



AMERICAN KENNEL CLUB
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Vaccination Update

Dr. Susan Anderson

Physical Therapy – What We Can do to Speed Recovery

Dr. Carl Jehn

My Dog Has Seizures – What Can I do? An Epilepsy Update

Dr. Cheryl Chrisman

Home Dental Care – You Can Save Those Teeth!

Dr. Susan Anderson

I Can't Leave my Dog Alone – Is This Separation Anxiety?

Dr. Terry Curtis

Big Hearts and Falling Dogs – Dilated Cardiomyopathy

Dr. Amara Estrada

Feeding Dogs for Agility

Dr. Richard Hill

Injuries in Active Dogs – Muscles, Bones and More

Dr. Dan Lewis

Osteosarcoma: The Nemesis of Large Breed Dogs

Dr. Rowan Milner

Those Leaky Dogs – Urinary Incontinence

Dr. Chris Adin

CANINE VACCINOLOGY

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Vaccination

The goal of vaccination is to stimulate the animal's immune system by giving a killed or weakened form of an organism. The animal will then be protected from becoming ill when exposed to the real disease. The two major parts of the immune system are humoral immunity (B-lymphocytes that produce antibodies) and cell-mediated immunity (T-lymphocytes that directly or indirectly destroy infected cells). Vaccination is, therefore, critical in the prevention and control of disease. For the most part, veterinary vaccines are safe and efficacious and have saved many animal's lives. An important point to remember is that vaccination is a medical procedure that should include the same considerations and reasoning skills required when deciding to use any medical treatment or surgical procedure. The goal should be to vaccinate more total animals in the populations and to vaccinate individual animals less frequently and only for diseases for which there is a risk of exposure.

Risk Assessment

The potential risk of exposure to various diseases should be determined for each individual patient. Vaccination recommendations will vary depending on the environment of the dog. Consider the differences of exposure for a Poodle living on the 20th floor of a high-rise who never goes outside vs. the racing Greyhound vs. the suburban Golden Retriever in a fenced yard vs. the rural bird dog who roams many acres.

Duration of Immunity

If humans generally receive only childhood vaccinations then why do dogs have annual revaccinations throughout their entire lives? This is definitely an area of intense research and controversy in veterinary medicine today. Most vaccines produced before 1995 were only evaluated for efficacy for several weeks after the last vaccination was given and the recommendations for annual revaccination was arbitrary. The exception, of course, is rabies vaccine that must provide 1 or 3 year duration of immunity under strict guidelines from the USDA. Since 1995, new vaccines for diseases that have not previously had an approved product must be shown to produce the duration of immunity advertised on the label. To really determine efficacy of vaccines, vaccinated and unvaccinated animals need to be kept for one to many years and then challenged with exposure to the real disease. Ideally, most of the unvaccinated animals that are challenged should get sick and/or die and most of the vaccinated animals should not get sick. The difficulty and expense (in dollars and animal lives) of such studies is great, so it is understandable why results from such studies are limited. Appropriate vaccination with distemper and

parvovirus vaccines probably affords long-term immunity (years) whereas vaccination with leptospirosis and kennel cough vaccines may last less than one year.

Core Versus Non-Core Vaccines

Core vaccines are those which should be given to every dog to provide protection against diseases that are serious, common or a risk to humans. These include parvovirus, distemper, adenovirus (infectious hepatitis) and rabies. Non-core vaccines are those given on the basis of risk assessment. These vaccines include coronavirus, parainfluenza, leptospirosis, bordetella, Lyme disease and giardia.

Maternal Antibodies

Newborns can respond immunologically at birth, but the response is slow and inferior compared to older animals. Protection for newborns is provided by antibodies that are passed in the colostrum from the dam to the puppies during first 24-72 hours after birth. Eighty-two percent to 98% of the maternal antibodies come from the colostrum, while only 2-18% of antibodies are transferred in utero. The amount of antibody the pups receive depend on the antibody titer of the dam and how much colostrum each pup receives. Maternal antibodies can interfere with the ability for puppies to respond to vaccination by inactivating the vaccine just as if it were the real disease. Vaccination, therefore, starts at 6-8 weeks of age when the levels of maternal antibodies are waning. There is a critical period of susceptibility to disease for puppies when there are not enough maternal antibodies to protect the puppies from disease, but too many antibodies to allow for active immunization. Exposure to disease during this time may result in sick puppies even though they may have received one or more vaccinations. The most common example is exposure to parvovirus.

Types of Vaccines

The two most common types of vaccines are modified live (attenuated) and non-infectious or killed vaccines. There are newer types of vaccines becoming available that include recombinant vector, recombinant protein and purified subunit vaccines. The infectious agent in modified live (MLV) vaccine has been modified so that it will produce an immune response but not cause disease as it replicates in the body. The advantages of MLV vaccines are that they provide rapid protection of a long duration, they are less allergenic, they produce cell-mediated, humoral and local immunity, they overcome maternal antibody more quickly and can often protect with one dose. Disadvantages of MLV vaccines include the risk of contamination with other infectious agents, the possibility of producing disease in immunocompromised patients, the possibility of reversion to virulence and the need to be handled more carefully to maintain effectiveness. Killed vaccines have the advantages of being safer in immunocompromised patients, not reverting to virulence and they are more stable in storage. The disadvantages, however, are that killed products are more likely to cause allergic reactions because they contain more foreign protein, they require the addition of adjuvants (non-specific immunostimulants), they require that two doses be given initially,

they require more frequent revaccination and they don't stimulate much cell-mediated immunity or local immunity. In regard to the newer types of modified live vaccines, the recombinant vector vaccines take the genetic code for key immunogenic proteins and insert them into a non-pathogenic organism like pox virus, herpes virus, bovine papillomavirus or Simian virus 40. The goal is to stimulate immunity without the potential of causing disease. Examples of this vaccine are Merial's Recombitek CDV for distemper and Recombitek-R for rabies protection. The newer types of non-infectious vaccines are a purified subunit vaccine containing just the specific proteins to stimulate a good immune response (Pfizer's Leukocell-2 vaccine for cats) and the recombinant protein vaccine where the desired gene is cloned into an organism that produces a desired protein then the protein is harvested and purified (Merial's Recombitek Lyme vaccine).

Vaccine Failures

Not all vaccines will effectively immunize due to host factors, vaccine factors or human factors. Host factors include whether the animal is incubating a disease, is debilitated or malnourished, has a high or low temperature, has existing maternal antibodies or some form of immunodeficiency. Vaccine factors that may cause failure to immunize include improper storage or handling, strain differences between the vaccine and the actual infectious agent, excessive attenuation and reversion to virulence. Human factors that cause vaccine failure are improper vaccination protocol, vaccine interference, improper mixing, improper route of administration, improper disinfectants and the concurrent use of some antimicrobials.

