Dog Owners and Breeders Symposium
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College of Veterinary Medicine

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*Cancer Treatment Update
*Endocrine Update
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Cancer Treatment Update
Dr. Rowan Milner, BVSc, MedVet
University of Florida

Introduction

Veterinary oncology is undergoing fundamental change. The old paradigm of "if it can't be surgically removed then euthanasia is the next step" no longer applies. The reason for this change is due to the following factors: pet owners are becoming more sophisticated in their requirements for specialized treatment, improved results due to better treatment protocols and drugs, a heightened awareness in private practitioner of the early signs of neoplasia leads to earlier detection of cancer leading to more successful treatment, and an increase in age of the pet population has lead to a population of pets with a greater risk of developing cancer.

A very important consideration in veterinary oncology is client counseling. Cancer has a very emotional connotation and requires tact and patience in giving "bad news" to the owner. In the medical field, physicians are taught to steer the middle course by using language that would dispel ignorance without dispelling hope. Veterinarians need to communicate clearly the options available to the owner. A recent report on people's response to bad news cited most respondents as retaining only about 15 percent of what was told to them on the first visit. It is therefore imperative to supply literature and information to owners about their pets' cancers that can be read at a leisurely pace. Veterinary oncology has learned a tremendous amount from the human experience, however there are some major differences. In animals, cure is often not a realistic goal but rather quality of life, and therefore when chemotherapy is used drug dosages are lower. As a result, they do not experience the same side effects associated with chemotherapy in man. However, complications associated with treatment do occur, e.g. drug induced pancreatitis, and bone marrow suppression.

Cancer is essentially a genetic disease. Two of the most important discoveries have been the identification of genes called oncogenes (onco meaning cancer) and cancer suppressor genes. *Oncogenes* exist in the normal cell but when cancer occurs, depending on the cause, these genes are modified. These genes are involved in regulating cell activity, e.g. growth and protein production, and when changed uncontrolled growth occurs. *Cancer suppressor genes* on the other hand, act to suppress uncontrolled cell growth. When they are changed cells develop "immortality." The normal check and balances that determine if a cell should divide are lost. It is worth noting that unlike oncogenes, cancer suppressor genes may be found in germ cells, allowing the defect to be passed from one generation to the next. It has become clear that no single gene is responsible for the transformation of a cell, but rather two or more oncogenes as well as cancer suppressor genes are needed.

There are also a number of pathways that may be involved in the generation of a particular cancer. These are via: heritable carcinogenesis, passive carcinogenesis, chemical carcinogenesis and physical carcinogens. Cancers are a collection of cells that are always changing. This phenomenon is termed *cancer progression*. Cancer progression generally results in disease that is more aggressive. This may partly explain the poor prognosis of cancers that are detected late. The major distinguishing feature between benign or malignant cancers is the ability of malignant

cancers to spread to distant sites.

Clinical Staging

Staging is the process of investigating and documenting the extension of a cancer within the body. This is achieved by physical examination, X-rays, ultrasound, CT, nuclear medicine MRI and biopsy.

Treatment of Cancers

Cancers are treated in several ways depending on their position, malignancy and tissue type. Most solid cancers are treated using surgery followed by chemotherapy and/or radiation.

Chemotherapy

Other than for cancers arising from immune system and bone marrow, chemotherapy is employed in combination with other treatment options (e.g. radiation and/or surgery). Chemotherapy's main function in these cases is to mop up leftover cancer cells or to suppress distant metastases. Few chemotherapeutic agents are without side effects and in some cases these toxicities are life threatening. Resistance to treatment, as is seen with antibiotic resistance, also occurs with chemotherapy and leads to therapy failure.

Radiation Therapy

Radiation therapy involves the treatment of cancers with radiation delivered by way of either modified X-ray machines (orthovoltage or linear accelerators) or radioactive sources such as cobalt-60. Radiation therapy works by causing free radicals (reactive molecules), which then damage the cells' DNA. Cells then die when they reach the stage of wanting to divide. This process is dependent on the susceptibility of the tissue and the presence of oxygen. Typically, tissue such as brain is resistant to the effects of radiation since the cells are not dividing whereas bone marrow cells are very susceptible due to their rapid division.

Surgery

This is the oldest method and still the most effective of treating cancer in the early stage. Surgical techniques are advancing all the time, which enable veterinary oncologists to achieve the ultimate goal of cure. In most cases of malignant cancer, surgery forms part of the therapy that may include chemotherapy and radiation.

Nutrition

Nutrition is probably the most neglected part of therapy for cancer. Research has shown that there are substantial changes in the metabolism of fats, carbohydrates and protein that occur in a patient with cancer. A specific cancer diet has been formulated by Hills known as n/d. This diet contains high levels of essential fatty acids (FFA) in the form of omega-3 FFA (fish oil is high in omega-3). Omega-3 has been shown to aid in the reversal of the metabolic changes seen with

cancer. This diet also has reduced levels of carbohydrate, the reason for this is that cancers prefer sugars to metabolize and produce less energy from this metabolism. The body has to then convert the lactic acid from this cancer metabolism back to a usable fuel for cells at a net loss of energy to the body. Correct nutrition in cancers will starve the cancer of nutrients and thereby aid in the treatment.

Alternative

Alternative therapy covers all the aspects, from herbal remedies to acupuncture. While these therapies may be beneficial, the vast majority of them have not been subjected to close scientific scrutiny. The use of antioxidants are beneficial in preventing cancers, however, they may be contraindicated during therapy. The reason for this is that most drug and radiation therapy for cancer work via the production of oxidants (free radicals) and concurrent use of antioxidants may cause the drugs to work sub optimally.

When we evaluate the effectiveness of therapy, we use the following terminology. The terms used by definition are:

Complete Response (CR): Disappearance of all cancer in all sites for a defined period of time.
Partial Response (PR): Decrease in size of all cancers by 50 percent or greater as measured by the sum of the products of two diameters for each cancer. There should be sustained decrease in cancer size, as defined for CR, and no new cancers should arise.
Stable Disease (SD): Decrease of 50 percent or an increase of 25 percent in the sum of the products of the diameters as measured for PR.
Progressive Disease (PD): Increase of 25 percent or more in the sum of the products of cancer diameters or the appearance of a new cancer.

Note: It should be remembered that remission does not necessarily mean cure but rather the disappearance of clinically detectable disease.

Common Cancer Types

Lymphoma in Dogs:

Dogs with lymphoma (cancer of the lymphoid tissue) typically present with enlarged glands, either locally or all external glands. Since the body has lymphoid tissue everywhere, it can occur in any organ. However, the multicentric (all peripheral glands form is the most common). When veterinarians stage lymphoma in the dog, the most important criteria they look for are: extent of the disease (does it involve the spleen and/or liver), whether or not the dog has systemic signs (e.g. weight loss, excess urine production), and type of lymphoid cell involved. These criteria are used to determine the chances for a meaningful response to treatment. Veterinary oncologists will treat most lymphomas as the therapy relieves many of the symptoms of the cancer and response can sometimes be unpredictable.

Treatment can take various forms depending on the site and extent of the lymphoma. However, chemotherapy remains the mainstay of treatment. Drugs most commonly used are cortisone,

alkylating agents and anthracycline antibiotics. Most treatments consist of multiple drugs, although there are single drug treatments. Therapy is divided into an induction period of 4-6 weeks followed by maintenance treatment for 6-24 months. The most effective combination is known as the Wisconsin-Madison protocol. This protocol gives the best remissions and survival times. However, since it has multiple drugs it also has a moderate level of toxicity.

The goal of treatment is to achieve a good remission of the cancer. Remission rates vary from three months to as much as 36 months or longer. Generally, a stage III lymphoma with no complications can be expected to remain in remission for 6-9 months followed by re-induction of remission for, on average, another 3-6 months. Cure of canine lymphoma is only rarely achieved.

Lymphoma in Cats

Lymphoma in the cat is similar to dogs, but with a number of exceptions. When cats are feline leukemia virus positive, their survival times are reduced. The more common form of lymphoma in cats occurs in the intestines and chest cavity. Cats generally have longer survival times than dogs. Therapy is similar albeit at a lower dose.

Perianal Cancers:

These are cancers arising around the anus. They are more common in the male dogs than in female dogs. The most common cancers are perianal adenoma (80 percent), followed by adenocarcinoma and anal sac adenocarcinoma (apocrine gland). Perianal adenoma occurs more commonly in intact males and older large breeds of dog. Testosterone secretion from the adrenal glands (with or without Cushing's) may stimulate perianal adenomas. Treatment includes surgical removal of the mass and castration.

Perianal adenocarcinoma is also seen in male dogs and metastasizes locally to the regional lymph node. Prognosis is good if the mass is smaller than five centimeters and can be removed completely with surgery. Castration does not make a difference. Anal sac adenocarcinoma (apocrine gland) is a highly malignant cancer with an increased occurrence in old female dogs. The cancer is often associated with increased urine production resulting in increased water intake. This is due to high calcium levels in the blood. Prognosis is generally poor as the cancer shows early spread and is often resistant to treatment.

Hemangiosarcoma

Hemangiosarcoma is a highly malignant cancer of the blood vessel wall. It is more common in the dog and occurs in the spleen, right atrium of the heart and skin. Large breeds of dog appear to have a greater incidence with German Shepherd Dogs having the highest incidence. Because this cancer occurs in deep organs, owners are often unaware that their dogs have the tumor until it suddenly ruptures, which leads to bleeding either in the abdomen or around the heart. These dogs commonly present in a shocked state with white gums and abnormal rhythm to the heart. Dogs may even die suddenly for no apparent reason. The best instrument to diagnose the presence of the cancer is ultrasound. When hemangiosarcoma occurs in the skin it can be due to

sun damage or metastasis from a distant site (e.g. spleen). Therapy for this cancer is generally unrewarding except for the skin (sun-induced) cancer. Average survival following removal of a spleen is three months.

Mammary Neoplasia

Mammary cancer in dogs is seen most often in older intact females. There is a clear statistical link between time of spaying a dog in relation to the number of seasons she has had. The earlier she is spayed the less likely she will develop mammary cancer; so much so that there is even a difference between first and subsequent seasons. Typical presentation is, as in humans, a lump that is felt within the mammary gland. Fifty percent of these turn out to be malignant which go on to spread to the local gland and then to the lungs. Treatment is mainly surgical with radiation and chemotherapy in some cases. Surgery invariably means radical mastectomy. In benign tumor lumpectomy can be done.

Breast cancer in the cat is a far more malignant cancer. Seventy-five to ninety-five percent of mammary cancers in cats turn out to be malignant, with a large number showing spread to the lungs on presentation to the veterinarian. Treatment in the early stage consists of surgery followed by chemotherapy.

Osteosarcoma

Osteosarcoma is a cancer of the bone-producing cells of the body. It is a highly malignant cancer, which results in destruction of the bone and in most cases early spread to the lungs. It is a particularly painful cancer, as are most bone cancers. The cancer occurs most commonly in giant breeds of dog, although not exclusively. The most common bones affected are the long bones of the limbs with the site on the bone being "away from the elbow towards the knee." The current gold standard for treatment is surgical amputation followed by chemotherapy using a drug called carboplatin. The author (University of Florida) is researching alternative therapies in dogs that cannot undergo amputation. Currently two clinical trials are running.

Mast Cell Cancer (MCT)

Mast cells occur throughout the body and take part in the inflammatory reaction associated with allergies. They contain histamine and other pro-inflammatory agents. Because of the histamine contained in MCT, these cancers can cause gastric ulceration and acute allergic reactions. Mast cell cancer commonly arises on the skin but can occur elsewhere. When veterinarians stage an MCT, they take into account the following criteria: position on the body, grade of the cancer, how quickly it grew, and the breed. Criteria that carry a poor prognosis are high grade MCT located in the inguinal (groin) area that have grown rapidly. Treatment varies depending on the former criteria but may include at the very least surgery and/or chemotherapy and possible radiation therapy.

Those Interesting Hypercalcemia Syndromes
Michael Schaer, DVM

Hypercalcemia occurs when the serum calcium concentration exceeds 11 mg/dl. In dogs, the most frequent causes include primary hyperparathyroidism and hypercalcemia of malignancy (pseudohyperparathyroidism), the latter being the most common. Bony spread of malignant neoplasms, kidney disease and certain inflammatory diseases can also cause hypercalcemia. Minor serum calcium elevation can also occur with Addison's disease, but it is readily rectified with fluid therapy. Other possible causes include hypervitaminosis D and day-blooming Jessamine ingestion. The newer cholecalciferol-containing rodenticides pose a particular threat to the pet animal.

Signs: The patient with hypercalcemia can show anorexia, nausea, vomiting, weakness, abdominal pain, constipation, increased thirst and urination, dehydration and depression. Hypercalcemia can adversely affect brain function, nerve conduction to muscles, cardiac excitation and kidney function.

Diagnosis: The initial medical workup should be extensive and include serum level determinations of calcium, phosphorus, sodium, potassium, chloride, creatinine or urea nitrogen, alkaline phosphatase and proteins. A complete urinalysis, CBC, as well as chest and abdominal radiographs should also be done. Further evaluation might require renal creatinine and phosphate clearance determinations, radioimmunoassay determination of plasma parathormone levels and a bone marrow cytology evaluation. Certain cancer syndromes can have increased plasma levels of parathyroid hormone related protein (PTHrP). The initial objectives are to stabilize the patient while diagnosing the cause. When all extra-parathyroid causes of hypercalcemia are eliminated, the primary focus should then concentrate on the parathyroid glands and their possible removal. It is important to note that the cardinal diagnostic features of primary hyperparathyroidism are elevated or inappropriately elevated parathyroid hormone (PTH) levels in the setting of hypercalcemia. Pure PTH levels should be low with the hypercalcemia of malignancy syndrome.

Treatment: The four management objectives are: 1) correct dehydration, 2) promote calcium excretion by the kidneys, 3) inhibit accelerated bone resorption, and 4) treat the underlying disorder. Even when the cause of hypercalcemia turns out to be a surgical disorder, the following medical guidelines should be done in order to normalize the serum calcium level as quickly as possible.

A) Saline infusion: Since hypercalcemia is a medical emergency, treatment must not be delayed while the etiology is being determined. The most important therapeutic measure is to rehydrate the patient. Since urinary calcium excretion is enhanced by saline infusion and since 0.9 percent sodium competitively inhibits renal tubular reabsorption of calcium, sodium chloride solution administered IV is the fluid of choice. The infusion should be rapid in order to produce an intense diuresis, but care must be taken to avoid plasma volume overload. Serum electrolytes must be monitored and supplemented (especially K^+) accordingly.

B) Glucocorticoids are particularly beneficial when treating hypercalcemia associated with lymphoma or other malignant tumors associated with hypercalcemia. When lymphoma is suspected it is best to obtain a tissue diagnosis prior to steroid administration. By counteracting the effect of vitamin D, glucocorticoids are efficacious in treating hypercalcemia caused by hypervitaminosis D. They might be particularly useful for the treatment of cholecalciferol rodenticide intoxication and dietary oversupplementation.

C) Other therapeutic measures for hypercalcemia can include the administration of IV and oral phosphate solutions, Mithramycin (Plicamycin), calcitonin, and bisphosphonates (etidronate disodium). The use of these agents must be done under close medical observation.

D) Surgical excision of a parathyroid tumor or hyperplastic parathyroid glands is the preferred treatment for primary hyperparathyroidism. Since the uninvolved parathyroid glands will be atrophied due to negative feedback effects from hypercalcemia, postoperative hypocalcemia should be anticipated as a potential life-threatening complication. This can occur several hours following the parathyroid tumor removal and should be counteracted by slowly infusing ten percent calcium gluconate at a dosage of two ml/kg IV every six hours immediately after surgery. This infusion should be appropriately titrated during the subsequent postoperative days. Dihydrotachysterol treatment is commonly needed for several days to weeks postoperatively in order to maintain normocalcemia. The dosage is titrated to effect pending the results of periodic serum calcium level determinations.

Ten Building Blocks of an Exceptional Breeding Program
I. Deborah A. Lynch
Executive Vice President
AKC Canine Health Foundation

1.   Become a Student of Your Chosen Breed
        A. Study the breed standard
        B. Observe judging at dog shows
        C. Practice positive appreciation
        D. Interview outstanding breeders
        E. Attend your national specialty and regional specialties
        F. Remember, for the first five years you are considered a novice

2.   Study Canine Genetics and Physiology
        A. Attend workshops and seminars
        B. Read books about dog breeding and canine genetics
        C. Keep informed of new advances and tests currently available
        D. Study structure and movement in your breed and all breeds
        E. Study dogs and pedigrees in your breed – keep notes

3.   Develop a Plan and Target Your Goals
        A. <u>Breeding Goals</u>
        A. Healthy dogs
        B. Advance breed performance and temperament
        C. Advance the breed standard
        D. Good permanent homes for all dogs produced

        B. <u>Performance Goals</u>
        A. What are your goals in conformation
        B. What are your goals in performance

4.   Develop Annual Objectives and Milestones
        C. <u>Sample Objectives</u>
        A. Finish a champion
        B. Put dual titles on your first dog
        C. Potential breeders should finish one champion and obtain at least one obedience
            title, and if applicable a performance title

                a) <u>Sample Milestones</u>
        A. First homebred champion
        B. First Specialty and Breed Wins
        C. Finish with all Specialty Wins

5.   Identify a Mentor

A. Observe breeders and exhibitors in your selected breed
B. Identify those that are successful and have extensive experience
C. Avoid becoming part of an entourage
D. Seek out breeders who are objective, positive, encouraging and experienced

6.   Select Your Foundation Dogs
   A. Start with a quality bitch
   B. Avoid buying a young puppy
   C. Purchase a dog at least six months old with a show record
   D. Pay attention to the dam's record - outstanding producer, natural whelper, good litter size, excellent health and temperament, predictable pedigree of producing and performing dogs
   E. Pay attention to the sire's record - quality producer, predictable pedigree of producing specific qualities, excellent health record
   F. Parents clear on appropriate tests

7.   Screen All Dogs For Appropriate Health Tests in Your Breed and
   Plan With Your Mentor
   A. Evaluate your brood bitch
   B. Identify strengths and weaknesses in phenotype and genotype
   C. Make a list of potential stud dogs, strengths and weaknesses of the dog and its get
   D. Learn to analyze a pedigree in your breed
   E. Review the pedigree and get with your mentor
   F. Make the decision

8.   Practice Critical Evaluation and Develop A Network of Friends
   A. Continually evaluate your breeding program
   B. Practice good sportsmanship
   C. Know the strengths of your competition
   D. Breed to the competition when it makes sense
   E. Develop a group of breeders in your regional area that have similar goals

9.   Always Put Your Dogs First
   A. Keep your dogs to a reasonable number
   B. Know that your home, which is now a show and breeding kennel, is not always the best home for every dog you produce
   C. Make each decision based on what is right for the individual dog
   D. Practice good animal husbandry

10.   This Is Your Hobby, Enjoy It!
   A. There will be ups and down
   B. Focus on fun and friendship
   C. The happiness of you and your dogs is number one!
   D. Always smile when you leave the ring!
   E. There may be a Best in Show next litter!
   F. THE DOGS WILL LOVE YOU NO MATTER WHAT!

II. Ear Care and Disease
III. Rosanna Marsella, DVM

Introduction
Otitis externa is one of the most frequent reasons for owners to seek veterinarians' help. The prevalence of otitis externa in dogs has been reported to be between 10-20 percent, although in more tropical climates it is probably closer to 30-40 percent. Unfortunately, the term otitis does not refer to a specific disease but to an inflammation of the external ear canal. It is a symptom of many diseases and not a specific diagnosis. The actual underlying causes of otitis are numerous. The purpose of this lecture is to review the general principles of ear care and the most important causes of otitis externa.

Physiology of the Ear Canal
The ear canal in the dog and cat can be divided into a vertical segment (which is continuous with the pinnae) and a horizontal segment that abuts the tympanic membrane. The canal is almost entirely surrounded by cartilage that offers stability to the structure. Besides the obvious auditory function of the external meatus, the canal also offers protection of the tympanic membrane and the middle ear from direct injury.

Preventative Ear Care and Ear Cleaning
Preventative ear care begins with a complete history and thorough physical examination. Historical information and physical findings are necessary to identify patients at risk. Specific information about previous and concurrent medical disorders is essential, because ear disease may co-exist with other disease or be secondary to systemic diseases.

