or bupivacaine can used with this technique. Complications are minor and can include dislodgement of the catheter.

An intratesticular block is a very simple technique that should be used in all dogs and cats being neutered. A dose of 2 mg/kg lidocaine can be split between the two testicles and to block the skin for the incision. It should be performed after the patient has been clipped and prepped. A 22-25 gauge needle is used and aspiration should occur prior to injection. Lidocaine travels up the spermatic cord and achieves the maximum effect in a few minutes.
A retrobulbar block with bupivacaine can be performed in patients having an enucleation. A 22 gauge, 1.5” needle is bent about 15 degrees and placed ventral to the eye along the lateral third of the lid, dorsal to the zygomatic arch. The needle is directed caudomedially. There are risks associated with this technique, so an alternative is to place a splash block in the orbit prior to closure.

A lumbosacral epidural is useful for patients having orthopedic surgery of the hind limb, urogenital surgery, and abdominal exploratory surgery. Post-operative analgesia can last 12-24 hours after administration of the epidural. Ideally, a preservative-free morphine is used at 0.1 mg/kg. The patient can be in lateral recumbency or sternal recumbency (with the hind limbs pulled forward). The landmarks and technique are described in detail in many resources. In cats, a sacrococcygeal approach is described for use in feline urethral obstruction. An epidural catheter can be placed for long-term, multi-injection use. This technique requires more skill of the practitioner and access to the specialized type of catheter used for this purpose.

Contraindications for an epidural include pyoderma or neoplasia at injection site, bleeding disorders, uncorrected hypovolemia and hypotension, sepsis, anatomical abnormalities, and preexisting neurological deficits to the area being blocked.

A brachial plexus block can be utilized in a patient requiring analgesia for the front limb distal to and including the elbow. The anatomic landmarks have been described for this technique, but it is ideal to use a nerve stimulator and insulated needle to improve accuracy by guiding the placement of local anesthetic close to the nerves. It is important to prevent the needle from puncturing the thorax and the large vessels near the brachial plexus.

The RUMM block provides anesthesia for a procedure in the distal thoracic limb by blocking the radial, ulnar, median, and musculocutaneous nerves. This technique has been described in detail by Trumpatori et al. (2010). Ideally, a nerve stimulator and insulated needle are used to locate the nerves.

Ultrasound-guided femoral and sciatic nerve blocks are the latest trend in veterinary analgesia. They are taking the place of the lumbosacral epidural for patients having surgery distal to the mid-femur. There are less potential complications than an epidural, but requires more technical skill by the practitioner and access to an ultrasound. There are several techniques described in the literature. Bupivacaine combined with dexmedetomidine is most commonly used.

There are a few clinical caveats to keep in mind when using a local anesthetic technique. These include considering that acidotic tissue will delay the onset of the local anesthesia, avoiding benzocaine-containing topical solutions in cats, and combining a local anesthetic with another drug (such as another local anesthetic, an opioid, or epinephrine) and what effect that might have on the patient.

References available upon request.
Figure 1. Sodium channel blockade by local anesthetic in the nerve cell

<table>
<thead>
<tr>
<th>Drug</th>
<th>Concentration (mg/ml)</th>
<th>Onset (min)</th>
<th>Duration (hr)</th>
<th>Toxic dose (mg/kg IV)</th>
</tr>
</thead>
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<tr>
<td>Mepivacaine</td>
<td>10</td>
<td>1-2</td>
<td>2-3</td>
<td>29</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>20</td>
<td>2-3</td>
<td>1-2</td>
<td>6-12 (cat)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>10-20 (dog)</td>
</tr>
<tr>
<td>Bupivacaine</td>
<td>5</td>
<td>10</td>
<td>4-6</td>
<td>2.0-3.8 (cat)</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>3-5 (dog)</td>
</tr>
<tr>
<td>Ropivacaine</td>
<td>2, 5, 7.5, 10</td>
<td>Shorter onset than bupivacaine</td>
<td>5-8</td>
<td>4.9</td>
</tr>
</tbody>
</table>

Table 1. Comparison of commonly used local anesthetics in veterinary medicine
DEALING WITH ANESTHETIC COMPLICATIONS...WHAT’S YOUR PLAN?
Lisa Sams Ebner, DVM, MS, DACVAA, CVA

There are several complications that can occur in the peri-anesthetic period that are detrimental to the health of the patient and also cause the medical team considerable anxiety. Owners are often warned to avoid anesthesia in their pets for fear of these complications occurring. With some planning on the part of the anesthetist, careful evaluation of the patient, and selection of drugs and/or interventions when needed the patient can be safely taken through an anesthetic episode.