Adverse Reactions

Complications can develop after routine vaccination that can be immunologic or non-immunologic. Immunologic reactions include Type I immediate hypersensitivity or anaphylaxis usually caused by killed vaccines especially Leptospira bacterin and rabies vaccine. Miniature Dachshunds seem to have a higher incidence of this type reaction than other breeds. Immune-mediated hemolytic anemia and thrombocytopenia are examples of Type II hypersensitivity. Modified live adenovirus-1 vaccine can cause Type III hypersensitivity (uveitis). Non-immunologic reactions include local injection site reactions, cutaneous granulomas, especially in Poodles and Bichon Frises, systemic fever and malaise. If modified live vaccines are given during pregnancy, vaccine infections can result in fetal malformations, death or abortion. Neonatal infections can occur if puppies less than 4-5 weeks of age are vaccinated with MLV vaccines. Some related Akitas have developed polyarthritis after receiving vaccines especially MLV. The dogs often must be euthanized by 2 years of age due to progressive disease and renal failure. Young Weimeraners with a primary immunodeficiency defect can develop hypertrophic osteodystrophy (HOD) and juvenile cellulitis usually associated with attenuated distemper vaccine. Vaccines can also cause illness if given to an immunocompromised animal, if the vaccine given is incompletely attenuated or if the vaccine is contaminated with another virus. Adverse reactions should always be reported to the manufacturer of the vaccine and to the U.S. Pharmacopia on-line at <http://www.usp.org/prn/vprp.htm> or by calling 1-800-487-7776.

Recommended Sites for Vaccination

The location where vaccines are given along with the manufacturer, type of vaccine and serial number should be recorded in the medical record. Site recommendations include giving the DA₂PP(L) subcutaneously in the right shoulder and rabies subcutaneously or intramuscularly in the right rear leg. Other vaccines and sites should be recorded in the medical record also.

Vaccination Protocol for Dogs at UF VMTH

Distemper, adenovirus type 2, parainfluenza and parvovirus (high titer) are given starting at 6-8 weeks of age and repeated every 2-4 weeks until the puppy is 16 weeks of age, Leptospirosis is included in the combinations when the puppy is 9 weeks of age. DA₂LPP is repeated in 1 year and then given on a biannual basis opposite rabies vaccine. Rabies is given at 12-16 weeks of age, repeated in 1 year and then given biannually (Alachua County requirements). Bordetella/parainfluenza is given intranasally to dogs at risk for infection at > 3 weeks of age or 7-14 days preboarding and repeated every 6-12 months of age. Corona virus vaccine, Lyme vaccine and giardia vaccine are not currently used at the UF VMTH.

Current Canine Vaccines

Distemper. Vaccinations against distemper usually produce solid and long lasting immunity. Reports of outbreaks of distemper in the 90's in the Midwest were found to be secondary to poor vaccination protocols or no vaccination at all rather than the appearance of a "new strain" of virus or ineffective vaccines. If distemper in puppies is a particular problem a heterotypic measles vaccine can be used for the first vaccination between 9-12 weeks of age. It will protect pups from distemper in the face of maternal antibodies. The vaccine must be given IM and not given later than 12 weeks of age so the pups don't develop permanent immunity to measles. Pups receiving measles vaccines require two more distemper vaccinations to provide immunity.

Infectious Canine Hepatitis. Canine adenovirus Type 1 causes hepatitis and the CAV-1 vaccine can cause acute anterior uveitis and corneal opacity (blue eye). To prevent this Type III hypersensitivity reaction CAV Type 2 vaccine is used to provide cross protection to hepatitis and reduce the risk of the adverse reactions.

Leptospirosis. Leptospira bacterin is the most allergenic component of most vaccines and can result in anaphylaxis. Combinations with other killed products such as coronavirus can increase the risk of anaphylaxis. Vaccination reduces the severity and incidence of the disease but not the carrier state. Immunity that is developed is of short duration (6-8 mos.). Also the serovars in the vaccine (canicola and icterohemorrhagica) are not cross protective to the serovars of the disease more commonly diagnosed (bratislava, grippityphosa and pomona). Puppies should not be given Lepto until they are 9 weeks of age because the bacterin can be immunosuppressive in young pups.

Infectious Tracheobronchitis. Infectious tracheobronchitis (kennel cough complex) can be caused by multiple agents that include parainfluenza, bordetella, adenovirus-1, adenovirus-2, herpes virus, distemper, reovirus and mycoplasma. The most common pathogens are parainfluenza and bordetella. The best protection seems to come from a modified live combination product of parainfluenza and bordetella that is given intranasally. The intranasal product begins protecting within 4 days by stimulating local immunity in the nasal cavity (IgA). The intranasal product protects against clinical disease and shedding of the virus. It can also produce mild clinical signs post vaccination. The vaccine can cause serious disease if given parenterally by accident. The injectable form of parainfluenza/bordetella is not as effective as the intranasal vaccine due to the mucosal nature of the disease. Two doses are required for protection and it protects against clinical disease but not virus shedding.

Canine Parvovirus. Breeds that seem more susceptible to parvovirus include Doberman Pinschers, Rottweilers, Labrador Retrievers, American Staffordshire Terriers and German Shepherds. The disease is most common in puppies < 6 months of age and is rare in dogs > 1 year of age. Maternal antibodies can interfere with active immunization so there is a critical period of susceptibility that was previously discussed. When giving conventional parvo vaccines it was previously recommended to give the last vaccine at 18-22 weeks of age especially for susceptible breeds. There is a new high titer/low passage vaccine that contains more virus and is more immunogenic, therefore, overcoming maternal antibodies and stimulating active immunity more quickly. Most manufacturers claim most puppies are protected by 12 weeks. It is recommended to vaccinate at 16 weeks to protect as many puppies as possible.

Canine Coronavirus. Coronavirus is primarily a disease of puppies usually < 6 weeks of age and is self-limiting. Studies show that if parvovirus infection is prevented, canine coronavirus is almost nonpathogenic. Vaccination should be considered in a kennel situation with confirmed coronavirus illness in young puppies.

Lyme Disease (Borrelia burgdorferi). Ninety-nine percent of Lyme disease cases occur in the northeast and mid Atlantic coast, the upper Midwest near the Great Lakes and in the Pacific Northwest. Tick control is most important as it takes 24-48 hours to transmit the organism during the feeding of ticks on the dog. Vaccines should be given to at risk: dogs in endemic areas beginning at 9-12 weeks of age and repeated in 3 weeks then repeated annually or if at risk.

Rabies. Rabies is an important vaccine to be given especially since once humans and animals develop the clinical signs of the disease it is 100% fatal. Rabies is a killed product and highly allergenic. It is very high in antigen and adjuvant so that animals can be protected with one dose instead of two doses 3-4 weeks apart.

Giardia. There is a vaccine available for Giardia called GiardiaVax by Ft. Dodge. It apparently will prevent clinical disease and reduce the severity and duration of cyst shedding. The vaccine is recommended to be given at 8 weeks of age and repeated in 2-4 weeks. It has a demonstrated duration of immunity of 1 year after challenge. The vaccine should only be considered on the basis of risk assessment.