Routine cleaning of the ear canal is not necessary and may be contraindicated in the healthy dog and cat. Most dogs do not require cleaning of the ears. Mild to moderate amount of wax is normal. Cerumen has antibacterial properties that help to reduce the over population of bacteria and yeast. Cleaning, when necessary, should be complete and non-irritating. A mixture of vinegar/water (1/10) is a good degreasing solution to remove wax and dry the excessive moisture in the ear canal. The liquid should be gently applied in the canal, the ear massaged to allow breakage of the cerumen and cotton balls used to remove the cerumen and wipe out the excess of liquid. Extreme care should be used when mechanically cleaning the ears. The use of cotton applicators should be avoided or limited as they may cause rupture of the tympanic membrane. Also powders should not be applied in the canal as they build up predisposing to the development of secondary infections.

Accumulations of cellular debris and exudates indicate the presence of ear disease. Swabs of this material should be collected and the canal should be cleaned. The color, texture and odor of the exudates from a diseased ear can provide clues regarding the underlying primary cause of the otitis and the perpetuating factors that may be involved. Dark brown or black, granular, dry (like coffee grounds) exudates characterizes infestations due to ear mites. A moist brown discharge tends to be associated with bacteria (cocci) and yeast infections. Purulent creamy to yellow exudates are most often seen with bacteria such as *Pseudomonas*. Waxy, greasy, yellow to tan debris is typical of a ceruminous otitis.

Thorough cleaning of the ear canals is vitally important for successful management of otitis for several reasons. Examination of the external ear canal and the tympanum cannot be complete until the canal is cleaned. Wax, oil and cellular debris may be irritating, prevent medication from contacting the canal epithelium, and produce a favorable environment for microorganisms to proliferate and inactivate certain antibiotics. Several products are available on the market and they should be used as directed by a veterinarian as some of them may interfere with the efficacy of the topical medications. Also some of them may be irritating if not completely removed thus appropriate flushing by a veterinarian might be required. These products are usually classified as either ceruminolytic or drying agents.

1. Ceruminolytic agents (e.g. Cerumene) emulsify the waxes and lipids to help flush them more readily from the ear canal. They contain surfactants and detergents (e.g. diotyl sodium sulfosuccinate or DSS, squalene, carbamide). In general such products should be applied 5-15 minutes prior to cleaning. General massage improves their effect. Most of these products are contraindicated with ruptured tympanum. However, frequently the condition of the tympanum cannot be determined until after the canal has been cleaned. In those cases the probability of ototoxicity may be decreased by flushing with water after the application of such agents. In a recent study several cerumuminolytic agents were applied in the middle ear and squalene was the only one that did not cause any damage.[1] However, it should be realized that there is no completely safe solution for cleaning the middle ear. Even water can cause ototoxicity. Some disinfectant cleansers, such as chlorhexidine, are contraindicated with ruptured tympanums.
2. Drying agents (e.g. Epi-Otic) are applied after the ear has been cleaned and is relatively dry. Most contain alcohol and one or more of the following: boric acid, benzoic acid and acetic acid. Some products are a combination and they tend to have less drying agents and mildly ceruminolytic than the standard desiccants (e.g. Epiotic, Oticlens).

When flushing an ear with a ruptured tympanum the use of saline of 1:1 or 1:3 dilutions of five percent acetic acid (white vinegar) are recommended. The fluid is discarded with every flush and suck cycle and the canal is filled again with clean saline. This is repeated multiple times using a fair amount of saline. The best results for deep ear cleaning or flushing are obtained with the patient under general anesthesia.

Cleaning cannot be done on very swollen, stenotic, ulcerated or painful ears. Such cases need to be treated symptomatically initially and cleaned at a later date when the inflammation has been reduced and the canals have opened.

Causes of Otitis Externa

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[1] Mansfield PD, et al. The effects of four commercial ceruminolytic agents on the middle ear. *JAAHA* 97; 33: 479-86.

Otitis may have numerous causes and a common classification is to break them down into predisposing, primary and perpetuating.[1] Predisposing factors are those that place a patient at risk but by themselves are not able to cause otitis externa. Primary causes are usually the actual inciting agent that directly causes otitis externa. Perpetuating factors are those that prevent the resolution of otitis externa once the problem has been established.

Predisposition Factors and Risk Assessment

The most successful management requires that these factors are recognized and, whenever possible, controlled. Early detection may prevent unnecessary pain/hearing loss and reduce the prevalence of chronic and refractory disorders.

1. Breed Predisposition and Anatomic Conformation

Otitis occurs more frequently in breeds of dogs that have pendulous ears (e.g. Cocker Spaniel) and those with hair growth in the ear canal. Originally this difference was thought to be secondary to variations in the temperature and/or humidity of the ear's microenvironment in dogs with different ear types, however no difference in temperature was found between ear types. It is becoming more and more evident that variations in the anatomy and the number of glands may predispose certain breeds to otitis externa. Dogs with longhair coats and pendulous ears should have the hair clipped frequently around the auricular orifice and the concave surface of the pinnae. Hair in the ear canal should be removed with a forceps and twisting (rather than plucking, which is more painful) the hairs out by twirling the forceps to improve ventilation in the canal. Great care should be used when removing these hairs as excessive trauma to the area may predispose to an infection. Stenosis of the ear canal (e.g. Shar-Pei) is another variation in the anatomy that can predispose dogs to otitis externa. In Shar-Peis the stenotic canal and the conformation of the pinna that is tightly folded over the external orifice increases the risk of otitis externa. Stenosis of the canal can also be acquired (e.g. abscess, neoplasm).

2. Climatic variations

In a recent study monthly variation in ambient temperature, rainfall, and relative humidity correlate positively with increases in the number of first-time otitis externa cases seen.

3. Life Style

Dogs used for activities that involve exposure to field are at increased risk of ear disease. Foreign bodies, especially plant material, often become trapped in the canal. These animals should be examined frequently.

4. Maceration of the Ear Canal

Any increase in the moisture of the ear canal can lead to maceration. Moisture in the canal, whether introduced by swimming, bathing, or inappropriate treatment may cause otitis externa of

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[1] Griffin CE, Kwochka KW, MacDonald JM. *Current Veterinary Dermatology: the art and science of therapy.* Mosby Year Book, St. Louis, 1993.

inflammation of the external part of the ear canal. A combination of water retention, epidermal maceration, increased ceruminous gland activity and secondary infections may be responsible for disease. Dogs that swim may benefit from prophylactic treatment with a drying agent (e.g. acetic acid).

5. Excessive Ear Cleaning

Mechanical trauma of the ear canal through vigorous hair plucking and the use of cotton swabs or other objects to remove wax, as well as the use of irritant topical solutions and excessive cleaning that alters the normal micro-flora, are all factors that predispose to the development of infections.

Primary Causes of Otitis Externa

When discussing the primary causes of otitis externa, it is important to remember that the epithelium of the external ear canal is simply an extension of the rest of the skin. Most causes of otitis externa are associated with generalized dermatologic conditions. A complete dermatologic history and work up may therefore be necessary in the diagnosis of many primary otitis externa cases. The most common causes seen in dermatology are atopy (inhalant allergies), food allergy, diseases of keratinization (e.g. primary seborrhea of Cocker Spaniels), and ear mites. It is critical to long-term management of otitis externa that a primary cause can be found.

1. Parasites

The ear mite (e.g. *Otodectes*) is the most common mite, being responsible for up to 50 percent of the cases of otitis externa in cats; in dogs the incidence is controversial but most authors agree that it is responsible for 5-10 percent of cases. They are most commonly found in the external ear canal, but can survive for some time on the surface of the skin, typically of the head and neck. In the ear the mites are protected by desiccation by a typical dark brown crust. In recurrent cases, it is possible that other in contact animals can act as asymptomatic carriers. Other mites that can be responsible for otitis include *Sarcoptes* (mite that causes scabies), *Demodex* (mite that causes the so-called red mange), *Eutrombicula* (chiggers) and *Otobius* (the spinous ear tick of dogs).

2. Micro-organisms

Dermatophytes (which cause "ringworm") are a relatively common cause of disease of the pinna and in rare occasions may cause otitis externa. Bacteria are most commonly perpetuating factors.

3. Allergies

Allergies are the most common underlying cause for otitis externa in dogs. They include inhalant allergy (also called atopy), food allergy and contact allergy.

Inhalant allergy is extremely common in dogs and cats and is the most common underlying cause for recurrent otitis externa in dogs. At least 50 percent of atopic dogs have bilateral otitis externa. In up to five percent of cases, otitis may be the only complaint. Atopic dogs tend to have itchy feet (e.g. they lick and chew their feet), itchy face (e.g. they rub their face against the carpet or pieces of furniture) and itchy ears. They are predisposed to secondary skin and ear infections that tend to recur after treatment unless the underlying allergy is well controlled. A familial history is present in most cases and strong breed predilection has been reported (e.g. Dalmatians, Terriers, Golden Retrievers). Clinical signs are initially seasonal. Progressive worsening with time is also typical. Diagnosis is based on history, clinical signs, exclusion of other diseases and intradermal skin test.

Food allergy is not as common as the inhalant allergy, but over 20 percent of these cases start with just otitis externa, and ear disease is present in 80 percent of the cases. Food allergy should be considered as a top differential for otitis externa in any young dog (less than one year of age). Food allergy is diagnosed by appropriate food trial (a novel source of protein is selected based on the individual history and used for a minimum of two months).

Contact allergy can result from medications used to treat otitis externa. Whenever a case of otitis externa fails to improve with therapy or worsens after therapy, a contact dermatitis should be suspected.

4. Foreign bodies

Plant material (fox tails), dirt, sand, impacted wax, loose hair and dried medications are frequently responsible for otitis externa. In most cases this is a unilateral otitis.

5. Diseases of keratinization (e.g. primary seborrhea of Cocker Spaniels)

Excessive and abnormal composition of cerumen in these cases is responsible for the development of otitis externa and secondary infections of skin and ears. It is usually seen in young animals.

6. Endocrine disorders

Hypothyroidism (decreased production of thyroid hormone) and Cushing's disease (disease associated with excessive production of steroid hormones) are the most common endocrine diseases that can cause otitis externa. If a middle-aged dog keeps relapsing with otitis externa and is not itchy, then endocrine diseases should be considered as possible underlying causes.

7. Autoimmune disorders

Pemphigus (disease in which the organism produces antibodies against component of its own skin) affects the pinna and may extend to the ear canal causing otitis. Lupus (other autoimmune disease in which the organism produces antibodies against various components of the body) can also cause ear disease.

Perpetuating Factors

They include anything that prevents the resolution of an already present otitis externa. Perpetuating factors are a major reason for poor response to therapy regardless of the predisposing factors and the primary cause. In early cases treating the primary cause might be sufficient to resolve the otitis, while in more chronic cases perpetuating factors have to be addressed to resolve the case.

1. Bacteria

In most normal ear canals a variety of bacteria can be cultured. Once predisposing and primary factors cause alterations in the ear canal environment, these bacteria may proliferate and perpetuate an inflammatory reaction. In most cases of chronic otitis externa bacteria such as *Staphylococcus* and *Pseudomonas* are present. Aggressive treatment is warranted as resistance to antibiotic may easily occur, especially in cases when *Pseudomonas* is cultured. Although bacteria are not a primary cause of otitis, once the infection is established, they can cause significant inflammation and damage. These dogs often present with purulent discharge in the ears. Pain on palpation of the ears is quite common and a strong odor is usually present. Diagnosis is based on cytology and culture. Initial topical therapy for a case of otitis externa is based on the results obtained from the cytology of the exudates, while in chronic cases is best based on results of culture and sensitivity.

Pseudomonas-related infections are extremely frustrating and difficult to treat. Most effective treatments include topical Polimixin B and systemic enrofloxacin or ciprofloxacin. Doses that are used are higher than the ones suggested on the label as resistance occurs rapidly. A commonly used dose for these drugs is eleven mg/kg twice daily. Dogs with OM frequently require two months of systemic antibiotic. As Polimixin B is rapidly inactivated by the exudates, aggressive cleaning is an essential part of therapy. Other topical treatments used for *Pseudomonas* include acetic acid (vinegar/water 1:1) and silver sulfadiazine (one gram of silver sulfadiazine is mixed with 100 ml of sterile water and 0.5 ml of the mixture is applied twice daily). Also pre-soaking the ear with edetate trisodium (tris-EDTA) 15 minutes prior to application of the antibiotic increases the efficacy. Finally, topical enrofloxcin (Baytril otic) can be used in *Pseudomonas* infections.

In cases where *Staphylococcus* is the cause of otitis, other antibiotics are usually used including cephalexin (22 mg/kg twice daily) and trimethoprim-sulfa (25 mg/mk twice daily).

2. Yeast

Malassezia is the most common perpetuating yeast that contributes to otitis externa. It is a budding organism with the shape of a peanut and is part of the normal flora (both skin and ears) of dogs and cats. It is a common complication with allergic otitis (80 percent of cases) and may result as a super-infection following antibiotic therapy. Grossly the discharge is thick, dark, and sweet smelling. Diagnosis is usually based on the physical findings and microscopic examination of the exudates (cytology). Topical therapy is usually sufficient and miconazole and clortrimazole (e.g. Conofite, Lotrimin) are the most commonly used ingredients. In rare

cases of otitis media due to *Malassezia*, systemic treatment is necessary and ketoconazole (Nizoral tablets) is used at five mg/kg twice daily for three to four weeks. Side effects include anorexia, vomiting and diarrhea.

3. <u>Progressive pathological changes</u>

Chronic inflammation stimulates the proliferation of the skin lining the ear canal. As a consequence, thickening of the canal occurs leading to stenosis of the canal. More importantly the skin is thrown into numerous folds, which inhibits effective cleaning and the application of medications. These folds act as a site for the perpetuation and protection of secondary microorganisms (e.g. bacteria). Laser surgery has been used successfully to correct excessive stenosis and thickening of the canal.

4. <u>Otitis media</u>

Otitis media (inflammation of the middle ear) results from chronic inflammation of the external part of the ear canal, rupture of the tympanic membrane, and establishment of infection in the middle part of the ear. Exudate in the tympanic cavity is difficult to treat with topical therapy and often remains as a source for infection. Otitis media is usually bacterial in origin. Clinical signs suggestive of otitis externa include head shyness and pain on palpation of the ears. Some cases of otitis media might present with head tilt, circling and dry eyes, but the vast majority does not have neurological abnormalities. As the tympanic membrane quickly grows back after rupture, otitis media may also be present even if an intact membrane is seen on otoscopic examination. Radiography cannot be used to completely rule out the presence of otitis media since 25 percent of confirmed cases had no radiographic evidence of the disease. In a study otitis media was present in 80 percent of cases of chronic, relapsing otitis externa therefore it must be considered as a possible cause of any refractory or relapsing otitis externa. Treatment of otitis media is based on bacterial culture/sensitivity results. Most cases require long-term antibiotic therapy (minimum of two months) and aggressive topical therapy.

Conclusions

Otitis externa is a very common clinical presentation in small animals.

A good history is very important in every case of otitis externa to identify predisposing and primary factors.

Aggressive treatment is needed in most cases of relapsing otitis externa as an infection of the middle ear might be present.

Canine Physical Therapy
Jason Wheeler, DVM
IV. University of Florida

Physical therapy or rehabilitation is a relatively new field in veterinary medicine. In human patients, physical therapy is a common and well-accepted form of treatment for conditions such as postoperative rehabilitation, osteoarthritis, and neuromuscular disorders. Until recently, there has been limited study of physical therapy in animals. Knowledge of physical therapy modalities in human patients has enabled veterinarians to adopt some of the techniques and procedures to small animals.

Rehabilitation of patients with acute and chronic musculoskeletal injuries involves the application of controlled challenges to tissues to improve strength, condition and function. The tissues most affected by injury are cartilage, muscle, ligaments, tendons and bone. Ultimately the goal of physical rehabilitation is to facilitate and maximize recovery and functional mobility following a neuromuscular insult. This can be accomplished in a variety of ways: 1) reduce pain and accelerate healing of injured and inflamed tissues; 2) maintain and restore normal range of motion in affected joints; 3) prevent fibrosis or soft tissue contractures in injured, weak and paralyzed limbs; 4) prevent disuse atrophy during the healing phase of neurologic or musculoskeletal insult; 5 gain strength and improve function in weak or paralyzed muscles; 6) provide a positive psychological effect maximizing both patient and owner well-being; and 7) provide the owner with individualized home care programs to maximize the animal's functional mobility and prevent injury to the owner and possibly further injury to the animal.

In order to detect functional limitations and primary impairments, an initial patient assessment is crucial prior to administering physical therapy. A thorough evaluation is required to document current baseline status as well as to document the efficacy of therapeutic interventions and protocols. Assessment tools may include a goniometer to measure range of motion, a girhtometer to measure limb girth and effusion measurements, and a method of measuring weight bearing status (force plate) and observational gait analysis. Factors affecting the type of physical therapy include the age and physical condition of the patient, the nature of the surgical condition, concurrent injuries, owner compliance, and the expertise of the person performing the physical therapy. Whenever possible, preemptive analgesia should be instituted to allow physical therapy to be performed to a patient that is as comfortable and pain free as possible.

Massage

Massage is the manipulation of superficial and deep soft tissues. Different types of massage include gentle gliding over the skin without moving the underlying tissues to vigorous cross fiber friction over muscles or ligaments to break up adhesions. Strokes may be linear, circular, or applying direct compression, while directing strokes toward the heart to help promote venous return. The beneficial effects of massage include relaxation of soft tissues, decreased muscle spasm and trigger points, increase muscle flexibility, improved venous and lymphatic flow with reduction of edema, and increased local blood flow. Massage is helpful in increasing blood flow to muscles to help "warm up" an area prior to activity as well as decrease stiffness after activity.

Passive Range of Motion

Passive range of motion is an important post-injury modality to reduce tissue adhesions, promote normal joint dynamics, enhance venous and lymphatic drainage, and prevent muscle and joint capsular contractures. This type of therapy should ideally begin the day of surgery and continue for two to three weeks. However, in the acute injury phase, full pain free use of joints may be limited. It is very important to reestablish full range of motion (ROM) as soon as possible. Permanent loss of motion can occur in as little as two weeks following some surgical procedures, which ultimately limits an animal's functional ability in the future.

Proper technique is very important when performing ROM. Over-aggressive ROM exercises will result in pain, reflex inhibition, delayed use of the limb and increased joint fibrosis. The goal is to stretch and realign soft tissues, not to tear tissues. Begin by flexing the joint until the first signs of discomfort, and hold for ten seconds while continuing to stretch. Repeat this exercise with extension of the joint. Repeat for ten to 30 repetitions of flexion and extension, three to six times daily. These exercises should be performed on all joints in the affected limb. ROM therapy is especially important for any joint surgery and in young dogs.

Cryotherapy

Cryotherapy (ice packing) is important following acute injuries or in the immediate postoperative period. Cryotherapy decreases blood flow, and reduces pain, swelling, inflammation, hemorrhage, and metabolic activity. Cryotherapy is most effective if applied for the first 72 to 96 hours, for 15 to 20 minutes, every four to six hours following injury or surgery. It is also beneficial for up to two weeks following physical therapy sessions. Although cryotherapy is beneficial for acute injuries, it is also sometimes helpful for chronic conditions because it reduces pain and edema, and it may reduce collagen destruction, and synovial or joint inflammation.

Cryotherapy can be performed with commercially available ice packs or by filling a sealable waterproof bag with crushed ice and wrapping it in a towel.

Heat

After 72 to 96 hours, heat application may be beneficial, especially prior to initiating therapy sessions. Heat increases blood flow, tissue extensibility of joint capsules, tendons, and scar tissue, and promotes general relaxation. Superficial heating agents generally heat the skin and subcutaneous tissues to a depth of one to two centimeters. This can be done with commercially available hot packs or warm moistened towels. Circulating warm water blankets may be used for large areas. Electric heating pads should not be used. Be careful applying heat to areas with reduced sensation or in animals that cannot move away from a painful stimulus. Heat is generally contraindicated if swelling or edema is present; cold therapy is typically used in these instances.