Complications can fall into a few categories: common, uncommon, complications of the surgical or diagnostic procedure, human error or idiosyncratic reactions. The focus of this presentation will be on dealing with common anesthetic complications seen in patients anesthetized by general practitioners. A brief review of preventable complications will be reviewed to remind the practitioner that simple steps taken by the health care team can avert a regrettable outcome.

The common complications that are general considerations of inhalant anesthesia include hypotension, hypoventilation, hypothermia, and hypoxemia. The author likes to refer to these as the 4 H’s. Regardless of the drugs selected and the health status of the patient, inhalant anesthesia can lead to one or more of these complications on a regular basis. This emphasizes the importance of adequate monitoring during the anesthetic episode. The approach the author takes to dealing with any complication that occurs while the patient is under general anesthesia is to always have a plan. So starting with plan A, followed by plan B, plan C, plan D, etc. until the problem is solved.

Inhalant anesthetics, such as isoflurane or sevoflurane, cause dose-dependent vasodilation and therefore can lead to hypotension. Hypotension is defined as a systolic arterial pressure below 80-85 mmHg or a mean arterial pressure below 60 mmHg. Blood pressure is commonly measured by oscillometric devices or ultrasonic Doppler flow detectors, with the latter accepted as being more accurate. In dogs, the systolic blood pressure reading obtained with the Doppler should be $\geq 90$ mmHg in order to feel comfortable that the patient’s mean arterial pressure is above 60 mmHg. This mean arterial pressure cutoff is important in the auto-regulation of blood flow to vital organs, such as the kidneys. The first plan (plan A) when hypotension is detected should be to check the level of anesthetic depth in the patient and consider turning the vaporizer down. Often an intravenous fluid bolus (plan B), typically the patient’s hourly fluid rate over about 10 minutes, is concurrently administered depending on the severity of the hypotension. The heart rate should also be evaluated in conjunction with the blood pressure. Plan C may involve administration of an anticholinergic if the patient is bradycardic and it is indicated. Continued hypotension is an indication that the patient may need vasopressor or positive inotropic drug support, such as a dopamine or dobutamine continuous rate infusion.

Hypoventilation is defined as an end-tidal carbon dioxide (ETCO2) level of greater than 45 mmHg detected by capnography. The use of capnography during anesthesia is an excellent tool that should ideally be used in all veterinary patients. However, due to the increased cost associated with this modality it is not always readily available to general practitioners. Other
subjective ways to detect hypoventilation in your patient is to observe the chest wall excursion on the patient and to monitor movement of the reservoir bag. A simple solution for hypoventilation is to turn the vaporizer down and administer intermittent positive pressure ventilation either manually or with a mechanical ventilator.

Hypothermia is especially common in smaller veterinary patients due to greater surface area-to-mass ratio. Preventing hypothermia during anesthesia is often easier than trying to treat it once it is already fairly moderate to severe. The anesthetist can minimize heat loss due to conduction by placing a warm-water circulating blanket or towel between the patient and the cold surgery table. A forced air warming blanket can also be incorporated around or on top of the patient. Other inexpensive heating techniques can include placing children’s socks or bubble wrap on the feet, heating up rice in tube socks to place next to patient, placing a space blanket over the patient. Intravenous fluid warmers can be helpful if they are placed close to the patient. Keeping the oxygen flow rate as low as safely possible will slow down the cooling effects of the oxygen on the respiratory tract.

Hypoxemia is not as common as the other 3 H’s, but has five causes that include hypoventilation, V/Q mismatch, decreased inspired oxygen concentration, a right to left shunt, and diffusion impairment. So the underlying cause of hypoxemia should be determined if possible as this will guide the treatment for the patient.

A thorough evaluation of the patient prior to anesthesia should include auscultation of the heart and palpation of the femoral artery for the presence of any pulse deficits. Any abnormality should be evaluated by electrocardiogram and possibly consultation with a cardiologist so the owner can be fully informed of the level of risk associated with their pet having anesthesia. The arrhythmia should ideally be stabilized prior to elective anesthesia. Cardiac arrhythmias should be carefully monitored under anesthesia, but may not require treatment if the blood pressure is stable and the arrhythmia is not worsening during the anesthetic episode.

Dysphoria in recovery should be quickly addressed to prevent the patient from harming itself or the caretakers in the immediate vicinity. Dysphoria should be distinguished from pain, but when in doubt an analgesic may be administered and the patient monitored carefully for a response. Typical treatment for dysphoria can include low-dose acepromazine or dexmedetomidine given intravenously.

Preventable complications in the anesthetic period can include human errors or equipment malfunction. Human error is not a matter of “if” but “when” it occurs. If a complication does occur, it is important to keep a level head and quickly communicate the problem to the peri-operative team. Holding a morbidity and mortality rounds afterwards to discuss complications will help to develop a plan to prevent or reduce future occurrences.

References available upon request.