Canine Rehabilitation

Dr. Carl Jehn

Introduction

In the past, the focus of traditional patient care has been on preoperative management, diagnostic techniques and surgical treatment. These aspects are indeed a critical part of proper patient management and typically provide the patient with a favorable outcome and adequate owner satisfaction. However, as owner's expectations increase, the standard of care increases as well. This improved standard of care is also influenced by the fact that the techniques and treatments utilized are becoming more sophisticated as specialization in the field of veterinary medicine becomes more common. Consequently, postoperative management with emphasis on rehabilitation has become an important part of this new standard of care.

What's available?

Canine rehabilitation is a relatively new field of study. Veterinarians and veterinary technicians generally do not receive a great deal of training on rehabilitation through their standard curriculum, although this is changing. Some veterinary medicine programs are now instituting elective courses on rehabilitation and hands on training in clinical rotations has become a part of several programs. While this is certainly helpful, in general, most veterinary medicine programs do not provide a full spectrum of training in this field. Acquiring sufficient education requires working with specialists in the field through continuing education courses and other resources. There are several certificate programs in the Netherlands, the United Kingdom and in the United States. A formal program which provides complete training in veterinary rehabilitation has been developed at the University of Tennessee and other programs are likely to develop elsewhere in the future.

Physical therapists are obviously very well trained in the field of rehabilitation. The training a physical therapist receives provides a comprehensive understanding of the principles and techniques involved in rehabilitation. Unfortunately, most physical therapy programs provide little or no education with regard to animals. There are opportunities for interested physical therapists to explore the field of veterinary rehabilitation through special interest organizations however, state licensing limits the amount of responsibility a physical therapist can have regarding a patient's care.

Collaboration with respect to animal rehabilitation can and does occur between veterinarians, veterinary technicians, and physical therapists. This collaboration is legal and, ordinarily, encouraged. It is important however that primary case responsibility be maintained by a licensed veterinarian and that any care provided by a physical therapist or trained technician, be supervised by the overseeing veterinarian. It is also important that a physical therapist performing rehabilitation not refer to themselves as a *physical therapist* or to what they are doing as *physical therapy*. Physical therapy is considered protected terminology and implies training in the field of human medicine and only

human medicine. In order to circumvent this logistical issue, the veterinary and physical therapy professions have begun to adopt the term *rehabilitation*.

What can you do as an owner?

The owner plays a critical role in the rehabilitation process, much as a person rehabilitating his or her own injury would be. Successful rehabilitation is dependent on proper instruction from the veterinarian and proper owner compliance. Each case must be evaluated on an individual basis and the rehabilitation program must be tailored to the patient, the injury, the owner, and the patient's rate of progress. To become proficient in canine rehabilitation takes years of practice and education. It would be impossible to cover all of the necessary information in a single lecture. What we *can* do is cover the basic methods used in canine rehabilitation and some of the principles behind them. The objective is to help you, the owner, become more aware of what services are available so that you are educated with the necessary information should you decide that your dog would benefit from rehabilitation.

Goals of rehabilitation

treatment have always played a major role in patient care and are obviously important. Furthermore, in the past this alone often provided a favorable outcome and acceptable owner satisfaction. Today's owner however expects a higher standard of care. This includes an increased focus on postoperative management. Why? What are the benefits in providing rehabilitation for your pet? The answer lies in the goals of rehabilitation: to decrease pain, to slow muscle loss due to disuse following injury or surgery, to reduce inflammation and swelling, to speed recovery, to improve function, to prevent re-injury, and to improve the patient's overall quality of life. These are all benefits that can be attained with rehabilitation.

Therapeutic interventions

Therapeutic interventions (the techniques used for the purpose of rehabilitation) fall under several main categories: therapeutic exercise, manual therapy, orthotic/prosthetic treatment, electrical stimulation, and thermal treatment. Determining what interventions are appropriate and when they are appropriate are important considerations to make when designing a treatment protocol that is best suited for your dog and his or her injury. In order to better understand when and what techniques are to be used, therapeutic interventions can also be classified in terms of the phase of rehabilitation in which they are used. The four main phases of rehabilitation include the following: the protection phase, controlled phase, return to function phase, and the return to high-demand phase. Below we will define each phase, review their respective goals, and discuss the most commonly utilized therapeutic interventions as they apply to each of these phases of rehabilitation.

Protection phase

The protection phase is the initial phase of rehabilitation. It typically lasts 1-7 days following injury. The goals of this phase are to decrease pain and swelling, increase motion, and slow muscle loss. To accomplish the goals of the protection phase we protect, rest, ice, compress, and elevate (PRICE). We protect and rest the injury by utilizing cage confinement, splints, bandages, braces, and minimizing activity. Icing the injury several times a day reduces ongoing inflammation. Compressing the soft tissues with pressure wraps prevents or reduces inflammation, as does elevation (although elevation can be difficult to employ depending on the patient and the nature of the injury).

Under the proper circumstances, passive range of motion (ROM) or joint mobilization, and therapeutic massage are additional techniques that may be indicated during this and the next phase of rehabilitation. Passive range of motion involves passively moving the involved joints through part or all of their range of motion to provide mechanical movement of the injured muscles and the joints. Passive ROM also provides drainage of inflammatory byproducts which accumulate in the affected tissues when they are not regularly used. Joint mobilization has similar benefits as passive range of motion and concentrates primarily on mobilization of the joint capsule and the surrounding ligaments. Therapeutic massage provides mechanical stimulation of the affected tissues and also helps to reduce swelling.

Controlled phase

The controlled phase usually occurs 7-21 days following injury. The goals of this phase are to resolve range of motion impairments, increase muscle strength, improve coordination and increase muscle endurance. To achieve these goals we employ controlled leash walks, splints, slings or carts for assisted standing or walking. We use aquatic therapy (standing, walking, or swimming in water), weight/load shifting, challenging balance with balance boards, walking on inclines and declines, or walking in sand. All of these modalities are techniques that are commonly used to encourage limb use and coordination, and does so at a *controlled* level. It is important to monitor your pet closely during these activities to avoid overexertion and causing re-injury. Re-injury can occur during *any* phase of rehabilitation and is considered a step backward in progress. Re-injury should be avoided at all costs. There are a number of indications that re-injury has occurred. Common signs include reluctance to continue with therapy, decreased stamina, increased signs of pain or lameness, or increased swelling or redness.

We can also begin to employ the use of heat therapy in this phase of rehabilitation. Here, the goal is not to prevent swelling with cold but to remove swelling which has already occurred using heat. Heating pads and hot water bottles are an excellent means of applying superficial heat to injured areas. Heating of deeper tissues however requires the use of modalities such as ultrasound. The use of heat therapy should be used carefully to avoid superficial burns or increased damage to deeper tissues when using ultrasound.

Return to function phase

Return to function typically occurs approximately 3 weeks following injury. The goals of this phase are to prepare the patient for normal daily activity. Examples of techniques that may be suitable to incorporate into a rehabilitation protocol at this phase of recovery include: leash walking, stair climbing, pole/cone weaving, walking in tall grass, swimming, jogging, playing tug-o-war, and using balance-boards. These activities start at a low level of intensity, which is often determined by the duration of the activity, and the intensity is slowly increased as progress is made by the patient (e.g. duration of activity is increased). As in the previous phase, we use these and other various techniques to encourage limb usage and put increased demands on the strength and coordination of the involved joints and muscle groups.