Therapeutic Ultrasound

When deeper tissue heating than can be accomplished with warm packs is warranted, therapeutic ultrasound is indicated. Ultrasound penetrates tissues up to five centimeters and heats tissues to 40-45ºC. Ultrasound offers all the beneficial effects of traditional heat therapy to deeper tissues as well as many non-thermal effects. These include increased cell diffusion and membrane permeability, increased calcium ion transport, facilitation of the inflammatory process, and increased production of fibroblasts, glucosaminoglycans and hydroxyproline. Therapeutic ultrasound typically uses a 1 or 3.3 MHz transducer. 3.3 MHz transducers are effective for heating superficial tissues and 1 MHz transducers penetrate deeper tissues. Variation in pulsed or continuous ultrasound can also vary the therapeutic outcome. Pulsed ultrasound produces primarily non-thermal effects, as the average energy is much lower. In continuous mode, the thermal effects are much greater.

Rehabilitative Exercises

As edema and inflammation begin to subside, the animal is more tolerant of manipulation to the surgical site. The pain begins to resolve, and additional activities may be added to the rehabilitation protocol. These activities include exercises, such as standing (neurologic patients or patients with pelvic trauma), walking with sling assistance, walks at slow speeds to encourage weight bearing, walking up or down inclines, climbing stairs, treadmill, wheel barrowing (encourage use of forelimbs), and dancing (encourage hind limb use). Therapeutic exercises are limited only by one's imagination. These exercises help to facilitate the speed of recovery, maintain pain-free range of motion and quality of movement, enhance performance and endurance, increase muscle mass and strength, improve daily function and prevent further injury.

Aquatic Therapy

Following removal of any sutures and complete sealing of the incision, aquatic therapy can be added. For small dogs a bath tub may be sufficient. If a whirlpool is available, it may also be used for medium to large breeds. Alternatively a swimming pool, lake or pond (with clean water and no alligators) may be used to allow animals to swim. Some dogs do not tolerate swimming and there is some risk to the handler and to the animal by damaging tissues if the animal thrashes around too much. Animals undergoing aquatic therapy should also be assisted into and out of the water and should not be allowed to freely run and jump into the water. However, swimming is an excellent means of improving muscle strength and joint mobility in a non-weight-bearing environment. Underwater treadmills have recently been developed for dogs which allows active use of muscles, appropriate gait patterning with limited weight-bearing, and enhanced cardiovascular fitness, while taking advantage of the buoyant effects of aquatic therapy to reduce weight-bearing stress on bones and joints. It should be noted that many patients are not well conditioned and they may only be able to tolerate two to five minutes of swimming when starting an aquatic program.

Summary

Optimal protocols for physical therapy and postoperative rehabilitation in animals are unknown. This is an exciting new area of patient management and has the potential to improve outcomes, increase patient quality of life, and enhance owner satisfaction and enjoyment of their companions.

The Current Status of Canine Vaccinations:
Are We Vaccinating Dogs with Too Many Vaccines Too Often?
Cynda Crawford, DVM, PhD
University of Florida

Introduction

Since the 1950's, veterinary medicine has done an excellent job in educating pet owners on the importance of vaccination. There is no debate over the health benefits that millions of dogs have derived from vaccination against distemper, parvovirus, infectious hepatitis, and rabies. The decrease in number of human, canine, and farm animal cases of rabies in recent years, despite an increasing incidence in wildlife, is largely attributable to vaccination of dogs.[1] Vaccination not only protects the individual dog, but also decreases disease prevalence and transmission in the canine population as a whole, even if some of the dogs are not vaccinated. For example, rabies is not effectively transmitted if more than 70 percent of the canine population is vaccinated.[1] Outbreaks of disease occur when the proportion of immune individuals decreases below a threshold, either because of decreasing immunity in vaccinated dogs or decreased number of dogs vaccinated. Thus, the population's immunity to an infectious agent needs to be maintained by vigilant vaccination of individuals at risk for exposure and transmission of disease.

More than 20 years ago, the American Veterinary Medical Association (AVMA) recommended annual revaccination of all dogs, a practice still common today. This recommendation was based on the assumption that immunity would dwindle in some dogs, so that frequent revaccination of all dogs was required to insure immunity in the population. Recommendation for annual revaccination of all dogs with multivalent or "combo" vaccines assumes that every dog is at significant risk for exposure to every infectious agent in the vaccine, and that each agent in the vaccine will stimulate the same degree of immunity that lasts the same amount of time.[1] Today, we know that these assumptions are neither rational nor scientifically justified, but are convenient for the veterinarian and pet owner alike.

Vaccination should not be a regimented, one-fits-all procedure.[1] The objective of vaccination is simply to give the right vaccine at the right time to the right individual to protect that individual from an infectious disease. To accomplish this objective, each dog should be evaluated with regard to age, lifestyle, disease prevalence in the community, potential for exposure to infected dogs and environments, and the severity of clinical disease, if any, after infection.[2] The benefit of vaccination is questionable if the potential for exposure is limited by lifestyle, the prevalence of disease in the community is low, and the clinical disease is either unapparent or mild. Vaccination is a medical procedure, and as such, requires individual assessment of each patient.

Fortunately, the most common effect of vaccination is stimulation of a protective immune response. Despite the intended benefits, vaccination does carry with it attendant risks. Now, a growing awareness of vaccine-related health problems is motivating veterinarians and pet owners alike to question the benefit of annual revaccination or "boosters" for adult dogs. The concern, controversy, and confusion centers around the question **"Are we vaccinating dogs with too many vaccines too often?"** [2]

Vaccine Types

There are three types of vaccines: killed vaccines, modified live vaccines, and recombinant vaccines.[1,3] Vaccines that contain killed viruses or bacteria also contain an adjuvant, usually aluminum hydroxide, to nonspecifically boost the immune response to the vaccine. The advantages of killed vaccines include lack of replication in the host, no chance of reversion of the infectious agent to virulence, and safer for use in dogs that are immunosuppressed or in breeding bitches housed in contact with neonates. The disadvantages include a higher incidence of adverse reactions to the killed organisms suspended in adjuvant, formation of lower amounts of antibody that do not remain at protective levels for very long, and requirement for frequent vaccinations to boost the immunity to protective levels. In addition, killed vaccines must be administered by intramuscular or subcutaneous injection. Examples of killed vaccines commonly used in dogs include those for coronavirus, leptospirosis, kennel cough, Lyme disease, giardia, and rabies.

Modified live vaccines (MLV) contain live viruses or bacteria whose virulence has been modified to allow replication in the host without causing disease. These vaccines simulate natural immune responses in that they stimulate rapid humoral and cell-mediated immunity that is sustained at high levels for long periods of time. Some of these vaccines can be given at the site where the pathogen normally invades the body, such as the nostrils, thus inducing a localized immune response that acts quickly to prevent invasion. Examples of commonly used modified live vaccines in dogs include those for distemper, parvovirus, canine infectious hepatitis, parainfluenza, and kennel cough. Both killed and modified live vaccines can be monovalent or multivalent. Monovalent vaccines contain only one infectious agent, whereas multivalent vaccines contain two or more.

Recombinant vaccines are genetically engineered vaccines created by inserting selected genes from an infectious agent into a nonpathogenic carrier agent that serves as a production factory. The proteins made from the selected genes are those that are critical in stimulating protective immunity to the infectious agent, and thus focus the immune response. The proteins can be harvested after production by the carrier agent and used as a purified protein vaccine, with or without adjuvant. An example of this type of recombinant vaccine is the new one for Lyme disease. Alternatively, a nonpathogenic virus into which the selected genes of the pathogen have been inserted can serve as the actual vaccine. Replication of the nonpathogenic carrier virus in the dog produces the pathogen proteins that induce a protective immune response. An example of this is the new recombinant vaccine for canine distemper.

Duration of Vaccine-Induced Immunity

Vaccine manufacturers in the United States are required by the USDA to conduct efficacy and safety studies for their vaccines prior to licensure.[1] The efficacy studies are challenge studies, the "gold standard" for demonstrating protective immunity provided by a vaccine. In challenge studies, non-vaccinated and vaccinated dogs are exposed to the virulent organism at various times after vaccination to determine the extent and duration of protection against disease. The manufacturer is not required to establish the full duration of immunity for the vaccine, but only to provide documentation of what they claim on the label, hence the recommendation for annual

revaccination.[1] However, minimum duration of immunity challenge studies are required for all rabies vaccines, and for all new vaccines using antigens that were not in use prior to 1995.[1] Vaccines produced by different companies may induce different durations of immunity against the same diseases. Trials to determine the duration of immunity beyond that of one year that would meet USDA guidelines can be cost-prohibitive. However, recent challenge studies performed by independent research groups have demonstrated that the **minimum** duration of immunity induced by modified live virus vaccines for distemper, parvo, canine infectious hepatitis, and parainfluenza is actually five to seven years, not one year![1] Furthermore, annual revaccination in these challenge studies did not provide any additional benefit in terms of the strength of the humoral immune response elicited, improved resistance to disease, or extension of the duration of immunity.

Thus, based on duration of immunity, annual revaccination of adult dogs is probably not necessary for protection against distemper, parvovirus, canine infectious hepatitis, and parainfluenza.[1, 2]

Who Should be Vaccinated with What and When?

Puppies less than six months old are more susceptible to the common infectious diseases than adults, and therefore are the primary target population for vaccination.[1] In the US, the incidence of clinical disease from distemper, infections hepatitis, and parvovirus in dogs older than one year of age is virtually zero.[1]

In 2001, the American Veterinary Medical Association Council on Biologic and Therapeutic Agents (COBTA) presented guidelines for vaccination of dogs.[2] The guidelines emphasized that there are inadequate data at this time to support a single best vaccination protocol, so veterinarians should perform a risk to benefit analysis for the use of each vaccine in each patient. The guidelines divide currently available vaccines into "core" and "non-core." Core vaccines are selected based on the prevalence of the infectious agent in the environment, the severity of the clinical disease that results from infection, the ease with which the infectious agent is transmitted between animals, and the zoonotic potential. Core vaccines include canine distemper, parvovirus, infectious hepatitis virus, and rabies. Non-core vaccines are those that are useful in situations where the risk of exposure is high and disease can be debilitating. These include vaccines for leptospirosis, parainfluenza, coronavirus, kennel cough, Lyme disease and giardia.

High levels of maternal antibodies acquired from ingestion of colostrums protect puppies from disease for the first six to eight weeks of life. After six to eight weeks of age, a window of susceptibility to infection is created because maternal antibodies are high enough to interfere with the vaccine-induced response, but not high enough to protect the pup from infection and disease.[1] This maternal antibody blockade is the most common cause of vaccine failure in puppies. Therefore, immunizations are repeated at timed intervals during the first four to six months of the puppy's life to insure development of a protective immune response. The pediatric series include the core vaccines for distemper, parvovirus, and infectious canine hepatitis starting with an initial immunization at six to eight weeks of age, followed by boosters every three to four weeks until 16 weeks old. Certain breeds have a higher frequency of individuals that do not develop vaccine-induced antibody titers during the routine pediatric

series. These breeds include the Rottweiler, Doberman Pinscher, Labrador Retriever, Alaskan sled dog, Pomeranian, and American Staffordshire Terrier.[4] For puppies of these breeds, boosters are recommended every three to four weeks until 24 weeks of age.

The most popular core vaccines are modified live vaccines that contain a combination of **distemper** virus, canine adenovirus type 2 for protection against **infectious hepatitis, parvovirus** and **parainfluenza** virus (DHPP or DA$_2$PP). Contrary to popular belief, combining these viruses into multivalent vaccines does not alter the immune response to each – no scientific study has shown that one virus suppresses the immune response to the other viruses given at the same time, so it is not necessary to alternate vaccination with each separately.[1] Several potentiated monovalent vaccines are available for parvovirus. Potentiated vaccines contain very high titers of parvovirus, and are most effective in overcoming the maternal antibody blockage in young puppies. The original parvovirus, CPV2, has been replaced over the years by two antigenic variants or biotypes called CPV2a and CPV2b. The CPV2b biotype is more prevalent in the US, while the CPV2a is more prevalent in Europe. Most licensed parvovirus vaccines still contain the original CPV2 virus, but the induced immunity is cross protective against both biotypes.[1, 5] A recently marketed recombinant vaccine for distemper (Recombitek CDV, Merial) uses the canarypox virus as a vector for a distemper virus gene that codes for a protein associated with protective immune responses.[3] The vaccine is effective but the minimum duration of immunity beyond one year has not been established.

The last core vaccine administered during the pediatric series is **rabies**. The rabies vaccine is the only one that is legally required due to the health threat to humans. Most states require an initial vaccination at 12-16 weeks of age, followed by a booster one year later. State laws vary on revaccination intervals for adult dogs, with intervals ranging from one to three years. Rabies vaccines contain large amounts of killed virus suspended in adjuvant, and have a minimum duration of immunity of one year or three years.

Based on the long duration of immunity for the core vaccines against distemper, canine infectious hepatitis, and parvovirus, Dr. R.D. Schultz at the University of Wisconsin proposed that a more ideal vaccination program would be one in which dogs were revaccinated one year after completion of the pediatric series, then at three-year intervals thereafter.[1] However, revision of current protocols for these vaccines should not be done without accurate epidemiological data about the prevalence of each disease in the community, and a careful risk assessment for each dog.

Non-Core Vaccines

Non-core vaccines are recommended only for dogs in situations where the risk of exposure is high and the disease can be debilitating.[2] These include vaccines for coronavirus, leptospirosis, parainfluenza, kennel cough, Lyme disease, and giardia. Most non-core vaccines require annual revaccination due to their short duration of immunity. In addition, most of these vaccines contain killed organisms suspended in adjuvant, which increases the risk for vaccine-associated reactions.

Canine **coronavirus** can cause clinical disease in pups less than six weeks old, but most are protected by maternal antibodies.[1] The clinical disease is very mild compared to parvo, and unlike parvo, is confined to the intestinal tract without any systemic involvement. Concurrent infection with coronavirus can contribute to the severity of clinical disease in puppies infected with parvovirus, but vaccination of puppies against parvovirus will prevent disease from both.[1] Therefore, it is difficult to rationalize the use of a coronavirus vaccine, but more doses of multivalent vaccines containing killed coronavirus are sold than those without, indicating that most pups and adult dogs are routinely vaccinated anyway. The vaccine, which contains killed virus in adjuvant, may be most useful for brood bitches in kennels where diarrhea is a problem in young pups prior to weaning. Vaccines containing killed coronavirus combined with killed leptospirosis bacteria targeted for use in puppies should not be used due to increased frequency of hypersensitivity reactions.[1]

Canine **leptospirosis** is a bacterial infection that causes kidney and liver failure in dogs of all ages. The bacterial species, *Leptospira*, has several different variants, called serovars, which are antigenically distinct from each other, thus antibodies to one will not protect against infection with other serovars. Vaccines for leptospirosis contain killed bacteria from the *L. canicola* and *L. icterohemorrhagie* serovars. The killed bacteria are usually incorporated into multivalent vaccines containing modified live distemper virus, parvovirus, adenovirus, and parainfluenza virus (DHLPP, DA$_2$LPP). The killed bacteria suspended in adjuvant are responsible for many hypersensitivity reactions, particularly in Dachshunds and other small breeds, and only induce a short-lived immunity of six to eight months.[1] However, widespread use of multivalent vaccines containing *L. canicola* and *L. icterohemorrhagie* for many years has been credited with the reduced prevalence of these two serovars in the canine population. New serovars, such as *L. Pomona* and *L. grippotyphosa* have now emerged as the predominant cause of canine leptospirosis, and the old vaccines do not induce protective immunity to these bacteria.[1] Recently, Fort Dodge has developed a new killed vaccine that contains *L. Pomona* and *L. grippotyphosa* for use in dogs at risk for exposure, but the duration of immunity is still less than 12months.[1]

Kennel cough, or infectious tracheobronchitis, is an upper respiratory tract disease caused by *Bordetella bronchiseptica* bacteria alone, or in concert with a variety of viruses such as parainfluenza, distemper, canine adenovirus type 2, and herpesvirus. The intranasal vaccine contains live avirulent *B. bronchiseptica* combined with modified live parainfluenza virus. This vaccine rapidly stimulates mucosal and cell-mediated immunity in the upper respiratory tract where the pathogens enter the body, so that vaccination three to five days prior to anticipated exposure provides protection. The intranasal vaccine can also be given to pups after three weeks of age, and is not subject to the maternal antibody blockade. However, replication of the attenuated bacteria and virus in the upper respiratory tract can cause mild clinical signs that resolve in a few days. The parenteral vaccines given by injection contain killed *B. bronchiseptica*, and thus require boosters two to four weeks apart to generate protective levels of immunity. These killed vaccines induce systemic immune responses that contribute to protection of the respiratory tract without inducing a mild clinical disease like the modified live vaccine, but do cause more vaccine-associated hypersensitivity reactions. Parenteral vaccines are safe to use in breeding bitches, but are subject to maternal antibody blockade when given to puppies. The duration of immunity for both the intranasal and parenteral vaccines is probably less than 12

months.[1] It is generally thought that stimulation of local immunity in the respiratory tract with an intranasal vaccine is superior to use of parenteral vaccines. Others have proposed that protocols incorporating both types of vaccines are superior to either vaccine alone. A recent study[6] found that administration of both the intranasal and parenteral vaccine once each in sequence afforded better protection and less severe clinical signs from *B. bronchiseptica* challenge than either vaccine alone.

Lyme disease is caused by *Borrelia burgdorferi* bacteria transmitted by tick bites. The vast majority (99 percent) of cases are in the northeastern, middle Atlantic and upper Midwestern states. Vaccination is recommended for dogs in these endemic areas, but not for dogs in areas of low prevalence, such as the southeast.[1] The Lyme vaccine contains killed bacteria suspended in adjuvant, and has produced postvaccinal lameness in dogs. In addition, the vaccine contains limited strains of the bacteria and may not induce cross protective immunity to other strains that cause disease. A new recombinant vaccine licensed for use in dogs contains purified bacterial out surface protein A (OspA) instead of whole bacteria, does not have an adjuvant, and challenge studies have shown a minimum duration of immunity of one year.[3]

Giardia is a protozoan that infects the gastrointestinal tract of birds and mammals worldwide, and causes explosive diarrhea, gas, and pruritic skin lesions in dogs. The infection responds to medical therapy, but reinfection rates are high because the infectious cysts shed in fecal material persist on the dog and in the environment. There is a new killed vaccine (GiardiaVax, Fort Dodge) licensed for use in dogs and pups older than six weeks that contains inactivated giardia trophozoites. Challenge studies have shown that the vaccine stimulated a strong antibody response within three weeks, and vaccinated dogs were less severely affected clinically and shed cysts for a shorter time compared to non-vaccinated dogs[1]. The decreased shedding of cysts was maintained for up to one year. In a clinical trial supported by the manufacture[7], six pet dogs with chronic giardia infections for months to years received two doses of the vaccine given three weeks apart, which eliminated clinical signs and shedding of cysts with two to eight weeks. However, in a recent independent study[8] in which digs infected with giardia were treated medically with or without the vaccine, medical treatment alone was effective without the vaccine, as long as the dogs were bathed and put in a different environment. The vaccine did not prevent recurrence of infection in dogs that were not bathed and moved to a different environment.

Assessment of Immune Status by Antibody Titers

One method to assess the adequacy of humoral immunity induced by vaccination is measurement of antibody titers to the infectious agent vaccinated against. The vaccine-induce antibody titer is compared to a standard titer associated with prevention of infection. Antibody titers are increasingly recommended as an objective method for determining the need for revaccination. However, antibody titers do not necessarily correspond to protection against disease.[1, 2] A high antibody titer does not guarantee immune protection, and a low or negative antibody titer does not mean loss of immunity and susceptibility to infection. Certain breeds have higher numbers of individuals that do not develop antibody titers after vaccinations. These breeds include the Rottweiler, Doberman Pinscher, Labrador Retriever, Alaskan sled dog, Pomeranian, and American Staffordshire Terrier.[4] Yet, dogs in these breeds with no or low antibody titers remain

healthy, presumably because of vaccine stimulation of other important immune system compartments such as cell-mediated immunity, mucosal immunity, and immune memory cells, all of which cannot be accurately or practically measured at this time. Unfortunately, there are no standardized tests for measuring antibody titers with reliable interpretations, so that submission of a serum sample to three different labs most likely would yield three different results with three different interpretations.[2]

In a recent study[4], serum antibody titers to distemper and parvovirus were measured in 1,441 dogs of various ages and breeds located across the US and Canada. In this population, more than 95 percent had adequate titers to distemper and parvo. For the 468 dogs with known vaccination histories, the interval of time after the last vaccination was one to two years for the majority (60 percent), two to seven years for 30 percent, and less than one year for ten percent. Based on these results, the authors concluded that annual revaccination of adult dogs may not be necessary, and that an acceptable alternative approach is antibody titer screening to determine the need for vaccination on an individual basis. An alternate vaccination protocol has also been proposed for puppies from families with known genetic predisposition to adverse vaccine reactions or immune-mediated diseases.[9] This protocol suggests using only monovalent vaccines for distemper and parvovirus with alternating administration every three to four weeks until the puppy has received a total of three doses of each vaccine. The vaccines are boosted at one year of age, again using monovalent vaccines given at least two weeks apart, followed by measurement of antibody titers to determine future need for revaccination.

Adverse Vaccine Reactions

Just as no vaccine is 100 percent effective in preventing disease, no vaccine is 100 percent free from causing an adverse reaction. An adverse event is defined as any undesirable consequence, including illness or a reaction, after the use of a vaccine, whether or not a cause-and-effect relationship can be established.[2] The most commonly recognized adverse reactions are the nonspecific reactions of immune system stimulation, including fever, anorexia, and stiffness for 24-36 hours after vaccination.[10] Another common systemic reaction, most frequently reported with killed vaccines, is immediate hypersensitivity of anaphylaxis, indicated by urticaria (hives) and pruritis of the face and ears followed by vomiting and/or diarrhea in some dogs.[10] These signs can occur immediately after vaccination or several hours later. Local reactions to vaccines with adjuvants include swelling, pain, lumps, and hair loss at the vaccine site. Other reactions include abortions and birth defects due to vaccination of pregnant dogs, and illness in neonates exposed to dogs recently vaccinated with modified live vaccines that are shedding the vaccine viruses into the environment.

For most currently available vaccines, the benefits derived far outweigh the risks for an adverse event when vaccination is performed in accordance with published standards.[10] With the technology available today, there is no way to accurately predict what vaccine will pose a threat to which dog and when.

Manufacturers of human vaccines are required to list on the vaccine label the type and frequency of adverse events that occurred during safety trials.[10] There are approximately 12,000 reports of adverse events annually for human vaccines, all of which must be forwarded to the FDA, the

federal agency that regulates drugs and vaccines for humans.[10] In contrast, animal vaccine manufacturers are neither required to list possible adverse reactions on vaccine labels, nor keep records of adverse events reported directly to them, nor forward reports to the USDA, the federal agency that regulates drugs and vaccines for animals.[10] There are approximately 10,000 reports of adverse events associated with animal vaccines annually in the US, but the vast majority of these are communicated directly to the manufacturers.[10] However, veterinarians and pet owners can report concerns directly to the USDA or to the Veterinary Practitioners Reporting Program, which is a volunteer watchdog organization that forwards reports of adverse reactions to the USDA, vaccine manufacturer, and the AVMA in an effort to protect animal health.

One of the best-developed surveillance schemes in the world for monitoring adverse reactions to veterinary vaccines is in the United Kingdom. In contrast to the US, vaccine manufacturers in the UK are legally required to record reports of adverse reactions and submit the reports to a regulatory agency.[11] The Veterinary Products Committee, an independent group that gives advice on the safety, quality, and efficacy of veterinary vaccines to the regulatory agency, recently published a report on vaccine-associated adverse events in the UK.[11] The overall annual incidence of adverse events in dogs from 1995 – 1999 was less than 0.1 percent per 10,000 doses of vaccines sold, which is similar to that reported in Australia. Toy breeds and puppies less than six months old had the highest incidence of the five-year reporting period. Anaphylaxix reactions were the most common type reported. The group concluded that there are insufficient scientific data to warrant changing revaccination intervals from that already approved by the regulatory agencies, and that the very low incidence of adverse reactions sdtronly supports continued vaccination. However, the group strongly emphasized that dogs should be individually assessed with regard to the need for each vaccine as well as the frequency of administration.

Immune-Mediated Diseases and Vaccinosis

There is increasing evidence suggesting that vaccination, particularly "**overvaccination**," is associated with development of immune-mediated disorders and chronic diseases, or "**vaccinosis**," in individuals that are genetically predisposed. Certain breeds appear more genetically predisposed to developing adverse reactions and immune-mediated diseases following vaccination, including the Old English Sheepdog, Akita, American Cocker Spaniel, Standard Poodle, Scottish Terrier, Shetland Sheepdog, Shih Tzu, Vizsla, Weimaraner, Irish Setter, Doberman Pinscher and Dachshund.[9] There are two published studies[12, 13] that linked vaccination and development of immune-mediated hemolytic anemia. More recent reports[9, 14] have suggested vaccine-induced development of Hypertrophic Osteodystrophy in Weimaraner puppies that were genetically predisposed to the disease. Other reports linking vaccination to development of joint diseases, neurological diseases, and thyroid disease are largely anecdotal, and await rigorous scientific validation. To date, there are no controlled scientific studies that prove a cause and effect relationship between vaccination and development of immune-mediated diseases or chronic diseases.[10, 11]

Due to concern about vaccination overload of the immune system with development of autoimmune diseases and "vaccinosis," there has been a proliferation of anti-vaccination websites offering alternatives to conventional vaccinations. One of the alternatives is "nosodes"

which are products prepared from infected tissues or discharges given orally.[2] General recommendations for use of nosodes include administration orally for three days the first week, then once weekly for three weeks, then once monthly for six months, then every six months thereafter. The dose is three drops for small dogs and six drops for large dogs. The nosode does not cause disease because of "homeopathic dilution," where the product is diluted enough that the amount of infectious material remaining is too little to cause disease. There has been only one controlled study on the efficacy of nosodes for protection against disease. This study[2] examined a nosode for parvovirus and found that 100 percent of the non-vaccinated as well as vaccinated puppies became infected when challenged with the virus.

Conclusion

Are we vaccinating dogs with too many vaccines too often? Probably. No doubt, there will be changes in the who, what, when and how for canine vaccinations. In general, canine vaccines are effective and safe, with benefits that far outweigh the risks. However, more rigorous controlled studies are needed on vaccine efficacy, duration of immunity, and safety to point out a scientifically sound direction for change. Change is hard, and human nature resists change unless there is compelling evidence to do so. It is important to remember that **the overall goal of vaccination is protection of the population as a whole, which can be achieved by vaccinating more dogs, but vaccinating each dog less**. To date, the best protocol for vaccination is individual assessment of every dog with regard to age, health status, risk of exposure to the infectious agent, prevalence of the infectious agent in the community, and severity of illness caused by the infectious agent.

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Nutrition for Working Dogs
V. Richard Hill, MA, VetMB, PhD, DACVIM, MRCVS

What factors are important?
1. **Type of Exercise**: Endurance vs. sprinting; most working dogs are endurance athletes.
2. **Training**: Slow increases in exercise and slow adjustment to new nutritional needs are best. Being a "weekend warrior" and suddenly increasing exercise and diet can result in injuries and digestive upset.

What is unique about the exercising dog?
Fat Metabolism:
1. Dogs do not get heart attacks and do not need to worry about increased fat.
2. Dogs burn fat twice as fast as people do.
3. All dogs' muscle fibers burn fat whereas some muscle fibers in horses and people do not
4. **Stamina improves when dogs are fed a high fat diet** (50% energy). This is the opposite of people who need increased carbohydrate for stamina.

Effect of diet on stamina in Beagles on a treadmill (Downey et al. 1989)

Dietary Protein (%energy)	Dietary Fat (%energy)	Time (min) to exhaustion	Distance (miles)
20%	30%	100	15
20-40%	50-70%	140	20

5. Dogs sprint faster when fed increased fat.
6. Dogs "tie up" less when fed high fat diets.

How much protein?
7. **Dogs require at least 30% energy as protein** for endurance exercise to prevent anemia.
8. Dogs do not require more than 24% energy as protein for sprint exercise.

Types of Pet foods
9. **Dry foods** are formed by an extruder and are **mostly low fat** (~ 25% energy as fat) because the extruder requires a low fat mixture. These are fine for dogs that are couch potatoes but do not contain enough fat for working dogs undertaking endurance exercise. More expensive dry diets have fat sprayed on after extrusion and tend to contain more fat (40% energy as fat). They are packaged in special greaseproof bags and are greasy to the touch. These should provide the staple diet for a working dog. It is enough on its own if the dog is not working. It is not necessary to feed diets designed for large breed dogs.
10. **Canned foods** contain 75% moisture and are more expensive but usually contain more fat and protein. The analysis on the bag cannot be compared with that on a dry food because the amount of water is greater in canned food. For a rough comparison, multiply the analysis on the canned food by four to compare with that of a dry diet. Working dogs which are normally fed dry food should be supplemented with canned food when they are working. Foods designed for growing dogs mostly contain increased protein and fat.
11. **Soft-moist** and **soft-dry** (e.g. Kibbles and Bits): These are intermediate but mostly low fat and are not suitable for working dogs.

Note that high fat is here defined as greater than 40% energy which is equivalent on the label to greater than 5% fat for a canned diet, greater than 13% in a semi-moist diet and greater than 18% fat in a dry diet. High protein is defined as greater than 30% energy, which is equivalent on the label to greater than 9% in a high fat canned diet or greater than 33% in a high fat dry diet.

Differences between pet foods

Differences between pet foods are often small. Foods with different names do not necessarily differ in composition. Some terms such as "premium" and "super-premium" have no definition and do not guarantee better performance. The major differences are:

12. **Dry vs. canned**: Canned usually contain more fat and protein than dry diets.
13. **Generic vs. proprietary**: Generic diets are usually made with poorer quality ingredients and are not necessarily tested on animals. Proprietary (popular & premium) brands made by national manufacturers are made with better ingredients and are usually tested on animals. "Generic" diets are inexpensive private label of a local or regional manufacturer. Pet food is well regulated but there is no policing of label claims within the state of Florida. It is therefore better to use a food that is sold nationally.
14. **Life stage and therapeutic diets**: These sometimes have different compositions and should be used only with a veterinary recommendation.

Supplements and treats

15. Unbalance balanced diets. Do not feed supplements. Treats should comprise less than 10% of the diet
    a. Too much meat can result in thin bones and fractures.
    b. Too much liver can cause stiff neck and joints.
    c. Too much calcium can cause joint problems.
16. Some treats and supplements may be beneficial:
    a. Chews: may be beneficial for dental hygiene.
    b. Antioxidants may be beneficial especially in dogs that are not properly trained or are eating a lot of extra fat but very high doses are not recommended. Some manufacturers are including increased antioxidants in their dog foods. If feeding a diet without increased antioxidants, 10 IU/kg vitamin E (as alpha-tocopheryl acetate) may be given daily or 50 IU/kg every week by mouth. Vitamin C may also be beneficial. It is probably best to give vitamin C immediately before exercise as it does not last long in the blood and 100 mg may be sufficient for the average medium to large sized dog. It should be noted, however, that high doses of antioxidants may have detrimental effects. Very high daily doses of vitamin E (1000 IU) and C (1 gram) are not recommended because our laboratory team found that these high doses appeared to slow racing greyhounds weighing 25-30 kg. Additional vitamin C in particular may not be beneficial and could exacerbate injury.
    c. Glucosamine or green lipped mussel powder may help dogs with arthritis but should only be used in consultation with your veterinarian.
    d. Fish oil may reduce inflammation of the feet in dogs working in snow. Some diets already contain fish oil and should not be supplemented.

Human food
17. Not complete and balanced so must have supplements such as vitamins and minerals added of more than 10% of the diet. This is not recommended unless diet has been formulated to be balanced by a professional with nutritional experience.
18. Uncooked meat represents a likely source of infection especially in young, pregnant, or infirm animals, or stressed animals such as working dogs.
19. Bones, especially spiky bones such as the vertebrae found in chicken necks, can get lodged in the esophagus. Too many bones can also cause constipation.

Neutraceuticals and Herbs
Quality, consistence, absorption, potency and efficacy are uncertain. Toxicity and therapeutic index have not been established. Some may prove beneficial in the future but are not currently recommended.

How much to feed?
Adjust food intake to maintain optimum body weight and condition. Do not feed too much. The slim-line model is best. *Ribs should be felt but not seen. There should be a waist visible from the side and from above.* The recommendation on the back of the packet can provide a guide but there is much individual variation. There is some evidence that lean dogs perform better than heavier ones. Lean dogs also live longer and have fewer joint problems.

Weigh your dog every two weeks and keep a record. Always use the same scale and do it before a meal but after urinating and defecating at the same time each day and not after exercise. Sudden changes in body weight are an indication of dehydration. Slow changes will give some indication of whether you are feeding too little or too much. Also keep a record of body condition score. Take a photo for comparison so you can see how things change over time. Your dog should have a body condition score of five on the nine point Purina scale.

A working dog, such as a Collie, working sheep needs almost twice as much food as a couch potato pet dog. A racing sled dog has the most extreme work out and may need twice as much again. When starting training, add ½ 16 oz. can for each 8 oz. cup of dry food normally fed. Over three days, reduce dry by half and double canned food. Then increase canned food as necessary to maintain weight, body condition and stamina.

When and how often to feed?
Do not feed directly before or during exercise. Do not exercise within 8-12 hours of a large meal or 4-6 hours of a moderate sized mea. Racing sled dogs have long bouts of exercise and short rests. The custom in these dogs is to race for 4-6 hours then take a rest for 2-4 hours during which a high fat and protein snack is fed. Dogs then race for an additional 4-6 hours before eating a full meal.

Water
Dehydration must be avoided. Offer water continuously during exercise. Pet dogs on average need 50 mL/kg per day (2-5 pints per day for a 45-100 lb working dog). Working dogs may need four or five times that amount (1-3 gallons/day). Adding three level tablespoons of table

sugar to a liter of water may help water absorption and will increase the recuperation of dogs at rest stops.

Salt and other electrolytes

There should be enough salt in the food if a balanced diet is being fed. Salt may improve water absorption in dehydrated animals. A recipe for oral electrolyte replacement solutions is provided below which can be used if a dog becomes dehydrated but a recent study showed no benefit of such a solution over plain water in working dogs. Giving sodium bicarbonate (a "shake") before exercise also has been shown not to be beneficial in dogs.

Oral rehydration recipe (level spoonfuls)
To one liter of drinking water add:
1. 3 Tablespoons of table sugar or 1 ½ tablespoons of glucose
2. ½ teaspoon of table salt
3. ¼ teaspoon of salt substitute
4. ½ teaspoon of baking soda

Use immediately.

Gastric Dilatation-Volvulus: "Bloat"

20. Predisposing factors suggested by epidemiology (Glickman et al):
    a. Large size
    b. Large depth to width ration for body conformation
    c. Male gender
    d. Being underweight
    e. Eating only one meal per day
    f. A faster rate of eating
    g. Fearful or nervous temperament
    h. An event perceived
21. Reduced risk:
    a. Table scraps or canned food included with a dry dog food
    b. Happy and easy-going character
22. Recommendation:
    a. Feed twice daily
    b. Add some canned food to dry diet
    c. Avoid stress associated with eating
    d. Do not allow to be underweight

Recommendation:
23. Feed a national brand pet food that says on the label that it has been tested using AAFCO approved feeding and is complete and balanced.
24. Feed a high fat dry food for maintenance. Add a canned food during periods of work.
25. Feed enough to keep dog lean.
26. Do not feed supplements such as meat, bones, calcium or vitamins.

27. Give 50 IU/kg vitamin E once a week by mouth. You may also give 100 mg vitamin C one hour before exercise and repeat every 6-8 hours during exercise but this may not be beneficial and could exacerbate injury.
28. Keep treats to a minimum (greater than 10% of the diet). Most of the diet should be pet food.
29. Give a dental chew once daily.
30. Make sure access to water at least every half hour during exercise.
31. Feed after exercise not before or during exercise.
32. Rest dogs after 4-6 hours work. If the dog is expected to work again within a few hours then give sugar water and a high protein, high fat snack.

Overview of Canine Dental Health and Disease
Susan E. Anderson, DVM, DABVP
(1) University of Florida

Why is veterinary dentistry important?
- Every pet has a mouth
- 85 percent of pets over two years have some evidence of periodontal disease
- Dental health contributes to overall health
- Goal should be prevention rather than treatment

Dental Disciplines
- Pedodontics: puppy dentition
- Orthodontics: guidance and correction of malocclusion
- Periodontics: treatment of supporting tissues of teeth
- Endodontics: treatment of disease affecting tooth pulp
- Exodontics: extraction of teeth
- Oral surgery: surgery of the oral cavity
- Restorative Dentistry: restoration of form and function
- Prosthodontics: construction of appliances to replace missing teeth and/or adjacent structures

Anatomy

Head
- Mesocephalic: German Shepherd Dog, Labrador
- Brachycephalic: Boxer, Bulldog
- Dolichocephalic: Collie, Greyhound

Dentition
- Incisors: nibbling, grooming
- Canines: holding, tearing; largest and strongest teeth
- Premolars: cutting, holding, shearing
- Molars: grinding

Tooth Structure
- Teeth designed to be self-cleaning
- Natural diets of fiber, sinews, tendons
- Conical in shape
- Few contact points to trap debris
- Alkaline pH of saliva deters bacteria

Periodontium
- Supporting structures of the teeth
- Gingiva, periodontal ligament, alveolar bone

Tooth Anatomy
- Crown
- Root
- Enamel: covers the crown, hardest substance in body
- Dentin: bulk of the tooth structure
- Pulp: blood and nerve supply
- Gingival sulcus: space between gingival and tooth, 1-3 mm
- Cementum: covers root

Dental Formula

Deciduous

$$2 \left\{ I\ \frac{3}{3}\ C\ \frac{1}{1}\ P\ \frac{3}{3} \right\}$$

Permanent

$$2 \left\{ I\ \frac{3}{3}\ C\ \frac{1}{1}\ P\ \frac{4}{4}\ M\ \frac{2}{3} \right\}$$

Eruption Times

Deciduous teeth
- Incisors: 3-4 weeks
- Canines: 3 weeks
- Premolars: 4-12 weeks

Permanent teeth
- Incisors: 3-5 months
- Canines: 4-6 months
- Premolars: 4-6 months
- Molars: 5-7 months

Normal Occlusion
- Upper incisors in front of lower incisors (scissors bite)
- Lower canine fits evenly between upper canine and 3rd incisor
- Premolars fit in a "pinking shear" fashion, interdigitate
- Upper 4th premolar fits outside (lateral) to lower 1st molar

Malocclusion
Class 0 occlusion: normal or normal for breed
Class 1 occlusion: jaw relationship normal but one or more teeth out of position
- Base narrow canines
- Anterior crossbite
- Lance canine (spear, tusk)
- Posterior crossbite

Class 2 occlusion: mandible short in relation to maxilla – brachygnathic
- Overbite
- Unilateral wry (1/2 maxilla short)

Class 3 occlusion: maxilla short in relation to mandible – prognathic
- Underbite
- Unilateral wry (1/2 mandible short)

Pedodontics
Missing teeth
- Never developed, never erupted, trauma
- Radiograph (x-ray)

Retained deciduous teeth
- No 2 teeth of the same type should occupy the same space!
- Causes malocclusion, crowding
- Increased incidence of periodontal disease and tooth loss
- Extraction as soon as possible
- Caution not to disturb developing permanent tooth

Interceptive orthodontics
- Early extraction of deciduous teeth to prevent "interlock"
- Removes interference to allow for maximal jaw growth
- Will not cure a pre-existing genetic problem
- DO NOT trim or cut deciduous teeth – pain, infection, potential damage to permanent tooth

Fractured deciduous teeth
- Frequent: running into objects, catching hard toys, overzealous play or tug-o-war
- Requires extraction
- If untreated: pain, infection, damage to permanent tooth especially enamel

Supernumerary teeth
- Extra teeth
- Usually incisors or premolars but can be canines
- Can cause crowding; if so, extract

Cranial mandibular osteopathy
- Inherited condition, most common in West Highland White Terriers
- Non-neoplastic (not cancerous) bone formation of the temporomandibular joint and occ spreads to mandible
- Pain, fever, reluctance to eat, difficulty opening jaw
- Treat symptoms of pain, lessens with age

Periodontal Disease
- Inflammation of the structures supporting the teeth
- Normal gingival: smooth, coral pink, well attached, sulcus 1-3 mm
- Plaque: accumulation of bacteria, glycoproteins, polysaccharides that adhere to tooth
- Tartar (calculus): plaque mineralizes within 48 hours, forms more readily in alkaline saliva