It is important that progression from one phase to the next and the intensity of the exercises used within each phase are done in coordination with and under the supervision of your veterinarian. Progression to the next phase is dependent on achieving the goals of each phase, the progress of the individual patient, and the nature of the injury. All of these factors must be taken into consideration when working with your veterinarian on your dog's rehabilitation.

Return to high-demand phase

Return to high-demand is typically reserved for working and athletic dogs. Progression into and the techniques used during this phase of rehabilitation depend a great deal on the nature of the dog's injury and their ability to achieve the goals outlined in the previous phases. You should consult with your veterinarian *ad nauseam* before progressing your dog to this level.

The activities utilized in this phase are also somewhat dependent on the activity for which the patient is preparing. For example, a dog preparing to enter into field trials will require a regimen of activity much different from a police dog. Regardless of the specific demands placed on that patient, the general guidelines for this phase of rehabilitation are to incorporate activities that pertain to the dog's physical demands while being maintained under controlled and supervised conditions. As before, the intensity of the exercises used in this phase begins at a lower level and is slowly increased over time as progress is made. At this phase of rehabilitation the patient continues to push the limits of their newly healed tissues. It is important that this be done in a controlled fashion and that the dog is closely monitored to avoid re-injury. Once the patient can be taken up to or *beyond* those demands encountered under their "normal" high-demand activity, without any evidence of re-injury, this phase of rehabilitation is complete.

Other treatment modalities

There are other modalities used in rehabilitation protocols which, under the proper circumstances, may be incorporated into the protocol guidelines discussed above.

Ultrasound is a form of treatment which provides what are called *thermal* and *non-thermal* effects. The thermal effects are those observed as a result of heating the targeted tissues. These effects are of particular use when targeting deeper tissues (> 3 cm below the skin surface) which cannot be reached by using superficial heat sources such as heating pads. Ultrasound heat promotes blood flow, increases the extensibility of the tissues, raises the pain threshold, stimulates the activity of enzymes involved in the healing process, and augments nerve conduction velocity. Ultrasound heat also alters the molecular bonding and viscoelastic properties of scar tissue (i.e. helps to break down scar tissue). Ultrasound's non-thermal effects include acoustic streaming (steady, circular flow of cellular fluid), microstreaming (micro-scale eddying), and cavitation (formation, growth and pulsation of vapor-filled bubbles). These non-thermal effects positively affect cellular activity. In general, ultrasound is useful in reducing pain, inflammation, and scarring, and helps to promote healing.

Neuromuscular electrical stimulation (NMES) is another treatment modality which is used to stimulate muscles in much the same way nerve fibers do. This is useful in patients with orthopedic disorders such as decreased joint mobility, joint contracture, swelling, decreased circulation, disuse atrophy, muscle weakness, loss of voluntary motor control, decreased sensory awareness, muscle spasms, pain, and gait abnormalities. Neurologic conditions such as victims of stroke, closed head injuries, spinal cord injuries, or paralysis may also benefit from NMES.

When used correctly, these and the other techniques discussed above are all extremely helpful in providing a rapid and strong recovery while experiencing a reduced level of pain and an improved quality of life. These techniques should be used under the supervision of a veterinarian familiar with the concepts of rehabilitation and should be accompanied by thorough client education and support. There are many uses and indications for physical therapy. The information covered herein is just an overview. If you have further questions or would like to know more about rehabilitation, consult with your local veterinarian for more information.

EPILEPSY AND YOUR DOG

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What is epilepsy?

Epilepsy is an electrical disturbance of the brain characterized by a brief upset of mental and body functions referred to as seizures. Nerve cells of the brain normally emit electrical impulses. These electrical impulses flow harmoniously together through the brain and keep the mind and body functioning properly. If brain nerve cells are injured or upset, they may occasionally emit excessive abnormal electrical impulses like an electrical "brainstorm". When this generalized electrical disturbance occurs, a seizure results.

What causes epilepsy?

Anything that can injure or upset the nerve cells of the brain can cause a seizure. Epilepsy may develop from mild residual nerve cell injury following head trauma or encephalitis. The first seizures may occur several months to a year after this injury. The delay in onset of the seizures often makes it difficult to determine what originally injured the nerve cells, but there is no active underlying disease process occurring in the brain and the condition is not inherited. This seems to be the most common type of epilepsy in dogs

In certain pure breeds of dogs especially Poodles, German Shepherds, Irish Setters and Beagles as well as others, a hereditary type of epilepsy occurs. In this type of epilepsy, the cells do not develop properly and are capable of periodically emitting these abnormal electrical impulses and producing seizures.

Seizures may also be caused by toxicities, encephalitis, low blood sugar, liver problems and brain tumors, so a veterinarian should evaluate dogs with seizures to determine what the proper treatment should be.

What happens during an epileptic seizure and what types of seizures do dogs have?

During a seizure the normal electrical impulses of the brain are disturbed and the dog may lose control of its body and become confused or unconscious for a short period of time. Only rarely do they become vicious during this period. Your dog is more likely to be frightened, if it is conscious.

In a mild generalized seizure, the dog may not lose consciousness, but will shake and lose control of the legs symmetrically on both sides of the body. They may be frightened and try to crawl to their owner or may hide. Some dogs salivate and/or vomit with this type of seizure. Although this seizure is mild, it can last 20 minutes or more. If the seizure continues beyond 20 minutes, a veterinarian may have to stop the seizure.

In a more severe generalized seizure, the dog falls upon its side unconscious with its eyes open. The legs may be very stiff, shake, or paddle but are symmetrically affected. The jaws may make chewing motions, be open or closed. The dog may also salivate, urinate and/or defecate during this type of seizure. The seizure usually lasts only 1-3 minutes, and then the recovery period begins. Dogs don't remember the seizure itself and are not in pain. If there are many seizures in a row or a long seizure over 5-minutes, this is an emergency and veterinarian may be needed to stop the seizure.

In a focal seizure, the dog is often conscious, but may momentarily lose control of one part of their body or may stare off in space at imaginary objects. The appearance of these focal seizures varies depending on the area of the brain involved. The seizures can be mild, but can then develop into a more severe form where they are on their side unconscious salivating, twitching and paddling but often one side of the face or body is more affected than the other.

What can be done for your dog during a seizure?

Once a typical complete generalized seizure has begun, there is usually nothing you can do to stop it without rectal drugs previously obtained from your veterinarian. If multiple seizures occur and involve repeated trips to the emergency service your veterinarian can prescribe diazepam (Valium) impregnated suppositories compounded to the correct dosage for your dog. The suppository is inserted through the anus into the rectum. The anus is the first opening just underneath the tail.