Predisposing Factors for Periodontal Disease
- Overcrowded, rotated teeth
- Retained deciduous teeth
- Soft, sticky diet with no brushing
- Slab fracture of tooth exposing rough surface for plaque accumulation
- Malocclusions
- Trauma
- Chemical irritants
- Systemic disease (uremia, diabetes)
- Open mouth breathing (dries saliva)

Clinical Signs of Periodontal Disease
- Halitosis
- Inflamed gums and/or bleeding
- Asymmetrical facial swelling
- Mobile teeth
- Gingival recession
- Nasal discharge
- Exposed roots
- Gingival pockets (> 3mm)
- Vertical and horizontal bone loss

Smaller Breeds More Predisposed to Periodontal Disease
- Closer teeth decreases tooth's self-cleaning ability
- Smaller the dog the thinner the supporting bone
- Bacterial and inflammatory by-products can damage thin bone quickly
- Bone thinnest at incisors, frequent location of disease loss
- Smaller dogs live longer
- Open mouth breathing more frequent

Stages of Periodontal Disease
Stage 1: Gingivitis
- Only reversible state
- Gingival becomes swollen, rolled and red

Stage 2: Early periodontitis
- Increased gingival pocket depth

- Up to 25 percent attachment loss
- Bacteria begins to change from aerobic to anaerobic
- Teeth stable

Stage 3: Moderate periodontitis
- Deeper pockets
- More cirulent anaerobic bacteria colonizes
- Up to 50 percent attachment loss (bone and gingival)
- Slight mobility of teeth

Stage 4: Severe periodontitis
- More than 50 percent attachment loss
- Increased severity of infection
- Salvageability of teeth is questionable
- Tooth mobility can be great

Treatment of Periodontal Disease
- Thorough prophylaxis – complete oral exam, supra gingival scaling, root planning/subgingival curettage, polish, flush, repeat exam
- Radiographs
- Root planing – closed (<5 mm) vs. open (>5 mm)
- Extractions, periodontal surgery, endodontics (root canal)
- COMMITMENT to home care
- Reassess in 1-3 months

Indications for Antibiotic Use
- Oral ulceration
- Severe periodontitis
- Evidence of systemic disease (renal, cardiac, diabetes, Cushing's)
- Additional surgery being performed
- Bone implants (hip replacement)
- Pulp capping

Antibiotics
- Ampicillin
- Amoxicillin
- Amoxicillin-clavulanate (clavamox)
- Clindamycin (antirobe)
- Metronidazole (Flagyl)
- Doxycycline

Other Dental Abnormalities
Caries (cavities)
- Bacterial degradation of enamel, not common in dogs
- Maxillary 1st molar most common

Abrasions (external source)
- Excessive grooming
- Toys especially tennis balls
- Rocks, fences, cages

Attrition (wear from other teeth)
- Malocclusion especially level bite
- Increased risk of fracture if mid tooth (canine)

Enamel hypoplasia (reduced formation of enamel, enamel dysplasia)
- Hereditary
- Systemic infection causing high fever during tooth formation
- Viral infection during tooth formation (distemper)
- Enamel organ damaged during early extraction of deciduous tooth
- Other trauma during formation

Discolored teeth
- Pink – purple – tan
- Pulpal hemorrhage and/or tooth death usually due to trauma

Inapparent oral nasal fistula
- Pocket that communicates with nasal cavity
- Lingual side of upper canine
- Rest of tooth may have little disease present
- More common in small dogs

Foreign bodies
- Wedged between upper 4th premolars
- Sticks, bones

Home Care
Brushing
- Gradual training with reward/positive reinforcement
- Ideal is daily
- Cleaning under edge of gums most important (gingival sulcus)
- Nothing truly replaces the mechanical disturbance of plaque
- Finger brush, gauze over finger (doesn't clean sulcus well)
- Pet designed brush or small, soft child's brush
- DO NOT use hand scalers or curettes regardless of training
    - Trauma to gingival
    - Excessive wear to enamel (not that thick)
    - Without polishing, roughened surface of the enamel allows plaque to return that much faster

Toothpastes
- Many choices
- Avoid human toothpaste
    - Too much fluoride – dogs don't spit
    - Foaming agents can cause vomiting

Rinses
- Chlorhexiding gluconate 0.12 percent oral rinse
  - CHX, Nolvadent, Hexarinse
  - Inhibits plaque formation
  - Bacteriostatic and bactericidal
- Zinc gluconate/Vitamin C
  - Maxiguard
  - Promotes healing of ulcerated oral tissues

Chew Toys
- Monitor to avoid swallowing, fractured teeth, choking
- Kong toys – dental
- Nylabone products
- Rawhides, some have tartar control coating (CET)

Diets and Treats
- T/D (Hill's Pet Nutrition) fibers designed to squeegee teeth
- Tartar Check (Heinz) coated with hexametaphosphate
- Dentabone (Waltham)
- Dental Chew (Waltham)

What's New With GDV?
Christopher A. Adin, DVM, DACVS
University of Florida

Background:

Gastric distention and volvulus (GDV) is an acute, life-threatening disease that primarily affects large and giant breed dogs. In dogs with GDV, the stomach rapidly fills with gas and fluid. Rotation of the stomach on its axis (volvulus) prevents emptying of the stomach contents and occludes the blood supply. Clinical signs in affected dogs include sudden swelling of the abdomen, non-productive retching and lethargy. Severe distention of the stomach can decrease the amount of blood returning to the heart, compromise breathing, and cause death of the stomach wall.

Diagnosis and Treatment:

Due to advancements in the medical and surgical treatment of dogs with GDV, mortality rates have decreased from 42 percent in the 1980s[1] to 18 percent in recent years.[2] Early diagnosis by a veterinarian followed by rapid intravenous fluid treatment and decompression of the stomach has become the standard of care. Dogs that are recovering from GDV require critical care and 24 hour monitoring due to the risks of irregular heart rhythms, bleeding tendencies and sepsis (infection).

Recurrence:

The rate of GDV recurrence approaches 70 percent if the stomach is not surgically fixed to the body wall (gastropexy) after the first incident. Fortunately, surgical gastropexy prevents recurrence in 90-100 percent of dogs. As a result, it is recommended that surgical gastropexy be performed after treatment for shock in dogs with GDV. At the time of surgery, the veterinarian is able to evaluate the viability of the stomach wall and other organs which may need to be removed or repaired, depending upon the degree of damage that has occurred.

New developments… The newest trend that I can foresee in veterinary medicine is a shift in focus from the *treatment* of GDV that has already occurred to the *prevention* of GDV in high risk breeds.

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Risk factors and epidemiology

Recent studies by Dr. Larry Glickman at Purdue University[1] have identified a number of risk factors for GDV which include: increasing age, having a first degree relative with GDV, faster speed of eating, raised food bowl, and specific breed-related risks (e.g. Great Dane). Overall, a pure-bred large or giant breed dog had a lifetime risk of 24 percent and 21.6 percent of developing GDV, with a 30 percent chance of death from this incident. For Great Danes, this lifetime risk increased to 42.4 percent.

Prophylactic gastropexy

Based on the aforementioned statistics, many surgeons (myself included) feel that prophylactic gastropexy is indicated for high-risk large and giant breed dogs. Prophylactic gastropexy is an elective procedure involving surgical attachment of the stomach to the abdominal wall in an attempt to prevent the occurrence of GDV. The procedure is typically recommended at the time of elective ovariohysterectomy or castration in pet dogs. Though elective gastropexy may be performed at any time in breeding animals, it would be most reasonable to perform the procedure early in life, as the incidence of GDV increases with age.

Prophylactic gastropexy may be performed through a standard midline abdominal incision or by laparoscopic techniques.[2] Laparoscopy involves the use of a tiny camera and light source so that the surgeon may operate through small incisions, avoiding some of the pain and prolonged recoveries that can be associated with more invasive surgical procedures. Elective ovariectomy may be combined with the laparoscopic procedure in non-breeding animals. Depending upon the technique employed, the complications associated with prophylactic gastropexy are minimal and are infrequent. When the risks of the occurrence of GDV and the associated cost and mortality rate of this condition are considered, prophylactic gastropexy would appear to be an excellent option for dog owners with large or giant breed dogs.

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Tick-Borne Diseases: An Emerging Threat
Dr. Brian J. Luria
University of Florida

Introduction

Our awareness in human and veterinary medicine that certain insects and arthropods transmit disease has been established for many years. In recent years, our knowledge has increased dramatically, mostly due to advancements in our ability to diagnose these diseases.

For a variety of reasons, ticks are appearing in greater numbers than ever. Ticks are a very important cause of debilitating and deadly diseases and conditions in both humans and domestic animals. Almost equal to the disease transmitting potential of ticks, is the fear and concern that arise among many owners and veterinary staff members when a tick is found on a dog or cat.

This discussion will focus on education regarding the diseases that ticks can transmit, how to diagnose and treat them, and how to prevent your dogs from acquiring ticks and the diseases they transmit.

Brief Review of Ticks

Ticks are blood feeding external parasites of mammals, birds, and reptiles throughout the world. Ticks are second only to mosquitoes as vectors of pathogens. A **vector** is simply an organism that transmits a pathogen. For this discussion, we will focus on four tick species, based on the diseases that they transmit.

1. The Black-legged or Deer Tick (*Ixodes scapularisI*)
This tick is concentrated in both the North and Southeastern United States as well as in areas surrounding the Great Lakes. It is the tick that transmits B*orrelia burgdorferi*, the causative agent of Lyme disease as well as the agents of Babesiosis.

2. The Brown Dog Tick(*Rhipicephalus sanguineus*)
This tick has a widespread distribution. It is the tick that transmits *Ehrlichia canis*, one of the causative agents of Ehrlichiosis as well as *Babesia canis* and *Babesia gibsonii*.

3. The American Dog Tick (*Dermacentor variabilis*)
This tick is widespread throughout the US as well as parts of Canada and Mexico. It is the most important vector of *Rickettsia rickettsii*, the causative agent for Rocky Mountain spotted fever in the eastern US.

4. Rocky Mountain Wood Tick (*Dermacentor andersoni*)
This tick is found mostly in the Northwestern parts of the United States. It is an additional vector for Rocky Mountain spotted fever.

Basic Tick Lifecycle

Depending on the tick and environmental conditions, the lifecycle of a tick can range from a few months to two years. Each developmental stage of a tick's life requires a blood meal in order to reach the next stage. Some species can survive for years without feeding.

Egg Stage
Female ticks lay eggs in secluded areas where vegetation is dense and several inches high. Adult females of some tick species lay about 100 eggs at a time; others lay 3,000 to 6,000 eggs per batch. Regardless of species, tick eggs hatch in about two weeks.

Larval Stage
After hatching, the larvae move into grass or shrubs in search of their first blood meal. If you or your pet passes by, they attach themselves and crawl upward in pursuit of an area of the skin that they can feed from. Then they drop off the host, back into the environment.

Nymphal Stage
After finding their first blood meal, the larvae molt into their nymph stage and begin searching for another host. Nymphs are the size of a freckle and often go undetected, increasing the chance for disease transmission.

Adult Stage
Once the nymph has had its blood meal, it matures into adulthood. Adult female ticks feed on a host for eight to twelve days. In some cases, they will increase to 100 times their original weight while feeding. While still on the host, the female will mate, fall off and lay her eggs in a secluded place – beginning the lifecycle again.

Review of Common Diseases

I. Lyme Disease (Lyme Borreliosis)

    A. Introduction
Lyme disease was named in 1977 when arthritis was observed in a cluster of children in and around Old Lyme, CT. The first case of canine Lyme disease was reported in 1984. Lyme disease is caused by the bacterium, *Borrelia burgdorferi*. These spiral shaped bacteria are transmitted to humans and animals by the bite of infected deer ticks in the genus *Ixodes* (Deer ticks). Ticks, small rodents, and other non-human vertebrate animals all serve as natural reservoirs for *B. burgdorferi*. This means that the Lyme disease bacteria can live and grow within these hosts without causing them to die.

    B. Transmission

        1. Black-legged ticks, also known as Dear ticks (*Ixodes scapularis*) are responsible for transmitting Lyme disease bacteria to humans and animals in the northeastern and north-central United States. On the Pacific Coast, the bacteria are transmitted by the western black-legged tick (*Ixodes pacificus*). Ixodes ticks are much smaller than common dog ticks. In their larval and nymphal stages, they are no bigger than a pinhead. Ticks feed by inserting their mouths into the

skin of a host and slowly taking in blood. Ixodes ticks are most likely to transmit infection via saliva after feeding for two or more days. This amount of time is needed for the Borrelia organism to travel from the tick's midgut to the salivary glands where it is transmitted.

    2.   In the spring, about 35 percent of ticks are thought to be harboring Borrelia, whereas up to 50 percent of adult ticks may be infected with the Borrelia organism in the fall.

C. Signs of Disease

    1.   Within days of the initial tick bite, animals will show flu-like symptoms (fever, malaise) and possibly enlarged lymph nodes (in humans, a "bull's-eye" lesion can be seen on the skin at this stage). Up to two to three months later, signs of an acute inflammatory process is seen (joints, heart, brain, eyes can all be affected). Over years of being affected, chronic arthritic changes can be seen.

    2.   About 75 percent of dogs will get acute arthritis which usually lasts two to four days and will often resolve on its own. However, dogs can have recurrent bouts of arthritis. After about 100 days post tick exposure, signs of acute arthritis resolve, even though animals are still infected.

D. Diagnosis

    1.   History, along with clinical signs, positive serology (see below), and prompt response to antibiotic therapy are usually the criteria for the diagnosis of Lyme disease in dogs. If animals are having acute arthritic signs, a sample of joint fluid can be taken for analysis. Affected animals often have high numbers of neutrophils (a white blood cell often present when fighting bacteria) in their joint fluid.

    2.   Serologic testing is the most common modality used for diagnosis. Serum is the liquid part of the blood that is left when the blood cells are removed. It is comprised of water (naturally) as well as a very high content of various proteins. These include albumin – a protein that aids in the proper retention of water in the bloodstream and globulins – which are antibodies. Serological tests refer to a laboratory test done on blood serum to measure antibodies to infections.

    a.   The most common screening serologic test for Lyme disease, as well as many other infectious diseases, is called an enzyme-linked immunosorbent assay (or ELISA) which quantifies the presence of specific antibody produced by the immune system against specific organisms, such as Borrelia.
        A positive serology detecting antibodies directed against *Borrelia burgdorferiI* is merely an indication of exposure to the organism or to a vaccine which contains antigen (single subunit vaccine) or multiple antigens (bacterin) to the spiorchete. An antigen is a substance capable of stimulating an immune response. **A positive serology by itself does not constitute a diagnosis of Lyme disease. Antibodies persist in infected animals and do not decrease after antibiotic therapy.

    b.   *The ELISA test cannot distinguish between antibodies produced against exposure to a natural infection and those produced against the vaccine for Lyme disease.* In order to separate these two, another serologic test called Western blot testing is traditionally done to confirm natural exposure.

c. Recently, a test has been developed that detects the presence of antibody directed against a very specific surface antigen of *Borrelia burgdorferi* that is produced only in response to active infection. This antigen is called the C6 antigen. The only commercially available test for this is made by IDEXX Laboratories, Inc., and is called the SNAP3Dx™. It is a combination test that also tests for *Dirofilaria immitis* antigen (Heartworm) and *Ehrlichia canis* antibody (see below). This test should help veterinarians determine if a dog has anti-*Borrelia burgdorferi* antibody induced by infection, rather than by vaccination.

E. Treatment

1. It is important to remember that prevention measures can be effective in reducing exposure to infected ticks, and most patients can be successfully treated with antibiotic therapy when diagnosed in the early stages of Lyme disease.

2. Antibiotics are the treatment of choice for Lyme disease in dogs, as in humans. Tetracyclines such as Doxycycline or lactam antiobiotics such as Amoxicillin have been found to be very effective. Animals should be treated for at least four weeks.

3. Dogs usually respond with clinical recovery within 24 – 48 hours; however the organism can persist, hence the duration of antibiotic therapy. Evidence to date indicates that in spite of adequate antibiotic therapy, infected individuals may be infected for life.

4. Anti-inflammatory medications, such as carprofen or etodolac can be used to alleviate arthritic pain in the short term. Corticosteroids, such as prednisone, should be avoided because of their ability to suppress the immune system.

F. Prevention, Tick Control and Public Health Concerns

1. Vaccinating dogs for Lyme disease is still a controversial issue in veterinary medicine and arguments for and against have been postulated over time. There are strong arguments in favor of vaccinating dogs in endemic areas against Lyme disease.

2. There are currently 2 different types of Lyme vaccines for dogs:

a. Whole-cell bacterin vaccine (LymeVax®, Fort Dodge; Galaxy Lyme®, Schering-Plough Animal Health), which consists of a complete killed *B. Burgdorferi*. This vaccine has been available for several years, however, with recent advancements in vaccine technology, it is the less preferable vaccine if one is to vaccinate their dogs.

b. Recombinant vaccine (rLyme®, Merial), which consists of only parts of *B. burgdorferi* that are thought to provide protection from infection and/or disease in dogs. This vaccine consists of outer surface protein A (OspA) of *B. burgdorferi*. The mode of protection appears to involve killing the bacteria in the tick, prior to its transmission into the host animal.

c. So, *what dogs should be vaccinated*? It is clear, based on the mode of action of these vaccines, particularly the recombinant vaccine, that vaccinating already infected or exposed dogs will not cure them and may not prevent them from being infected. **Naïve, or unexposed, dogs in endemic areas appear to be the best candidates for vaccines.**

       d. A fatal kidney infection by *B. burgdorferi* has been reported in some dogs in endemic areas where antibiotic treatment had little effect. There has been no convincing evidence that vaccines could be related to this disease, however, the possibility of a connection does exist.

  3. Tick control is discussed in detail at the end of these notes.

  4. Dogs do not appear to be reservoirs for human infection.

II. Ehrlichiosis

  A. Introduction

Canine ehrlichiosis is an infectious rickettsial disease of dogs caused by a variety of different ehrlichial species, the most notable one being *Ehrlichia canis*. Rickettsia are rod-shaped, coccoid, or diplococcus-shaped, often pleomorphic bacteria that cause various diseases. *E. canis* was first recognized in Algeria in 1935 and first reported in the United States in 1963. The disease gained prominence due to devastating losses of military working dogs stationed in Vietnam. Ehrlichiosis is an illness characterized by a reduction in cellular blood elements.

  B. Transmission

  1. *Ehrlichia canis* is transmitted by the vector tick *Rhipicephalus sanguineus*, or brown dog tick. This tick is also responsible for spreading Babesiosis (see below). This tick prefers to feed on dogs over humans.

  2. Canine infection occurs when salivary secretions from the tick contaminate the attachment site during ingestion of a blood meal.

  C. Signs of Disease

  1. There are three phases of disease seen with Ehrlichiosis: acute, subclinical and chronic:

       a. Acute: After an incubation period of 8 – 20 days, the infected dog enters into the acute phase, which lasts about two to four weeks. During this time, the organism multiplies within circulating white blood cells and is transported to all areas of the body. Fever, depression, malaise, anorexia and weight loss, discharge from the eyes and nose, enlarged lymph nodes and occasionally swelling of the legs can all be seen during this time. Platelets, a cell line responsible in part for blood clotting, often are decreased during this phase of disease by a variety of mechanisms.

       b. Subclinical: Occurs six to nine weeks post-infection and is characterized by persistence of low platelet count, variably low white and red blood cell counts in the absence of obvious illness. Dogs with an adequate immune system may eliminate the infection during this phase.

       c. Chronic: Dogs unable to effectively rid the organism become chronically infected. Elevated globulin levels in the blood are often seen. In this stage of disease, many dogs develop bone marrow suppression (the bone marrow is where the majority of our cells are made). Clinical signs in this stage are variable. Some dogs show no signs of illness, while others can show a variety of clinical signs, including many bleeding abnormalities, particularly from the nose.

2. Laboratory abnormalities that can be seen include deficiencies in all cell lines, red, white and platelet. Occasionally a dramatic increase in the lymphocyte white blood cell line is seen.

3. Bone marrow examination usually reveals low cell numbers with varying degrees of suppression of all cell lines. However, elevation in the number of plasma cells, another white blood cell, is a frequently reported finding.