During the seizure your dog should be held or otherwise protected from falling off the bed or down the stairs or injuring itself in any way during the seizure. Dogs do not swallow their tongue during the seizure. The uncontrollable chewing movements of the jaws may accidentally pinch your fingers if you put your hands into their mouth. If an object is placed in your pet's mouth, breathing may be obstructed and suffocation may occur. If your pet consistently bites and injures its tongue during the seizure, an object can be carefully placed between the back teeth on one side, but care must be taken not to obstruct breathing. Dogs may be held and gently stroked or put in a padded environment during the seizure, but should not be tied up. In a mild seizure, pet owners report they can calm their dog by stroking or holding them and stop or shorten the seizure.

What should be done for your dog during the recovery period after a seizure?

After the seizure, your dog often goes through a recovery period. The appearance and length of this period will vary from animal to animal. Some dogs want to urinate or defecate, if they did not do so during the seizure. Some are thirsty and hungry and others are exhausted and want to sleep. Your dog should be allowed to urinate, defecate, eat, drink and rest; then they usually return to normal.

Some dogs are very hyperactive during the recovery period. They may constantly pace and bump into objects. If the hyperactivity is severe, your dog may require sedation by drugs to keep them from injuring themselves during the recovery period. Diazepam (Valium) suppositories may reduce the hyperactivity following the seizure. The recovery period length varies from a few minutes to several hours with different dogs and seizure types.

The seizure and recovery period are frightening for some people. However, dogs rarely die in a brief seizure and usually have no memory of it. Multiple seizures close together or long seizures can cause brain damage and emergency veterinary assistance should be obtained to stop the seizure. Most dog owners adjust to the possibility of an occasional seizure and can properly care for their pet during and after the seizure.

Can epilepsy be cured?

Epilepsy is rarely cured but the seizures in most pets can be controlled with treatment. Treatment keeps the abnormal electrical impulses of nerve cells to a minimum, so that surrounding nerve cells will not be disturbed and the seizure cannot occur. If seizures are mild and infrequent like a few times a year, often no treatment is given. Mild seizures may be treated with herbal therapy, acupuncture or homeopathy administered by a

veterinarian specially trained in these modalities. Acupuncture may be helpful alone or combined with more traditional drug therapy in dogs with more frequent or severe seizures.

When seizures occur once a month or in clusters (multiple seizures in a row over a short time) drug therapy is often prescribed. Every dog reacts differently to the drugs needed to control seizures and dosages have to be regulated for each individual. It may take a larger dosage of medication to control seizures in a small dog than in a large dog. It takes patience and working closely with your veterinarian to find the right drug and the right dosage to control your dog's seizures with minimum side effects. Usually the seizures can be kept to a minimum and the dog can be normal in between them.

Owners of epileptic dogs often find it helpful to keep a calendar and record dosage and time of daily medication and the occurrence of any side effects and seizures. The veterinarian can review the calendar and try different medication regimens to determine what best controls the seizures without sedation or other side effects.

Phenobarbital and potassium bromide (KBr) are the most common drugs used to control seizures in dogs. The dog metabolizes diphenylhydantoin (Dilantin) differently than humans and this drug is less effective in dogs than humans. Phenobarbital and KBr are quite inexpensive and usually can be given every 12 hours to control seizures. Phenobarbital may be used first if seizures occur multiple times a week but KBr may be used first if seizures are less frequent.

The other anticonvulsants used in man may be tried when all else fails but they have more side effects and are expensive. The veterinarian will usually begin with a single anticonvulsant and adjust the dose until the seizures are controlled and there are no side effects of sedation. If there are side effects or the seizures are not controlled, a serum phenobarbital level is obtained. In dogs with seizures that are difficult to control, the oral dosage should be increased until the serum level is 30 micrograms per milliliter. If serum phenobarbital levels occur above that level, then liver damage may occur.

The most common side effects of the phenobarbital and KBr are sedation and rear limb weakness. If the side effects do not pass in 24-48 hours, the dose should be decreased with a veterinarian's guidance. A few dogs may have bizarre behavior or be hyperactive on phenobarbital. This is not a permanent change and will go away as the medication wears off. For these dogs, mephobarbital may be a better drug choice. Increased appetite, thirst and urinations may be a side effect of phenobarbital and KBr. If these signs are severe, the drug should be decreased or changed as the dog may gain excessive weight.

KBr takes 3-4 weeks to be effective but may have less sedative side effects than phenobarbital. KBr must be given with food as your dog may be nauseated and vomit if it is given on an empty stomach. KBr is toxic to human and so gloves should be worn during administration and all contact avoided. After 3-4 months serum KBr levels can be evaluated; seizure control is achieved at levels between 2-4mg/ml. In dogs with cluster seizures (multiple seizures at one time) the dosage may have to be increased until the

serum level is 4 mg/ml. If seizures are not controlled with medication, adding acupuncture may control seizures.

How long should medication be continued?

Once medication is begun, it usually is given indefinitely and often for life. The dosage may have to be changed periodically to keep the seizures controlled. Drug tolerance to phenobarbital is common and the dosages may have to be increased the longer the animal is on the medication. Seizures may be well controlled for a period of time and then the dog may have multiple seizures, which do not respond to medication as well as before. Dogs may go through this periodically and with dosage adjustments the seizures can still be controlled again satisfactorily. Many dogs that have cluster seizures respond well to KBr at higher doses. If there are no seizures for a year, the dosage may be decreased by your veterinarian. Epilepsy rarely spontaneously disappears so some level of medication is often needed for life. Medication may be reduced every 6 months if the dog remains seizure free, but a veterinarian can provide assistance with dosage reduction. Never completely withdraw a drug all at once, as this may send your dog into multiple seizures.

Is there any special care for my epileptic dog?

The epileptic dog needs medication every day at a regular time, usually every 12 hours, but in some cases every 8 hours. The rest of the care is the same as for any other animal and consists of a good balanced diet, exercise and regular check-ups by a veterinarian. A complete blood count and serum chemistry profile and bile acids examination should be obtained every 6-12 months to ensure no liver problems are occurring. The primary aim of seizure control is for the animal to lead a normal healthy happy life and most of them do. For some dogs, over-excitement such as grooming or visits from company exacerbates seizures. A little extra medication given prior to the excitement may stop these seizures and a veterinarian will determine the best dosage. Other dogs have seizures during sleep and are not affected by excitement. Some dogs develop a pattern of seizures that occur every few weeks like clockwork. Any patterns can be detected by studying a completed calendar. Increasing drug dosages during high-risk periods may prevent seizures.

If an epileptic dog becomes sick for any reason, expect that the seizures may become more frequent, especially if the animal has vomiting and diarrhea and does not get the needed amount of medication into its system. If the body chemistry changes due to any disease process particularly liver and kidney disease or thyroid problems, the dosage of medication may have to be changed. Once the disease process is corrected, the seizures become controlled again. Some female dogs have more seizures during their heat period. If this is the case, spaying the dog can correct this. If an epileptic dog needs a tranquilizer for travel do not use phenothiazine derivative tranquilizers (acepromazine) as these drugs may precipitate seizures. Often just increasing the anticonvulsant dose (particularly phenobarbital) will calm them for travel. Diazepam (Valium) may also be used, but consult a veterinarian about the effects of combining this with any current medication.