4. Serum proteins are often abnormal. Elevated globulins are commonly seen. Sometimes this is seen as elevation of all of the globulins equally, or just one of them. If only one globulin is elevated, described as a monoclonal gammopathy, this can easily be confused with a type of cancer called Multiple Myeloma. Often plasma albumin is low.

D. Diagnosis

1. Morulae (intracellular inclusions composed of clusters of organisms) are observed rarely and almost exclusively during the acute stage of infection. However, if these are observed, they are definitively diagnostic.

2. Serologic testing is again the most common means of diagnosing Ehrlichiosis. The indirect fluorescent antibody (IFA) technique is currently recommended. The only drawback is that there is often cross-reactivity amongst different species of *Ehrlichia*.

3. Polymerase chain reaction (PCR) can be used to confirm diagnosis, as this is a molecular test that tests for the presence of DNA of the ehrlichial organism.

4. As discussed in the Lyme disease discussion, IDEXX Laboratories has marketed an in-house ELISA antibody test for *E. canis* that is calibrated at approximately 1:100. Again, it needs to be said that a positive test should be interpreted in combination with history, clinical signs, or laboratory abnormalities since a positive test does not necessarily mean that the dog has active disease.

E. Treatment

1. Tetracyclines or Doxycycline, administered for at least three weeks is the treatment of choice for Ehrlichiosis. Dramatic clinical improvement generally occurs within 24-48 hours after initiation of appropriate antibiotics.

2. If an animal is in the chronic phase of the disease and has bone marrow changes, it can take up to 120 days for the bone marrow to regenerate following treatment.

F. Public Health Concerns

There is no evidence that direct transmission of ehrlichial species from dogs to humans occurs.

III. Rocky Mountain Spotted Fever

A. Introduction

1. Rocky Mountain spotted fever (RMSF) is an infectious rickettsial disease of dogs, which is characterized by sever vascular damage. Canine susceptibility to *Rickettsia rickettsii* was demonstrated in 1933. Recent reports emphasize that, contrary to previous literature, untreated naturally-occurring RMSF can result in death. Clinical reports suggest that RMSF is a much more common cause of disease in dogs than was previously recognized.

2. Despite its name and original description as a disease of humans in the western United States, the majority of human cases of RMSF occur in the

southeastern US. Human cases of RMSF have been reported from nearly every state in the US, western Canada, Mexico, and South America. Distribution of the disease is related to the distribution of the vector ticks *Dermacentor variabilis*, the American dog tick found in the eastern US, and *Dermacentor andersoni*, the wood tick, which is the principal vector in the western US. Canine RMSF has been recognized in most southeastern states, New York, Massachusetts, and Ohio.

3. Rocky Mountain spotted fever is caused by *Rickettsia rickettsii*, a very small bacterium that must live inside the cells of its hosts. They are difficult to see in tissues by using routing histologic stains and generally require the use of special staining methods. Rocky Mountain spotted fever was first recognized in 1896 in the Snake River Valley of Idaho and was originally called "black measles" because of the characteristic rash. It was a dreaded and frequently fatal disease that affected hundreds of people in this area.

B. Transmission

R. rickettsii is a small intracellular parasite in the family Rickettsiaceae. The organism is a member of the spotted fever group rickettsiae, which includes both pathogenic and nonpathogenic rickettsiae. Dogs and rodents comprise the mammalian reservoir for *R. rickettsii*. Following tick bite, infection may occur in humans, dogs and cats. Within the general tick population, few ticks contain infective *R. rickettsii*. However, there are geographic centers that contain large numbers of infective ticks. Attachment of a tick to a host for five to 20 hours is required before infection can take place.

C. Signs of Disease

1. *R. rickettsii* is transmitted to the dog by a tick bite. The rickettsiae enter the circulatory system and replicate. Rickettsiae cause direct damage to cells lining the vascular system, resulting in vascular inflammation and death of the cells, swelling of the skin, hemorrhage, which if severe can cause low blood pressure, shock and death. Central nervous system swelling may contribute to the development of neurologic signs, rapid clinical deterioration, and death. Fluid accumulation in the lungs may occur, and may be detected with an X-ray. Clinical signs include rapid breathing, difficult breathing or coughing in some dogs. In severe cases, acute kidney failure may occur. Due to increased vascular permeability, fluid therapy should be used with caution, when treating dogs with Rocky Mountain spotted fever.

2. Some dogs develop mild illness following experimental and naturally occurring infection with *R. rickettsii*. In addition to the infective dose or strain variation in rickettsiae, breed predisposition may play a role in determining the severity of illness. For example, some have observed severe disease in Siberian Huskies, whereas Deerhounds sustain high antibody titers without prior evidence of associated illness.

3. Clinical signs in canine infection are identical to human cases of RMSF. Unlike Ehrlichiosis in which chronic infection can persist, the total duration of illness following *R. rickettsii* infection is generally short (two weeks or less). For this reason, canine RMSF is a disease that presents in the spring and summer (April to September). Fever, loss of appetite, depression, vomiting, diarrhea, and neurologic abnormalities are typically associated with the clinical presentation of

the animal. Redness and discharge from the eyes, nasal discharge and coughing are frequent findings. In some dogs, weight loss is very severe, considering the short duration of illness. Joint pain and/or muscle pain may represent the only or most prominent clinical finding.

4. Bloody nasal discharge, blood in stools, blood in the urine and areas of bruising occur in some dogs, but may not develop unless diagnosis and treatment are delayed for five or more days after the onset of clinical signs. Bleeding into the eye is a consistent finding, even early in the course of the disease. Scrotal swelling, hemorrhage, and testicular pain are frequently observed in mail dogs. This finding correlates with the disease in man and experimental infections in rodents.

5. Neurologic signs including pain, loss of balance, tilting of the head, stupor, seizures, and coma may occur in dogs with RMSF. Similar to Ehrlichiosis, this presentation can mimic canine distemper in the young dog.

D. Diagnosis

1. The marked variation in clinical presentation allows RMSF to mimic numerous other infectious and noninfectious diseases. Seasonal occurrence, history of tick infestation, fever, or the previously described clinical findings would suggest the possibility of RMSF.

2. Decreased platelets, generally mild in degree, are the most consistent finding in blood counts. Biochemical abnormalities reflect the effects of generalized vascular damage and vary with the severity and duration of infection. Low protein levels, elevated kidney function tests, and increased liver enzymes (serum alkaline phosphatase, alanine animotransferase) may occur in dogs with RMSF. In general biochemical abnormalities are mild. If joint swelling is present, inflammatory cells may be present.

3. Confirmation of a diagnosis requires either direct immunofluorescent testing for *R. rickettsii* antigen in tissue biopsies, or serologic testing utilizing an indirect fluorescent antibody test. Evaluation of acute and convalescent sera with greater than or equal to a fourfold increase in antibody titers confirms a diagnosis of RMSF. Timing of sample collection for acute and convalescent sera will greatly influence the serologic results. Cross reaction with other spotted fever group rickettsiae and persistent tick exposure to *R. rickettsii* complicates the interpretation of serologic results from clinical patients with suspected RMSF. Direct immunofluorescent testing of tissue biopsies provides the opportunity for rapid diagnosis of RMSF. *R. rickettsii* are generally more readily demonstrated in human patients in areas of hemorrhage prior to initiation of treatment. This also appears applicable to canine patients, although organisms may be more readily identifiable in clinically unaffected skin from dogs. If acute phase sera are obtained several days after the onset of clinical signs, antibody titer to *R. rickettsii* antigens will be high.

E. Treatment

Tetracycline or doxycycline is the treatment of choice. However, chloramphenicol and enrofloxacin are equally effective. A rapid clinical response occurs in dogs without neurologic signs following the initiation of treatment. If fever persists, another diagnosis should be considered likely. Delay in diagnosis

and initiation of tetracycline of the use of antibiotics lacking efficacy for treating rickettsial diseases may result in a fatal outcome. Due to sever vascular damage, fluid therapy should be utilizes with caution.

IV. Babesiosis

    A. Introduction

        1. Babesiosis is a tick-borne hemoprotozoan (blood) disease. The organism is called Babesia, the disease is called Babesiosis. There are two primary infecting species: *Babesia canis* and *Babesia gibsonii*. Babesia species can cause an acute hemolytic anemia in dogs. This is a low red blood cell count caused by the destruction of the red blood cells within the animal's body. *Babesia* has worldwide distribution. In a high percentage of dogs with acute or potentially chronic babesiosis, it will contribute to an immune-mediated hemolytic anemia, where the red blood cells are destroyed by the animal's own immune system. Babesiosis is a cyclical disease, similar to malaria. Dogs that recover from the initial infection show variable and unpredictable patent periods alternating with dormant periods.

        2. Historically, *Babesia gibsonii* was considered endemic in Asia, Africa and the Middle East, but was not recognized in the United States until 1979. Subsequently, *B. gibsonii* infection was recognized in dogs from California and most recently in Pit Bull Terriers in the southeastern US. In most instances, the presenting problem was immune-mediated hemolytic anemia. To date, a competent tick vector for the organism has not been identified in the US; therefore, establishment of the organism within the tick population may not be possible. As the efficacy of currently available drugs for treatment of *B. gibsonii* is limited, veterinarians should report any suspected cases to the State Veterinarian. Should *B. gibsonii* become established within a tick population in the US, the consequences could be serious.

    B. Transmission

        Babesiosis is thought to be transmitted by the vector tick *Thipicephalus sanguineus*, or brown dog tick, the same tick that transmits *Ehrlichia canis*. This has not been definitively demonstrated.

    C. Signs of Disease

        Acute phase is of short duration, and is where the dog is initially infected with the disease. If the dog does not die outright from the infection, then it moves on to the next phase. Subclinical phase can last months or years. It is characterized by a fine equilibrium between the parasite and the immune system of the host. This equilibrium can be disturbed by a number of things: environmental stress, additional diseases/infections (especially Ehrlichiosis), immunodeficiency, spleen removal, surgery, stress, hard work, immunosuppressive treatment, or use of corticosteroids. The dog may exhibit few clinical symptoms during this phase, beyond intermittent fever and loss of appetite. If the equilibrium is disturbed, the parasite will begin to slowly grow in number and the dog will move into the next phase. In the chronic phase, if the dog's system remains unable to clear the parasite, it enters this final phase. The most obvious initial signs to an owner are a cycle of: lethargy, loss of interest in food, and a gradual loss of body condition especially evident around the eyes and along the spine. Other symptoms are:

upper respiratory problems (coughing or labored breathing), vomiting, constipation, diarrhea, ulcerative stomatitis (sores in the mouth), edema (swelling), abdominal swelling (ascites), bleeding under the skin or a rash (purpura), low white blood cell count, clotting problems, joint swelling, back pain, seizures, weakness, increased liver enzyme, low platelet count, hyper reflective eyes, enlarged lymph nodes, enlarged spleen, septic shock, depression.

D. Diagnosis
1. A combination of a regenerative anemia, elevated bilirubin, low platelet count, elevated kidney values, elevated globulins and urinary "casts" are all common with Babesiosis.
2. Indirect fluorescent antibody tests are available and will demonstrate antibody titers against the organism.
3. Definitive diagnosis is based on organism demonstration in red blood cells.

E. Treatment
1. Until recently, there has not been an approved anti-babesia drug available in the United States for treatment of canine babesiosis. Imidocarb diproprionate (Imizol®, Schering-Plough Animal Health) is currently available for the treatment of babesiosis in dogs. In acute babesiosis, the therapeutic response is rapid, with increasing production of new red blood cell values documented within 12 to 24 hours. In Africa and other regions of the world, imidocarb diproprionate is considered efficacious for treatment of *E. canis*, as well as *B. canis* infections. Some reports of success using metronidazole or clindamycin have been reported.
2. Although multiple therapies are being studied, there is no definitive treatment established for *B. gibsonii* infections in dogs.

Preventive Care
V. Tick Control
A. Advancements in the products available to control fleas and ticks have revolutionized their prevention in veterinary medicine. In the past, we were recommending multiple products, from whole house flea bombs to topical powders, all containing chemicals less than ideal regarding exposure to our pets and to their owners. Now, more safe and effective products have been developed for use in dogs and cats.
B. Topical "spot-on" products
1. Frontline and Frontline Plus
A topical product which kills both fleas and ticks. The active ingredient is Fipronil. It is marketed to prevent fleas for up to three months, and prevent ticks for up to one month. Although not established, the label claims that ticks will die prior to beginning a blood meal. Frontline PLUS contains an insect growth regulator as well, which prevents eggs laid by fleas from developing into adults.
2. KillTix® (Bayer)
a. Label claims a single application will repel and kill ticks for up to four weeks. Apply KillTix® montly when ticks are a threat in your area. In more acute infestations, repeat applications can be made, but not more than every three weeks.
b. Can ONLY be used in dogs.

C. Tick Collars
   <u>Preventic® Collar</u>
        Active ingredient is amitraz. Label claims that it kills ticks for up to three
        months. Studies have shown similar efficacy between fipronil and
        amitraz.
D. Revolution® is not effective at preventing ticks.
E. Vaccination
Other than the Lyme disease vaccine discussed above, no other vaccines are available to
prevent infection.

How do I remove ticks from my dog?

If possible, use blunt forceps or tweezers. Place tips around the tick where it is attached to the
skin. Remove tick with a steady pull away from the skin. DO NOT JERK OR TWIST THE
TICK. Take care not to crush or puncture the body of the tick or to get any fluids form the tick
on you. After removing the tick, cleanse the area with mild soap and water and wash your hands
with soap and water. Remedies such as matches, petroleum jelly or nail polish do not cause ticks
to detach.

Other Resources

1. American College of Veterinary Preventive Medicine (ACVPM) www.acvpm.org
2. Centers for Disease Control and Prevention (CDC) www.cdc.gov

Basic Genetics for the Dog Breeder
Anne P. Lannon, DVM

Basic Principles of Genetics

One of the basic principles of genetics involves the difference between phenotype (i.e., a dog's physical makeup/appearance) and genotype (a dog's genetic makeup, which is passed on to the offspring). Dog breeders try to assess the genotype of a dog based on its phenotype. As we all know, the top winners are not always the best producers for just this reason. It is important to examine not only the dog you are interested in using but also its littermates, parents, and offspring, if possible. Try to determine what traits the dog possesses and how strong (prepotent) is the potential for transmission of these traits to its offspring.

Disorders that puppies are born with are referred to as congenital disorders. Remember that a genetic disorder is one that is determined by genes, whereas a congenital disorder is merely one that is present at birth and may or may not have a genetic basis. Some genetic disorders manifest themselves prior to birth and thus are both genetic and congenital. One example is renal dysplasia, a disease in which the kidneys do not fully develop. Other genetic disorders, such as primary lens luxation, as seen in some of the terrier breeds, may not be expressed until later in life. An example of a disorder that would be congenital but not genetic would be herpes virus, or puppies born with defects such as missing limbs because their dam received corticosteroids during pregnancy.

Fundamentally, genetic diseases are due to abnormalities in deoxyribonucleic acid (DNA), which is the material of which genes are made. Genes occur in pairs – one allele or member of the pair is inherited from the mother and one from the father. Abnormalities in genes, called mutations, can involve one or both members of a pair. A dog with different alleles in regard to a given characteristic (e.g., one mutant allele and one normal allele) is called a heterozygote. A dog with two identical alleles, whether mutant or normal, is called a homozygote.

Each genetic disease is inherited in a particular way. With a recessive pattern of inheritance, both alleles of the pair must be abnormal to produce the disease. Affected animals have inherited one mutant allele from each parent, both of which appear clinically (phenotypically) normal. Mendel taught us that when two of these "carriers" of the gene are bred, a certain percentage (25 percent) of their offspring will show the trait.

One example of a recessively inherited disease is cystinuria in the Newfoundland. Cystinuria results from a kidney disorder that allows cystine crystals, or "stones," to form in the urine. The stones can potentially block the urinary tract, especially in males due to their anatomy.

Another example of a recessively inherited disease is phosphofructokinase (PFK) deficiency in English Springer Spaniels. This disorder is caused by an enzyme deficiency that causes affected dogs to have diseased red blood cells and muscle cells. The hallmark of the disease is pigmenturia (dark urine), which commonly develops after strenuous exercise. Jaundice or anemia can also occur during these episodes. Fortunately, these episodes are rarely fatal, but care must be taken to avoid any stressful situations that could precipitate a crisis.

A second mode of inheritance is the dominant pattern, in which only one member of the gene pair needs to be mutant to produce the disease. In classic dominant inheritance, every affected dog in a pedigree has an affected parent, which also has an affected parent, and so on.

One example of a dominantly inherited disease is dermatomyositis, a disease of the skin and muscle seen in Collies and Shetland Sheepdogs. The syndrome is characterized by skin lesions such as vesicles (blisters), redness, ulcers, crusts, scales, and hair loss. These lesions are most commonly seen on the face, ears, and the bony areas of the feet and legs, as well as the tips of the tail. In very mild cases where there is no muscle involvement, the skin lesions may spontaneously resolve, or may even be missed by the owner, but if these dogs are bred they can produce the disease.

Another, less common mode of inheritance is X-linked, which refers to genes located on the X chromosome. In X-linked recessive diseases, the affected animals are usually male. They receive the mutant gene from their mothers, which are heterozygous carriers that do not have the disease. Two examples of X-linked diseases are Hemophilia A (seen in some lines of German Shepherd Dogs) and X-linked severe combined immunodeficiency (SCID), which has been seen in the Basset Hound and the Cardigan Welsh Corgi.

Sex limited inheritance is a mode of inheritance in which an autosomally transmitted trait (one not transmitted on the sex chromosomes) is expressed in only one sex. One example is persistent mullerian duct syndrome in the Miniature Schnauzer.

Finally, we see diseases that are inherited in a complex pattern not fitting any single gene mode. These defects result from the cumulative action of a number of different genes (polygenic) rather than from abnormalities in a single pair. Cleft palate, bite problems, and hip dysplasia are examples of defects inherited in this manner.

Avoiding Genetic Diseases

First, it is vitally important that you know what genetic diseases are a problem in your particular breed. Breed clubs are doing an excellent job of keeping abreast of health concerns in their respective breeds. Also, the American Kennel Club has created the Canine Health Foundation, which devotes significant resources to canine health research in the areas of genetics and breed-related health problems.

If you breed a litter and suspect a problem with one or more puppies, have your veterinarian examine the litter carefully. If he or she is unsure of what the problem is or whether it is a genetic problem, have your veterinarian refer you to someone who can help. There are universities in all areas of the country now that are working on genetic diseases in dogs.

Screening for Disorders

Your breeding stock should be screened for those genetic diseases for which tests exist. Hip and elbow dysplasias, eye disorders such as progressive retinal atrophy (PRA), heart defects, and

bleeding disorders are examples of genetic diseases for which routine screens are conducted in breeds shown to have these problems. Keep in mind that the age at which these defects can be detected varies by breed.

Screening for recessive genes is much more difficult. Several diseases (and I'm happy to say more all the time) have been identified at the biochemical or molecular level, and a laboratory test utilizing a few drops of blood or a few cells can be used to identify animals affected by and/or carrying the gene for these diseases.

We need to make a distinction between a direct DNA test and a linkage based DNA test. With a direct DNA test, the disease gene itself has been clearly identified and it is the presence or absence of that gene that is used to identify affected, carrier, and/or clear dogs. Linkage based tests measure the presence of microsatellites (large chunks of DNA consisting of repetitive sequences that stand out from the rest and serve as markers) that may be located <u>close</u> to the gene for a genetic disorder on a chromosome rather than identifying the gene itself.

One example of a direct DNA test available to detect a genetic disease is the test for PFK deficiency. This test was developed at the University of Pennsylvania by Drs. Urs Giger and Bruce Smith. The test is based on the fact that diseased (affected) dogs have two mutant PFK gene copies, carriers have one normal and one mutant PFK gene copy but are clinically normal, and normal dogs have 2 PFK genes with the correct code. The test requires only a few drops of blood from which the DNA is extracted and tested by the use of a polymerase chain reaction (PCR), a modern laboratory technique that results in bands showing up in the area of either the mutant allele, the normal allele, or in the case of carriers, both.

For recessive diseases (or traits) for which there is no laboratory test for carriers, test breeding remains the only way to identify carriers. Test breeding consists of breeding a dog that is suspected to be a carrier either to a dog that has the disease or to a proven carrier. If even one puppy is affected, then the dog in question is a confirmed carrier. However, if no puppies are affected, you still cannot be absolutely certain of the test dog's status – in any one litter the actual ratio of affected to normal animals may be very different from what was expected. The more normal puppies born, the greater the likelihood that the dog is normal and not a carrier.