Dogs on KBr should not have their diet changed as changes in salt intake can affect serum KBr levels.

If an epileptic dog has seizures that are difficult to control, we also recommend using a daily heartworm medication such as filaribits or using once a month interceptor. Some flea control products may worsen seizures so check with your veterinarian.

Will epilepsy become worse with age or shorten my dog's life?

Epilepsy rarely becomes worse with age and some dogs that develop seizures at a very young age may even improve and be able to discontinue medication, as they grow older. Epilepsy with seizure control does not shorten your pet's life. If the seizures are not controllable with anticonvulsants and acupuncture, the dog should be re-evaluated for some serious brain disease or generalized illness in the body. If your dog only has a few seizures a year, this is considered quite well controlled and they should have a healthy long life.

Home Dental Care - You CAN Help Save Those Teeth!

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Periodontal disease is the most common problem of dogs and cats seen by veterinarians with more than 85% of dogs over 3 years of age being diagnosed. Periodontal disease is inflammation of the periodontium caused by plaque. The periodontium consists of the gingiva, the alveolar bone, the periodontal ligament and the cementum. Plaque is the biofilm composed of bacteria, saliva, proteins, oral debris and cells. After 24 hours, plaque becomes mineralized and turns into calculus (tartar). Then as more plaque deposits, it also mineralizes, creating layer upon layer of buildup. Continual inflammation stimulated by the trapped bacteria causes progressive destruction of the periodontium leading to tissue loss, bone loss and eventually tooth loss.

Tooth brushing is the gold standard of home dental care. It is still the most effective means of controlling plaque and periodontal disease. The ideal is daily brushing although brushing 3 times per week still has definite benefits. Your dog will also require periodic professional periodontal therapy by your veterinarian (scaling, polishing, curettage and possibly extractions), however, the benefits of professional cleaning are shorter lived without effective homecare. There are many toothbrushes to choose from. It is important to choose one with soft bristles of a size that will fit the patient's mouth in addition to being comfortable for the "brusher." The brush should be held at a 45° angle to the gum line and moved in small circular motions concentrating primarily on the outside surfaces

of the teeth. A rubber finger brush or gauze wrapped over the finger may be useful in the initial training period but will not clean as well as a brush.

Start training your dog to have its teeth brushed as early as possible – puppy hood is best but it is never too late. Go slowly and gently using rewards and positive reinforcement like a special treat, a walk, playtime and /or a special toy. A flavored veterinary toothpaste or low sodium chicken/beef broth can be used to make the brush more acceptable. Avoid human toothpaste as the fluoride content can be toxic (dogs don't spit) and the foaming agents can cause vomiting. Baking soda should not be used either because too much sodium when swallowed can be detrimental especially in heart or kidney patients.

There are other adjunctive products that can enhance oral care but will not replace brushing. These include diets, treats, chews, toys, rinses and gels. Look for products awarded the Veterinary Oral Health Council (VOHC) Seal of Acceptance that identifies products that have met a pre-set standard for prevention of accumulation of plaque and tartar (calculus).

Dental diets include T/D by Hill's Pet Foods in regular and small bites. The fibers are designed to wipe the teeth instead of shattering when the dog bites down on the kibble. Dry Eukanuba Veterinary Diets by the Iams Company have a Dental Defense system. This is a coating of sodium hexametaphosphate (HMP) that reduces tartar by making it more soluble in saliva. Innovative Veterinary Diets (IVD) has a Canine Dental Formula with a larger kibble size coated with HMP and additional antioxidants added.

Treats and chews include Tartar Check (biscuits coated with HMP), rawhide chews (Chew-eez, C.E.T. Oral Hygiene Chews, C.E.T. HEXtra Chews, Enzadent Oral Care Chews), pig ears, Pedigree Dentabones, Pedigree Dentastix and Greenies. Be aware of the calories some treats can add and also be aware of the possibility of choking or gastrointestinal upset. Avoid using very hard treats or toys like natural bones, nylon bones or cow hooves since they can cause tooth fractures requiring a trip to your veterinarian for treatment.

Rinses and gels generally contain 0.12% chlorhexidine gluconate (CHX, Nolvadent, Hexarinse) or zinc/ascorbic acid (Maxiguard) which are antibacterial. There is a product, Pet Oral Hygiene Solution, by Oxyfresh that can be added to drinking water that contains Oxygene and zinc acetate.

There is a product called Oravet (previously ProVSeal) that is a waxy polymer applied to the teeth following a professional cleaning that inhibits bacterial adhesion to the tooth. The product gradually wears off but it helps to protect against plaque formation while the mouth heals after extraction or surgery. A home product can then be easily applied by the pet owner once weekly to maintain the seal. The product is tasteless, odorless, invisible and non-toxic.

Resist the temptation to use hand scalers or curettes regardless of training. It can cause trauma to gums and excessive wear to the enamel which is quite thin in dogs. Also, without polishing, the roughened surface allows plaque to adhere much faster and easier.

Big Hearts and Falling Dogs - Dilated cardiomyopathy

Amara Estrada, DVM, Diplomate ACVIM (Cardiology)

Overview

The term cardiomyopathy literally means "sick heart muscle." It relates to any disease of the heart muscle that may be manifested by muscle dysfunction or by arrhythmias (abnormal heart rhythms). *Dilated cardiomyopathy*, or DCM, occurs when the heart muscle is thin, weak, and does not contract properly. The condition can lead to congestive heart failure, in which fluid accumulates in the lungs, the chest or abdominal cavities, or under the skin. Because of reduced blood flow to the rest of the body, DCM also can result in weakness, fainting, and exercise intolerance. Abnormal heart rhythms, or arrhythmias, frequently accompany DCM, and can complicate the treatment of dogs with this disease. In rare cases, supplementation with substances such as L-carnitine or taurine may dramatically reduce signs in individual patients. For most dogs, the main goals of treatment are to lessen signs due to congestive heart failure, treat any arrhythmias and to attempt to improve the heart's ability to pump blood. The long-term outlook for dogs with DCM is usually quite poor, and most dogs with DCM eventually die from the disease. Despite the poor long-term outlook, however, many dogs with DCM can benefit from medical treatment that helps control symptoms.

Affected Animals

DCM most commonly affects large or giant purebred dogs, but it also can be seen in smaller breeds such as Cocker Spaniels, and in mixed breed dogs. Some studies have shown a male predominance. In Doberman Pinschers, the males seem to develop more severe disease at an earlier age. With the exception of Portugese Water Dogs, dogs are usually middle aged to older at presentation. The dogs most frequently diagnosed with DCM are Doberman Pinschers, Boxers, Irish Wolfhounds, and American Cocker Spaniels. Saint Bernards, Afghan Hounds, Newfoundlands, Golden Retrievers, Great Danes and Old English Sheepdogs have also been known to develop DCM. The disease in Doberman Pinschers deserves special mention as the prevalence has been estimated to be very high with about 50% of the breed affected.

Clinical Signs

Signs may be consistent with right heart failure, left heart failure, or both. Right heart failure signs can include abdominal distention due to ascites, jugular venous engorgement or pulsation, hepatomegaly (liver enlargement), pleural effusion (fluid around the lungs),

peripheral edema (fluid within tissues), pericardial effusion (fluid surrounding the heart), and weight gain due to fluid retention. Left heart failure signs can include cough due to pulmonary edema (fluid within the lungs), shortness of breath, tachypnea (fast breathing), and dyspnea (difficult or labored breathing). Some signs can be seen with right or left sided heart failure, including fatigue and weakness, exertional dyspnea, gallop rhythm, pallor, increased capillary refill time, cyanosis (blue color to skin), cool extremities, and weight loss.

Symptoms

Dogs with dilated cardiomyopathy may remain asymptomatic for years prior to presentation because mild to moderate disease does not result in clinical signs. Dogs are usually presented to the veterinary hospital when they are in congestive heart failure. It is also common for dogs with DCM to present for collapse (or “syncopal”) episodes due to arrhythmias and low cardiac output. Arrhythmias such as atrial fibrillation and ventricular tachycardia are common in dogs with DCM. Dogs can show symptoms due to right-sided congestive heart failure including abdominal enlargement; distention of the veins in the neck or other parts of the body; and fluid accumulation in the abdomen or chest, in the sac around the heart or underneath the skin, especially in the legs and on the underside of the body. This fluid retention can lead to perceived weight gain. Other dogs will show evidence of lung problems due to left-sided congestive heart failure, including shortness of breath, rapid, shallow breathing, difficulty resting comfortably at night, and coughing. It is also possible for dogs with DCM to present with signs of both right and left heart failure.

Description

The primary abnormality occurring with DCM is impaired function of the ventricles due to decreased strength of the heart muscle. The left ventricle pumps blood returning from the lungs to the rest of the body, and the right ventricle pumps blood from the rest of the body to the lungs. When the heart muscle fails, this blood is no longer pumped out of the heart efficiently. Blood then “backs up” into the lungs, the chest cavity or the abdomen. Reduced output from the heart may result in signs such as weakness, exercise intolerance, fainting, and shock. Heart valve insufficiencies, abnormal heart rhythms or arrhythmias, and the results of the body’s compensatory responses to reduced heart muscle performance can compound the problems seen with DCM. Ventricular arrhythmias can often lead to sudden death, especially in Boxers and Doberman Pinschers. The development of atrial fibrillation can have important short-term and long-term consequences for dogs with DCM and can also lead to clinical signs of exercise intolerance and collapse.

Diagnosis

The diagnosis often is first suspected when symptoms compatible with DCM are present in a large or giant purebred dog or Cocker Spaniel. Physical examination abnormalities frequently include the presence of an extra heart sound called a gallop rhythm, or a soft

heart murmur. Arrhythmias can be detected while listening to the heart with a stethoscope and while feeling for the pulse or heartbeat. Abnormal lung sounds are heard in dogs with left-sided heart failure, while distention or pulsation in the jugular veins, liver enlargement, or abdominal fluid accumulation may be present in dogs with right-sided heart failure. Femoral arterial pulse quality may be diminished. Chest x-rays should always be evaluated in patients suspected of having heart disease. Heart enlargement and blood vessel changes consistent with heart failure may be seen, and fluid accumulation in or around the lungs can be identified if heart failure has developed.

The most definitive diagnostic test for DCM is an echocardiogram, an ultrasound evaluation of the heart. Heart chamber dilation and enlargement, reduced heart muscle wall thickness, and decreased heart muscle wall movement are the hallmarks of DCM. In addition, mild heart valve abnormalities may be seen. An electrocardiogram (ECG) may identify abnormal rhythms or evidence of cardiac enlargement. The most common rhythm disturbance occurring with DCM is atrial fibrillation, a condition characterized by a tremendous increase in the rate of firing of the atria, the uppermost chambers of the heart, coinciding with an increased rate of contraction of the ventricles, the lower and larger chambers of the heart. Other rhythm disturbances, including premature ventricular beats or ventricular tachycardia, may be detected. In Boxers and Doberman Pinschers, a 24 hour ambulatory ECG, called a Holter monitor, can be used to diagnose heart disease prior to echocardiographic abnormalities. This is usually performed in an attempt to identify affected dogs who have not yet shown clinical or echocardiographic signs of disease. The goal of these evaluations is to screen dogs being used for breeding purposes in hopes of eliminating affected animals from the breeding population.

Prognosis

The prognosis for DCM dogs that are presented *in congestive heart failure* (which is most of them) is poor but there is breed variability. Remember that these dogs have a very long pre-clinical phase in which they were asymptomatic. The mean survival for a Doberman Pinscher in heart failure is 90 days. Those Dobermans that present in biventricular failure or with the presence of atrial fibrillation do even worse and only survive, on average 3 weeks. Many Dobermans with DCM die suddenly due to a fatal arrhythmia, presumably ventricular fibrillation. For other large breed dogs, average survival following the first episode of congestive heart failure is 3-6 months. These numbers are *averages*, meaning some dogs may live 2 months while others may live 9 months. If it is found that a nutritional deficiency such as taurine or L-carnitine deficiency is the cause of the DCM, then the prognosis is good as many of the cardiac changes can reverse.

Treatment

DCM generally is not curable, and spontaneous recovery is unlikely. The primary goals of treatment are to lessen clinical signs of heart failure and to prolong survival. Treatment of an individual dog is dictated by the severity of its signs at the time of diagnosis, and the presence or absence of changes such as congestive heart failure and

arrhythmias. Medications are used to treat the consequences of heart muscle failure, to attempt to improve the heart muscle's ability to contract, and to normalize or improve rhythm disturbances. Drugs used to accomplish these goals include diuretics, digoxin, antiarrhythmic agents, angiotensin converting enzyme inhibitors, and recently, pimobendan. Supplementation with amino acids such as taurine or L-carnitine may be helpful for some dogs with DCM.

The primary drug to reduce fluid accumulation secondary to congestive heart failure is furosemide, marketed as Lasix. It can be used to treat acute, life-threatening fluid accumulation or to control and prevent congestive abnormalities in chronic settings. Digoxin is used for several reasons in the treatment of DCM. It may help increase the heart's ability to contract and slow down the ventricular response rate in dogs with atrial fibrillation. The use of drugs called angiotensin converting enzyme, or ACE, inhibitors has been shown to benefit people and dogs with DCM by reducing the signs due to heart failure and improving exercise tolerance. ACE-inhibitors have many effects, including blood vessel dilation, which reduces the resistance the heart has to pump against. Other types of blood vessel dilators can be used in the short-term or long-term treatment of DCM to reduce the load that the heart has to pump against to get blood to flow. Antiarrhythmic drugs, such as mexilitene or atenolol, may be used for dogs with ventricular arrhythmias due to DCM. A new drug, not yet approved in the United States but seeking FDA approval, is pimobendan, marketed as Vetmedin. Pimobendan is a drug that improves the contractility of the heart as well as dilating blood vessels. These two actions combined help to increase cardiac output and improve cardiac function in dogs with DCM. Colleagues in Europe have reported much clinical success with pimobendan. While it does not cure the condition, it does seem to improve survival and clinical signs in dogs with DCM.