There are several problems with test mating, including the fact that many genetic disorders are not recognized until the offspring are several years of age. Another problem is that test breeding may produce affected and/or carrier pups. Finally, in most breeding programs it is unrealistic to think that only noncarrier, normal animals can be used for breeding. In some breeds, this is virtually impossible. A more realistic approach would be to identify carriers so that if they are vitally important to a breeding program they can be bred only to noncarriers; the breeding program can then be continued using the normal noncarrier offspring from these crosses.

Pedigree Evaluation

Careful evaluation of pedigrees should also be done prior to breeding. There are three basic types of breeding programs one can undertake based on pedigrees:

The first type of breeding program is inbreeding. The general definition of inbreeding is the mating of two individuals that are related to each other through one or more common ancestors. The closer the relationship, the greater the degree of inbreeding.

The second type, linebreeding, is actually just a form of inbreeding that usually involves the breeding of a sire with more distantly related relatives than its daughters, sisters, or dam.

Finally, outcrossing is a third type of program that involves breeding two dogs with no common ancestors. This may be almost impossible in breeds with a limited foundation but can be used in breeds where foundation stock came from various sources. Although there is less chance of recessive genes being expressed, there also tends to be less uniformity in the litter. This could, however, be a way to introduce desirable traits that your stock is lacking.

Conclusion

Many people feel that genetic diseases are on the rise. Certainly more genetic diseases are recognized today because there are more sophisticated methods of diagnosis available. The development and institution of diagnostic tests, therapies, and preventive strategies for acquired diseases (such as infection, nutritional imbalances, and toxicities) have greatly reduced morbidity and mortality in puppies, hence the inherited diseases have become relatively more apparent.

The availability of genetic testing and counseling for breeders is also on the rise. Such services are expected to increase nationwide over the coming years as veterinarians, researchers, breeders, and breed organizations focus increasingly on the problems (and solutions!) to genetic defects in dogs.

I strongly feel that by working together to solve these problems we will continue to enjoy and extol the virtues of purebred dogs for many generations to come!

Recommended Readings

1. Ackerman L: The Genetic Connection: A Guide to Health Problems in Purebred Dogs. Lakewood, CO, AAHA Press, 1999.
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Websites of Interest

AKC Canine Health Foundation: www.akcchf.org

Canine Genome Project: www.fhcrc.org/science/dog_genome/dog.html

Cornell Genetics Course: www.ansci.cornell.edu/cat/cg01/dogcourses.html

Optigen, Inc.:   www.optigen.com

VetGen, LLC: www.vetgen.com

Neurologic Emergencies
R.M. Clemmons, DVM, PhD
University of Florida

The development of neurologic disease in dogs and cats can be stressful to the owner, since the clinical signs may be acute and severe. Some conditions do represent clear emergencies whereby immediate intervention can not only be life saving, but also can reduce the suffering of the pet and reduce the recovery time if treated decisively and swiftly. On the other hand, a number of neurologic problems look serious, but are not truly emergencies. Understanding when a neurologic problem is a true emergency as compared to when it is only something that should be tended to as soon as possible can be important. This distinction prevents delaying treatment, while not unduly costing the owner in needless emergency fees and procedures. Here are a few examples of true neurologic emergencies and some conditions that are not. Overall, seizures, coma, paralysis, vestibular disease and nervous system trauma are examples of conditions that may be true emergencies regardless of cause along with the important aspects of treatments.

Seizures

A reproducible change in behavior can be called a seizure. Most commonly, seizures are associated with an increase in voluntary motor activity, an alteration in consciousness, and an increase in involuntary motor behavior. Severe seizures result in what most people recognize as "grand mal" seizures when the animal is on its side and shows alternating extension and flexion of the limbs. A single, simple seizure that lasts less than two minutes is probably not of immediate concern. Contact with your veterinary team in a timely manner is indicated and discussion about what should be done for diagnosis and treatment. On the other hand, more than three seizures in an hour or a single seizure that lasts more than five minutes probably constitutes a true neurologic emergency. This may be called status epilepticus, a special case where seizures occur one after another without abatement. If these seizures are not stopped, the resultant hypoxia may result in irreparable brain damage. The goal, then, is to stop the seizures. This requires injectable medications, including diazepam, pentobarbital and Phenobarbital. Although pentobarbital is not an anticonvulsant, it can sedate the patient long enough for phenobarbital to work. Give diazepam (to effect) at 0.5-1.5 mg/kg IV. If the seizures continue, repeat and give both pentobarbital and phenobarbital at 2 mg/kg IV. Remember to check the glucose level and give if low and consider calcium glucconate if the seizures don't seem to be controlled. Once the seizures are under control, the patient should be examined to determine the cause of the seizures and anticonvulsant therapy with phenobarbital and/or KBr continued while achieving the diagnosis. Since there may have been hypoxia from the seizures, use of antioxidant steroids (Solu Medral or Solu Delta Cortef) may be given at 15-30 mg/kg and repeated every five to eight hours as needed for 24-48 hours. In most referral centers, status epilepticus is now treated with Constant Rate Infusions (CRI) of diazepam at 0.25-0.5 mg/kg/hour. Once the seizures are controlled, the rate is reduced and oral medication with maintenance anticonvulsants is begun. Caution with CRI of diazepam is that it should be given separately from fluid infusion and by a central intravenous line that reduces the risk of peripheral vasculitis from the diazepam. After the emergency is controlled, then diagnostic tests can be performed to find the underlying disease process that led to the seizures.

Flow Chart for Treatment of Status Epilepticus:

- Give IV diazepam 0.25-0.5 mg/kg
- If continues:
    - Give IV pentobarbital 2 mg/kg and IV phenobarbital 2 mg/kg
    - *OR* Start CRI IV diazepam .025 - .05 mg/kg/hour
- Start maintenance anticonvulsants

Coma

Acute loss of consciousness (coma) or onset of stupor (almost complete unconsciousness but arousable with vigorous stimuli) is usually an emergency. Initial evaluation should take care of the "ABC's" of emergency medicine: make sure there is an Airway, support Breathing and assist Cardiac function as needed. Once this has been evaluated, consideration should be give to the location and cause of the problem. Cerebral diseases usually have normal to small pupils, Cheynes-Stokes respiration (crescendo-decrescendo breathing pattern) and a slow heart rate. Mid-brain diseases have fixed pupils that are midrange or dilated, hyperventilation and rapid heart rate. Caudal brain stem lesions have myotic pupils and irregular respiration and heart rate. The common causes include head trauma, intoxication (organophosphate, ethylene glycol, etc.), hypoglycemia, hepatic encephalopathy, meningoencephalitis, hydrocephalus, neoplasia and cerebrovascular disease. So, treatment may depend upon finding the causative factor and developing an appropriate treatment plan. Initially, provide support with oxygen therapy if blood gasses indicate problem or to potentially reduce cerebral edema. Give methylprednisolone (Solu Medral) 30 mg/kg. If meningitis is suspected, consider support with IV sulfadimethozine at 15 mg/kg every twelve hours and cefazolin at 22 mg/kg every eight hours. If cerebral edema is suspected give IV mannitol at 0.25 – 2 gm/kg over 10 – 15 minutes followed in 15 minutes by furosemide at 0.5 – 1 mg/kg. This can be repeated every four hours, if needed. Once the patient is stable, referral to a center where CT scan or MRI, CSF analysis and 24-hour tertiary care can be provided.

Paralysis

Acute loss of function of two or four legs resulting in paralysis is a common emergency situation. The "dynamic factor" (how quickly the spinal cord is damaged and with what force creates the damage) dictates the severity of clinical signs. The amount of traumatic force imparted by a small amount of material traveling rapidly is greater than a larger amount going slowly. In the worst case, this means that time for intervention is also quite short. In most cases of paralysis, definitive treatment must be started before 24 hours in order to achieve the greatest success. In some cases, this time is shorter and treatment may need to be started within four hours of the initial injury. Unfortunately, delaying treatment to see the outcome may preclude success. We treat severe paralysis as a medical and surgical emergency. In patients with complete motor and sensory paralysis, the patient should be treated for acute spinal injury and be immediately referred to a center that can diagnose and definitively treat the problem. In patients

who are paralyzed but retain deep pain, then it is possible to treat them for acute spinal injury and observe them for signs of improvement. If they are worse or no better within 24 hours, they then constitute an emergency referral. On the other hand, it is best to treat these patients as true emergencies at the outset. In patients with mild paresis or mere back pain, they can be worked-up for the rule/out and referred if they do not make improvements in five to seven days. These later patients may benefit from surgical intervention, but may not represent immediate emergencies, as do patients with paralysis. In general, paralysis of a single limb is not an emergency if the patient is stable, but may need methodic work-up during the first few days to weeks.

Initial medical management of acute paralysis consists of giving 30 mg/kg of methylprednisolone (Solu Medral or Solu Delta Cortef) IV, initially; followed by 15 mg/kg every eight hours for the first 24 – 48 hours. Also during this time, referral can be arranged and initial diagnostics performed. The patient is stabilized and referred to a facility capable of emergency surgery. There is probably no rational reason to give these patients dexamethasone since the antioxidant action of the short-acting methylprednisolone is superior in the eventual outcome, a fact established over the last decade. There is also not place for the use of NSAID (non-steroidal anti-inflammatory drugs) medication in acute spinal injury. With rapid surgical intervention, patients who are paralyzed with deep pain have a 90 percent chance of significant recovery if treated in the first 24 hours. In dogs with no deep pain, the chances for recovery are less and probably around 75 percent in the first 24 hours. In the next 48 hours, these dogs have a 50 percent chance of recovery; while, after 72 hours, their chances fall to around 25 percent (which is essentially the same as without surgery).

Vestibular Disease

All cranial nerves have the potential to develop specific syndromes that are clinically classified as idiopathic disorders. This is probably due to the fact that each cranial nerve represents a unique developmental anatomy from their respective brachial arches. This also gives them a unique antigenic signal allowing very specific immune attack upon them. Idiopathic vestibular disease represents one of these cranial nerve syndromes.

Clinically, idiopathic vestibular disease presents as an acute onset of vestibular signs with severe imbalance, due to its sudden onset and the severe nystagmus, which is associated with the onset of the disorder. Since the eyes are unable to fix on the horizon and the vestibular mechanism is defective, there is severe vertigo. This often results in the rolling described by the owners. This can be mistaken for a seizure, which it is not. During the early phases of idiopathic vestibular disease, the patient often experiences nausea to the point of frequent vomiting and inappetence. The head tilt will be toward the side of dysfunction and the nystagmus will be horizontal or rotatory with the fast-phase away from the head tilt. If supported, there are no other neurologic deficits and proprioception is normal.

The diagnosis of idiopathic vestibular disease is tentatively made by the presence of acute clinical signs in the absence of other physical findings. The minimum database includes physical examination, otoscopic examination and neurologic examination. The lack of findings (other than the peripheral vestibular signs) supports the diagnosis. The signs of idiopathic

vestibular disease are regressive, meaning that they disappear without treatment over time. As such, the fact that the disease is self-limiting is how the final diagnosis is achieved. The nystagmus will usually improve or disappear all together within three to five days of the onset. The patient will, then, improve in their imbalance and be more able to function normally. This improvement will continue until minimal deficits will remain. It is possible that there will be a residual head tilt. If the head tilt persists beyond six months following the onset of signs, it is likely to be permanent.

There is no treatment that will hasten the recovery from idiopathic vestibular disease. Corticosteroids probably do not offer an effective treatment. On the other hand, since idiopathic vestibular disease may represent an immune disease, anti-oxidant steroids (such as Solu Medral) may decrease severe symptoms. During the early phases, anti-vertigo drugs might make the patient more comfortable. Generally, I use dephenhydramine at 2 – 4 mg/kg every eight hours as needed. Diphenhydramine is a centrally active anticholinergic, antihistamine that helps reduce vertigo and nausea. Assuming that the regressive course becomes evident, then I monitor the patient periodically for the signs of continued improvement.

Anecdotal evidence suggests that idiopathic vestibular disease may represent toxicity to eating certain strains of lizards. Owners often notice the cat with a lizard in its mouth just prior to the onset of clinical signs. However, experimental feeding of the suggested lizard species to cats does not lead to the disease. It is still possible that laboratory conditions do not mimic field conditions. On the other hand, idiopathic vestibular disease occurs in many animals and in animal species where exposure to lizards plays no role in the condition. It is most likely that idiopathic vestibular disease is an immune-related condition affecting the unique antigens presented by the vestibular nerve. It can recur and is often more severe on recurrence.

So, while acute idiopathic vestibular disease is a common problem and frightens the owners, it represents (generally) a good disease that will go away on its own. On the other hand, since the signs are so severe, it usually is "treated" as an emergency if only to make the owner feel better. If the vertigo can be reduced, the patients are more comfortable. That is the actual goal of initial assessment. If the signs do not regress, then more aggressive diagnostics can be done.

Trauma

Central nervous system trauma is a common cause of neurologic emergency and may lead to stupor or coma or paralysis as discussed above. Fractures should be stabilized. Pain should be controlled. Shock and fluid disturbances should be prevented and normal tissue perfusion should be maintained. IV methylprednisolone (Solu Medral) should be given at 30 mg/kg to protect the nervous system and to prevent further neurologic damage. In cranial injuries, IV mannitol (0.25 – 2 gm/kg) followed by furosemide (0.5 – 1 mg/kg) should be given. With cranial trauma, sedative drugs should be avoided so that levels of consciousness can be monitored. Once the patient is stable, referral for appropriate diagnostics and surgical corrections should be done. Remember that trauma often affects other systems as well as the nervous system. On the other hand, waiting for those systems to heal before treating the neural injuries will probably decrease the chances for neural repair. The neural injuries may take precedence, treating the other injuries at the same time.

Advanced Reproduction Symposium
Ann Lannon, DVM

Artificial Insemination from the "Male Perspective"

Some of the advantages of artificial insemination include the ability to breed to stud dogs no matter where they are located. Obviously we are referring to fresh cooled or "chilled" semen or frozen semen. Certainly, if the dog is local, a natural or fresh semen breeding is easily achieved. Also many popular stud dogs are not always readily available due to being on show/trial circuits, or if he is a heavily used stud dog the choice of frozen semen may be the only option. A huge advantage of using chilled or frozen semen is elimination of the need to transport the bitch, especially distances that would involve air travel. It is becoming increasingly difficult to find inexpensive ways to ship these days, and the stress (to owner and bitch!) is decreased.

Other indications for doing an artificial breeding would include a temperament problem such as a very sensitive, shy young stud and/or a non-receptive bitch. Structural problems in some breeds may preclude a natural breeding. For example, some standards call for very straight stifles which may make mounting difficult, or some of the giant breeds are so heavy that it is difficult for the bitch to support the male. Also, a structural problem due to trauma may make it necessary to do an artificial breeding.

I think a big indication for A.I. for many breeders is fear of infection, and I think it is very important to stress that neither the sheath/penis of the male or the vagina of the female are sterile. If you are getting back totally negative cultures, something is wrong with the culture! The normal flora of the vagina contains a huge cross section of normal bacteria, including the dreaded mycoplasma. Also in true cases of a severe sheath infection or vaginal infection, breeding should probably be delayed anyway until the infection is treated.

Finally, an artificial insemination is relatively quick (other than surgical insemination of frozen semen) – we've all experienced those hour-long ties and you wonder of your back and legs will give out as you assist!

There are also disadvantages to artificial insemination. First of all, your insemination timing becomes critical with chilled or frozen semen breeding. If you have the dog and bitch available and are doing either natural breedings or fresh semen A.I., you can pretty much cover the cycle starting with either the first day of an estrus vaginal cytology slide or when the bitch accepts the male. With chilled or frozen semen, it is critical to time ovulation, utilizing more extensive testing. In these cases, costs can really go up. Costs include semen collection and preparation, semen shipment, and for the bitch, veterinary exams, vaginal cytology/vaginoscopy, and serum progesterone and/or LH testing to determine optimum breeding time.

In addition, in some cases, depending on the skill of those involved, you may see decreased conception rates and litter sizes with artificial insemination.

My feeling is that if you have the dog and bitch available and there are no problems precluding a natural breeding, then that is the way to go. At Guide Dogs for the Blind I managed a breeding

colony of Labradors, Golden Retrievers and German Shepherds. We averaged ten litters a month to meet the needs of the training program for our blind clientele. We had 150 bitches and 50 stud dogs who lived in family homes in the community and went everywhere with their families. They would come in for breeding as needed, and we did almost exclusively natural breedings. We had a conception rate of 97 percent and an average litter size of nine! Certainly there are times when artificial breeding is needed and it's great that our technology has improved so much in these areas, but let's not forget about natural breeding.

The use of fresh cooled or chilled semen has some additional AKC regulations involved, so let's touch on those. First of all, the semen must be collected and extended by a licensed veterinarian. In addition, the insemination of the bitch must be performed by a licensed veterinarian in order for the resulting litter to be registerable. The certification form for a chilled semen breeding contains certifications from the owner, co-owner, or lessee of the dam on the date of mating, owner or co-owner of the sire on the date of mating, the veterinarian collecting and extending the semen, and the veterinarian inseminating the bitch. Nothing like a little paperwork!

We discussed the collection of fresh semen in the article concerning the breeding soundness exam. Obviously, it is vitally important that a complete semen evaluation be completed after collection of semen that is going to be used for a chilled breeding. Assessment of motility, concentration, volume, and morphology should be completed and it is important that the semen be normal in all respects to optimize results.

The collected semen (collected in the same manner as for a fresh semen breeding or semen evaluation) is evaluated and then immediately mixed with an extender. The extender prolongs the life span of the sperm and is either skim milk based or egg yolk based. Commercial extenders are available from companies such as Synbiotics but recent studies have shown that milk based equine extenders are also suitable (and much cheaper!). The dilution of semen to extender is normal 1:1 to 1:3, depending on the amount of the semen. Remember that we want to utilize only the first and second fractions of the ejaculate for a volume of 2-3 cc's max. If more than that is collected the ejaculate should be centrifuged and then extended.

Packaging can utilize commercial canine or equine systems but any container that will maintain a temperature of four degrees (refrigerator temperature) will suffice. Examples are thermos bottles of Styrofoam boxes containing cold packs. Obviously the semen must be shipped to arrive to the bitch at the proper time for inseminations (ideally two days after ovulation or four days after the LH peak). Semen is generally viable for up to 48 hours (24-48 hours) once packaged for shipment; therefore overnight shipping is used most commonly. Some things to keep in mind regarding shipping: check whether the company will deliver and/or pick up on Saturdays. If the company won't pick up on Saturday then you won't be able to do a Sunday or Monday insemination. Airline "counter to counter" service may need to be utilized if weekend service is necessary. An official AKC litter registration application form should be included with the shipment with the appropriate information filled out and the signature of the veterinarian who collected and extended the semen. It is also a good idea to have the results from the initial semen analysis included.

On the bitch's end, the veterinarian should always check the motility of the semen after it has been warmed and prior to insemination. If there has been a problem during shipment and the sperm are all dead, the stud owner will need to be contacted as soon as possible!

As far as timing the inseminations it is recommended that there be two inseminations either three and five days or four and six days after the LH peak (which is most commonly estimated utilizing progesterone kits). If only one insemination is possible it is recommended that it be four days after the estimated LH peak.

The chilled semen should be warmed for a minute or so in a warm water bath, after which the semen is deposited vaginally at the cervix. Sterile insemination pipettes are attached to plastic syringes for insemination. The pipette is inserted at the top of the vulva, first vertically and then horizontally to follow the normal anatomy of the bitch to the level of the cervix. Following insemination some people recommend "feathering" or tickling the dorsal part of the vagina with the inseminator's finger and elevation of the bitch's hindquarters to maximize the insemination. Keep in mind that research has shown that elevation for more than one minute was no more useful that five, ten or fifteen minutes, so keep it short!

Communication between all involved parties is the most important aspect of utilizing fresh chilled semen successfully!

Conception rates with chilled semen A.I. depend on appropriate timing, appropriate handling of semen during collection, extension, and shipment, and correct semen placement. Conception rates are estimated at 59 – 80 percent depending on these factors.

Frozen semen breedings present their own set of challenges, and truly timing is everything! Timing is even more critical when you are dealing with frozen semen due to the short life span of semen post-thaw. If the bitch is to be inseminated surgically and hence only one insemination will be possible, we recommend that the inseminations be done three to four days post ovulation or five to six days after the LH peak. It is recommended that both LH and progesterone testing be done to insure insemination at the proper time. With trans-cervical insemination two inseminations can be done, on days three and five or four and six post LH peak.

The freezing process begins with collection of the sperm-rich fraction and extension but the process is much more involved than with chilled semen. Basically, the process involves centrifugation in order to decrease the number of straws needed, and then the semen is extended for the first time at a 1:1 ratio and refrigerated for one hour. Then a second extender is added over a 30-minute period. Finally, the straws are filled, sealed, and refrigerated for about two hours before being placed on a freezing rack suspended above liquid nitrogen for six minutes before finally being placed directly into the liquid nitrogen for storage. Quite a process! Semen that has been frozen in liquid nitrogen is good indefinitely. It is always a good idea to thaw a straw after freezing to ascertain how well this particular dog's semen freezes.

Usually frozen semen is shipped in a vapor shipper, also know as a dewar. The shipper does not contain any free liquid nitrogen but the vapor is enough to keep the straws at the appropriate temperature during shipping.

Prior to use, the straws are thawed in a 37 degree centigrade water bath for 60 seconds. It is important that the veterinarian evaluate post-thaw motility before insemination. Thawed sperm have a maximum life span of 12 – 24 hours after insemination into the bitch.

There are two methods of insemination of frozen semen. The first is surgical, and a laparotomy incision is made while the bitch is under general anesthesia, the same as if she were going to be spayed. The semen is drawn into a syringe through an insemination rod and a 22-gauge needle is utilized to inject the semen directly into the uterine body or horns.

The disadvantages of this approach would be the risks of general anesthesia and the stress to the bitch; however, there are many who have great success with this approach.

The other method is trans-cervical insemination, where semen is deposited into the uterus in an awake and standing bitch utilizing one of two catheterization methods. The Norwegian method utilizes a long steel catheter with an outer protective nylon sheath. The inseminator palpates the cervix and the tip of the catheter is inserted vaginally and then through the cervix into the uterus. This method has not been utilized much in this country as the cervix is very difficult to palpate abdominally and hence there is a steep learning curve – perhaps those Norwegians have especially sensitive fingers!

In this country, the New Zealand method first introduced by Dr. Marion Wilson is gaining growing favor and is the method I was taught while I was at Guide Dogs for the Blind. This method utilizes a vaginal fiber optic endoscope (actually a cystoscope commonly used to examine the bladder) to visualize the cervix and then a plastic urinary catheter is inserted via the endoscope and the semen is deposited through the catheter by attaching the syringe with the thawed semen to the end of the catheter.

Conception rates vary considerably for frozen semen due to the many variables, and have been estimated to be anywhere for 11 – 80 percent.

The Canine Male Breeding Soundness Exam
Anne P. Lannon, DVM

There are many reasons why a dog may be presented to a veterinarian for a breeding soundness exam. This may be a young, unproven dog that the owner wants to be assured is fertile prior to introducing him at stud. Perhaps it is an older proven dog that has recently had a few "misses." Alternatively, this could just be a serial examination of a popular stud dog. It is a great idea for these popular males to have an exam every six months or so and then the owner can present the results as proof of his fertility.

It is important to consider the age and breed of the dog presented for several reasons. First, a very young dog with fertility issues brings congenital defects into the picture. On the other hand, an older dog may have other health issues to deal with. Plus it is important to realize that the AKC requires that a semen evaluation and breeding soundness exam be performed by a veterinarian for dogs under seven months or older than twelve years of age who have sired a litter. In addition, breed related health or genetic issues may play a part in a fertility problem, and certainly different breeds mature at different rates.

A complete breeding history is very important. If the dog is proven, we want to be sure to check on the bitches they have been bred to and ascertain their reproductive history before incriminating the male. Who did the breedings, how were the breedings performed, and how often were they bred? How often has the dog in question been used and what was the environment? Medical history and any medications being used must be known, as well as the status of the last brucellosis test and when it was done. Genetic history may also be important – any history of infertility in the family?

A complete physical examination is indicated for all dogs, starting with body condition and body weight. Eyes, ears, nose and throat, heart and lungs, abdomen, musculoskeletal, coat, and skin are all examined for signs of abnormalities. Special attention is paid to skin conditions that may indicate an underlying endocrine disease that may have an effect on the reproductive system.

Special attention is, of course, paid to the reproductive tract. First the prepuce is examined for tumors, abrasions, or abnormal discharge. The normal prepuce contains a small amount of yellow-green discharge but it should not be profuse (which would indicate a condition known as balanoposthitis) or bloody. The penis should be easy to extrude from the prepuce. Pain or failure of the prepuce to be moved may signal an anatomic abnormality such as persistent frenulum or preputial stenosis. The non-erect penis should be light pink and examined for abnormalities such as tumors or signs of trauma. The scrotum should be examined for thickening, lesions, tumors, or dermatitis (skin disease). Scrotal dermatitis can be very painful and can cause the dog to severely self-traumatize the area by licking and chewing. This is often caused by harsh cleaning substances used on runs. The testicles are palpated carefully for size, which should correlate to the size of the dog, symmetry (they should be symmetrical), consistency (firm but not hard), and pain. Soft, small testicles may signal testicular atrophy, whereas enlarged, painful testicles may indicate orchitis, or an inflammation or infection of the testicles. The epididymes are palpated as the testicles are palpated and any swelling or asymmetry is noted. Last, but certainly not least, the prostate should be palpated digitally via the

rectum to determine size, consistency and evidence of pain. Prostatitis is a common problem and can be due to many causes.

The semen collection is sometimes done prior to the examination of the reproductive tract, especially with a sensitive dog. It is recommended that there be five to ten days of sexual rest prior to collection. If the dog has never been used or it has been a long interval since the dog has been bred or collected we will usually recommend collection of a second ejaculate because you may see an increase in detached heads and distal droplets (both are secondary defects of sperm) due to prolonged storage in the epididymis.

Collection equipment consists of a latex or plastic cone (artificial vagina), plastic collection tube, and a small amount of water soluble, sterile lubricating jelly. Obviously many of us have had successful collections with much simpler equipment, i.e., manual stimulation and a Dixie cup!

We want the dog to be as relaxed as possible, and footing should be secure. In a veterinary clinic it is optimal to cover the linoleum flooring with rubber nonskid mats. A bitch in estrus is essential to get an optimum, complete ejaculation. If an estrus bitch is not available you can utilize the vaginal secretions of an estrus bitch that have been collected onto gauze pads and frozen. When needed, these pads can then be thawed and given to the stud dog to smell or spread on the vulva of an anestrus teaser bitch.

Manual stimulation is utilized first to create an erection and then, once strong thrusting has begun, the prepuce is slipped behind the bulbus glandis and pressure is applied behind the bulbus to simulate the "tie."

When the dog ejaculates, there are three fractions to the ejaculate. The first fraction is the clear pre-sperm fluid. The second fraction, the sperm rich fluid, is easily identified because of its milky appearance. These first two fractions are usually produced during rapid thrusting by the male and are usually collected together. Normally the first two fractions will result in 1 – 3 ml of fluid. The third fraction mainly consists of prostatic secretions and is usually collected separately for analysis of prostate abnormalities. The semen should be evaluated immediately after collection.

The semen evaluation consists of evaluating color and volume, motility, concentration and morphology of the semen/sperm cells. Color and volume can be easily assessed immediately. The color of the semen should be milky white. A yellow or reddish color suggests contamination with urine or blood. The volume is easily estimated if a graduated cylinder was used for collection. If you have over 3 ml of volume you have included prostatic fluid in the collection.

Motility (sperm movement) is estimated immediately because sperm slow down as they get cool. A drop of semen is placed on a clean glass slide and examined under the microscope. The percentage of sperm swimming rapidly forward is then estimated. Samples containing greater than 70 – 80 percent of progressively motile sperm are considered to be normal.

The total sperm count is determined by multiplying the volume by the sperm concentration. Sperm concentration is determined by utilizing a counting chamber called a hemocytometer. First the cells are diluted, and then they are placed in the hemocytometer and counted microscopically. Normal dogs should have greater than 200 million sperm per ejaculate.

Morphology, or the structure of the sperm, is evaluated by preparing smears of the semen and then staining them with special stains. One hundred sperm are observed both for primary abnormalities, which are more serious (examples are proximal droplets, double heads, or double tails) or secondary abnormalities such as bent tails, distal droplets, or separated heads or tails. It is generally felt that 80 percent of sperm in an ejaculate should be morphologically normal.

Infertility in the Stud Dog
Anne P. Lannon, DVM

It is important to realize that ejaculates can vary tremendously on a daily basis depending upon use of the dog. Therefore, several collections should be taken on separate days before a semen abnormality is diagnosed. Frequent ejaculation may decrease sperm counts, which is why it is recommended that the dog be rested for at least 48 hours prior to semen evaluation. Also, a dog that is inexperienced or frightened during the collection procedure may give an incomplete ejaculate, and collection on a different day under different conditions may yield a better result.

Once a representative sample is obtained, the first piece of information to be derived is whether sperm are present or absent in the ejaculate, what are their numbers, and are they normal in appearance.

Aspermia is a complete lack of sperm in the semen sample because the dog failed to ejaculate. Aspermia can have several causes. First of all, an inexperienced dog may fail to ejaculate due to nervousness, or a very young dog of a late maturing breed may not have reach sexual maturity and so physically is incapable of ejaculation. Pain, a second cause of aspermia, may be caused by an infected prostate, joint pain due to arthritis, or an injury. Psychological factors may include a very subordinate or passive male, or a more experienced dog that has a strong preference for certain surroundings. There are some drugs such as amitriptyline, which are sometimes used for behavioral disorders, and naproxin, which is used as a non-steroidal anti-inflammatory medication, which have been reported to cause ejaculatory dysfunction. Finally, retrograde ejaculation is an example of a neurologic dysfunction that causes the dog to ejaculate backwards into the bladder instead of through the reproductive tract. This is caused by blockage of nervous system receptors in the bladder neck, and is easily diagnosed by obtaining a urine sample by systocentesis immediately after semen collection.

In contrast to aspermia, azoospermia involves normal ejaculation of seminal fluid, but the fluid does not contain sperm cells. Azoospermia must be differentiated from an incomplete ejaculation where the dog only ejaculates the pre-sperm fraction of semen. This can happen with a young, inexperience, or very nervous dog. We can differentiate the two by measuring a substance called alkaline phosphatase in the collected fluid. Alkaline phosphatase is found in very high levels in epididymal fluid, therefore if the alk phos level is very high, then the fluid collected is from the epididymis and the dog did have a complete ejaculation.

Causes of azoospermia can be divided into pre-testicular, testicular, and post-testicular causes. Pre-testicular causes of azoospermia would be non-reproductive tract diseases or physical problems that affect sperm production. Examples would be endocrine diseases such as Cushing's Disease, pituitary problems, or hypothyroidism. Inguinal or scrotal hernias are physical abnormalities that can affect sperm production. Finally, a high fever can definitely kill sperm and shut down sperm production (as can high external temperatures for that matter).

Disorders involving the testicle are common causes of azoospermia and hence infertility. These disorders include intersex states, such as female pseudohermaphrodites that have male external

genitalia and female gonads. These problems are uncommon and are usually caused by administration of androgens (testosterone producing substances) to the dam. There is also a phenomenon of XX sex reversal which is inherited as a recessive trait in the American Cocker Spaniel, Kerry Blue Terrier, German Shorthaired Pointer, and a few other breeds. These dogs have male external genitalia and testicular and/or ovarian gonadal tissue but have two X chromosomes.

Traumatic testicular injury can certainly cause azoospermia, as can a disease called autoimmune orchitis, where the dog is producing antibodies against his own testicular tissue and sperm cells.

Testicular hypoplasia and degeneration refers to testicles that become shrunken due to any disease process or disorder. Sperm production decreases and finally fails as cells die and degenerate.

Finally, testicular cancer or neoplasia may also be a cause of azoospermia by direct destruction of testicular tissue by causing inflammation and elevation of testicular temperature, or by producing hormones that have a negative influence on sperm production.

Post-testicular causes of azoospermia in the dog are those causing outflow obstruction of sperm from the reproductive tract and include epididymal segmental aplasia, which just means that there is a portion of the epididymis that was either never formed during development or is no longer present due to trauma. A spermatocele can also cause obstruction and is an area of swelling in the epididymis due to a blockage that will not allow the semen to continue through the tract. This results in a sperm granuloma, which presents as a firm, non-painful swelling in the epididymis. Testicular size is normal with these problems, and they may be unilateral or bilateral, but if they affect only one side the dog may still be fertile.

A more common presentation than azoospermia is oligospermia, which means that there are sperm cells in the ejaculate but they are reduced in number from what would be considered normal. Any of the causes of azoospermia that we have previously discussed may cause a decrease in sperm numbers as opposed to a total absence of sperm, especially if the problem is diagnosed in its earlier stages.

A decrease in sperm numbers is a common sign of inflammation and/or infection in the reproductive tract such as prostatitis due to benign prostatic hypertrophy, prostate infection/abscess, or even prostatic cancer. Infection or inflammation in the testicles is referred to as orchitis and may be caused by bacterial infections and, in the epididymis, by brucellosis. Autoimmune orchitis is also a common problem, and we previously talked about high temperatures, or hyperthermia, causing decreased sperm numbers.

Asthenospermia refers to reduced motility in the ejaculate. This is most commonly due to collection and handling procedures such as too much water soluble lubricant, exposure to latex, or allowing the semen to become too cool. However, reduced motility can also be caused by testicular tumors or infections in the reproductive tract. In very rare cases it may be caused by immotile cilia syndrome, which is a congenital abnormality causing absent or irregular, asynchronous motility of all ciliated cells in the body. Heritability of this syndrome is unknown

but breeds in which affected littermates have been reported include the Bichon Frise, Springer Spaniel and Old English Sheepdog.

A few other, less common, abnormalities include teratospermia, which is when we see increased morphological or structural problems with the sperm cells themselves. These may be secondary abnormalities that may still allow normal fertility but usually indicate a problem in storage or transport, or primary abnormalities which are more serious and usually indicate a problem with sperm production. Hematospermia, or blood in the semen, may be seen with benign prostatic hypertrophy, penile lacerations, or infection/inflammation of the testes or epididymis. Finally, pyospermia, or pus in the semen, may be seen with prostatic, epididymal, or testicular infections, or may also be contamination from a sheath infection.

So now we have discussed all of the different abnormalities that may be noted when evaluating semen, and we may or may not have a good idea of what may be causing the problem. We may need to do some diagnostics so that we can figure out what the problem is. We should start out testing with a few basics, the first being brucellosis testing if it has not been performed recently. Secondly, we need to rule out any underlying systemic problems by doing a CBC (complete blood count) and chem. screen. The CBC may give us a clue to infection of inflammation if the white blood cell count is high, or it may clue us in to anemia if the red blood cell count is low, or even parasites if there is a high count of cells called eosinophils. We should also include a heartworm screen if this has not been performed recently. The chem. screen tells us about kidney function, liver function, blood sugar level, and other internal systems. Thyroid testing is a must if there is a history of skin disease, unexplained weight gain, and/or lethargy. Hypothyroidism does not just cause reproductive problems and is very unusual in young dogs. Finally, it is important not just to do a T4 level, as this is the total amount and may not indicate the amount of thyroid hormone available for your dog to use. Free T4 is a better measure of that and is included in most thyroid panels.

The sex hormones FSH, LH and testosterone are all necessary for normal sperm production, so it stands to reason that these would be great things to measure to ascertain the reproductive health of our dogs. Unfortunately, it isn't easy!

Observation of normal libido (sex drive) gives a rough estimation of normal testosterone levels. If libido is low you can measure testosterone levels but you must realize that testosterone is released in a pulsatile manner, therefore a single random sample is not diagnostic. You would have to do three blood samples at 30-minute intervals, and it is still rarely of diagnostic value in evaluating sperm production as levels remain in the normal range in dogs with a wide variation in the health of the tubules in the testicles that produce testosterone.

Canine specific assays of LH and FSH are not readily available commercially and hence are rarely of help in ascertaining a problem.

Ultrasound is an excellent tool to use to diagnose prostate or testicular problems. Neoplasia (cancer), cysts, and abscesses can all be diagnosed via ultrasound, and an ultrasound-guided biopsy can be done. The epididymes can also be ultrasounded to check for the presence of a spermatocele or sperm granuloma.

Cytology of both the sperm-rich and prostatic fractions of the semen can be evaluated by just putting a drop on a slide, and then preparing a thin film using another slide, allowing it to dry, staining it, and looking under the microscope. A prostatic wash is another way to collect prostatic fluid if a dog is in too much pain to collect a semen sample. A urinary catheter is advanced to the level of the prostate and is flushed with sterile saline and the fluid aspirated with a syringe for analysis. If bacteria or other abnormal cells are seen a sample should be submitted to the laboratory for culture and sensitivity. I recommend culturing for aerobic bacteria, anaerobic bacteria, and mycoplasma.

We may also want to check the alkaline phosphatase level of the semen sample. This is the test we mentioned earlier that can be used to differentiate azoospermia, or no sperm in the ejaculate, from an incomplete ejaculation, from an incomplete ejaculation. We simply submit a semen sample to the lab to ascertain the level of this enzyme, which is found in very high levels in the epididymis. It can also be sued to differentiate an obstructive problem causing azoospermia from a degenerative process.

A urinalysis should be performed to ascertain kidney function and evaluate for a possible urinary tract infection. The sample should be obtained via a cystocentesis (needle introduced into the bladder) to avoid contamination from elsewhere in the tract. The urine is evaluated for the presence of WBCs, RBCs, bacteria, crystals, sugar, and to see if the dog is concentrating his urine normally. If there are WBCs and bacteria present in the sample this is indicative of infection and a culture and sensitivity should be done. Finally, as we discussed, a urinalysis can be very helpful in diagnosing the problem of retrograde ejaculation. The urine should be collected by cystocentesis immediately after semen collection to look for the presence of large numbers of spermatozoa.

Let's say your testing has all come up non-diagnostic to this point. You may then consider doing an epididymal and/or testicular biopsy and submit it for histopathology to see if you can get a diagnosis. However, these are invasive procedures and may themselves result in damage and failure of spermatogenesis. These procedures are usually used in the case where the owner wants an answer but realizes we are at the end of the line anyway as far as future fertility goes.

I just want to mention one other test that is available through several universities. The karyotype test is generally used in the case of a young dog when a congenital abnormality is suspect (like the XX sex reversal). Certainly if the animal has abnormal or ambiguous genitalia this test should be run. Either a blood sample or a skin biopsy can be submitted and the test involves a process that allows the individual chromosomes to be evaluated for abnormalities. Normal males have a karyotype of 78 XY and normal females are 78 XX.

So what treatments are available in cases of canine male infertility? To be honest, the prognosis is poor for many types of azoospermia. For example, a congenital problem in a young dog with no evidence of ever having produced spermatozoa is unlikely to respond to therapy. On the other hand, a problem such as hypothyroidism or other systemic illnesses can certainly be treated. Bacterial prostatitis, epididymitis, and orchitis can be treated with long term (often 6-8 weeks) antibiotic treatment, and toxins or drugs causing problems can be removed. Treatment

for obstruction would be surgical but this has rarely been done in dogs. Retrograde ejaculation was mentioned earlier and this condition is sometimes reversible with administration of pseudoephedrine or phenylpropanolamine at appropriate levels.

This is one instance where time can be the most appropriate therapy because spermatogenesis (sperm formation) and maturation take approximately 75 days in the canine, and it may take several cycles before sufficient numbers of functional sperm appear in the ejaculate. Therefore, several months of sexual rest may be necessary to treat some disorders. Semen evaluations can be done every six weeks or so to ascertain progress.

Oligospermia has a better prognosis than azoospermia and generally reflects infection/ inflammation of the reproductive tract causing decreased numbers of sperm in the ejaculate. Infection again can be treated with antibiotics, and prognosis depends on the chronicity of the condition, i.e., how much inflammation, fibrosis, and degenerative changes have already occurred.

Breeding management can be done with oligospermic dogs since they are not necessarily infertile. Breeding should be by natural mating if possible and progesterone testing of the bitch should be done to maximize each breeding by being as close to the time of fertilization as possible. Intrauterine insemination may be beneficial for these breedings, and the dog should be collected every two to four days to maximize fertility.