Taurine deficiency was found to be a cause of DCM in cats in 1987 and since then has been supplemented in commercial cat foods, thus drastically reducing the disease prevalence. Although taurine is not an essential amino acid in dogs (they can make it in the liver, while cats cannot), some dogs with DCM have been identified to be taurine deficient. Breeds that have been found to be taurine deficient include American Cocker Spaniels, Golden Retrievers, Newfoundlands, Dalmatians. We recommend that all American Cocker Spaniels and Golden Retrievers as well as any 'non-typical' breed with DCM should have plasma or whole blood taurine analysis performed.

L-carnitine is a compound that plays an important role in fatty acid metabolism which is important for normal cardiac function. Carnitine deficiency in the heart muscle has been shown to be potentially reversible in at least one family of Boxers with DCM. Testing for carnitine deficiency is not easy as blood or plasma levels of carnitine may be normal while cardiac muscle levels are decreased. The only definitive way to diagnose cardiac carnitine deficiency is through a cardiac muscle biopsy. When plasma deficiency is documented, this means the dog has a systemic deficiency that is likely to be the primary cause of DCM. This situation is relatively rare. Supplementation with L-carnitine for all dogs with DCM can be tried as such supplementation is not known to be harmful. L-carnitine supplementation is very expensive, however, and is likely of no benefit for dogs

with normal myocardial carnitine levels. Other substances, such as coenzyme Q-10, have been used in the treatment of DCM but have no proven benefit.

Prevention

Affected dogs should not be bred. Early screening of dogs of breeds that have a high incidence of DCM may help identify important changes prior to the onset of signs. This can help prevent the breeding of dogs that could pass DCM on to their offspring.

FEEDING DOG FOR AGILITY

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Dog agility is one of several activities involving pet dogs which are gaining popularity. Agility trials involve dogs completing an obstacle course as quickly as possible under the direction of a handler. Courses increase in complexity with the level of competition but commonly include 13-18 different obstacles over a course that is 120-200 yards in length. The assigned course time in which dogs are expected to complete the course assumes a speed of approximately 2 yds/s for the simpler courses and 1.5 yds/s in the more complex courses (UKC regulations). Thus, dogs run at approximately 7 km/h over a very short distance but are required to jump, climb, start and stop and change direction rapidly.

Most nutritional studies involving exercising dogs have been performed either on dogs undertaking sprint exercise such as Greyhounds or dogs undertaking endurance exercise such as Beagles on treadmills or sled dogs in teams pulling loads. Greyhounds sprint for 17-57 seconds over a distance of 300-900 meters at an average speed of approximately 60 km/h (37 mph), whereas racing sled dogs run for up to hundreds of kilometers daily in long distance races of 10-1800 km trotting at 16 km/h (which results in an average speed of 7 km/h over several days when allowing for rest stops). Greyhounds undertake what is called 'supra-maximal' exercise because the rate of oxygen uptake necessary to sustain this rate of sprinting exceeds the maximum rate at which oxygen can be taken up by the body. The extra energy for very rapid sprinting is generated without oxygen. This produces lactic acid which then builds up in the blood. In contrast, long distance running involves 'sub-maximal' exercise. The oxygen required to maintain this type of exercise does not exceed the maximum rate of uptake and concentrations of lactic acid do not increase in the blood. I am unaware of any nutritional studies that have been performed on agility dogs but the type of activity they perform has some characteristics of both these extreme types of exercise and some of the principles derived from studies in other athletic dogs may be applied to agility dogs.

Nutrition should not be treated as a substitute for training, however. Most dogs sleep for more than two-thirds of each day and these dogs cannot be expected to perform optimally at the weekend if they have been lying on the couch for the rest of the week. Training

(frequently repeated exercise) increases bone mass, red blood cell turnover, heart size, maximal oxygen consumption, use of fat for energy and stamina. Training reduces heart rate, lactic acid production and oxidative free radical formation and probably reduces the risk of injury. The benefits of training far outweigh any potential benefit that might be achieved by modifying the diet. Owners of agility dogs should, therefore, exercise their dogs more regularly rather than seek nutritional solutions to improve their dog's performance. This training may involve sprinting, endurance or resistive exercise or interval training in which aerobic and anaerobic exercise are alternated but the ideal type of training remains unknown. It is possible, for example, that sprinting, resistive or interval training may increase muscle hypertrophy and improve the performance of agility dogs more than endurance exercise.

As a reference for the studies that support the statements in this presentation, the reader should turn to the 2004 National Research Council publication on the Nutrient Requirements of Dogs and Cats. It is available in a prepublication form at present and should soon be published in its final form.

ENERGY REQUIREMENTS

The energy required by a dog is composed of energy for maintaining normal body systems when the animal is at rest and not eating (the basal metabolic rate or BMR) plus energy used in consumption of food (dietary thermogenesis) plus energy for keeping itself warm in a cold environment (cold-induced thermogenesis) plus energy for exercise. The energy requirement of a dog, therefore, consists of the energy required in the absence of exercise plus the energy required for exercise.

The energy required for running is proportional to the distance traveled irrespective of the speed (approximately 1 kcal/kg body weight (BW) for each horizontal km traveled). Thus, Greyhounds require little energy because they travel only short distances, whereas sled dogs require massive amounts of energy. Acceleration requires additional energy (approximately 3 kcal/kg BW for each horizontal meter traveled and there is an additional cost of climbing or jumping (0.007 kcal/kg BW for each vertical meter) but dogs recover much of the energy used going up hill when they come down again. Agility dogs travel only 200 m and, therefore, would require little energy (≈ 4 kcal for an average 20 kg BW dog) to complete a trial if that trial involved trotting in a straight line. Agility dogs have to accelerate and turn corners, however. A generous assumption would be that half each trial will involve acceleration or turning corners. This would approximately double the energy requirement (equivalent to an additional 4 kcal/trial for an average 20 kg BW dog). To this must be added an additional cost of climbing or jumping (an additional 0.6 kcal for an average 20 kg dog to climb 4 meters during a trial). This is in addition to the cost of standing which is about half as much again as basal metabolic rate, i.e. 0.2 kcal for standing in addition to BMR of 0.5 kcal for a 60 s trial for an average 20 kg BW dog). Overall, therefore, this generous estimate of the energy required for each agility trial is very small (no more than 8.6 kcal for an average 20 kg BW dog or 0.4 kcal/kg BW above BMR) when compared to the total daily energy requirement of a dog

(720 kcal daily for BMR or 1200 kcal daily for maintenance for an average 20 kg BW dog in kennels).

It can be concluded, therefore, that an active agility dog probably requires only slightly more energy each day than an active dog in kennels, i.e., an agility dog is likely to require 120-160 kcal ME/kg BW^{0.75} daily, which is equivalent to 1100-1500 kcal daily for a 20 kg BW dog. This recommendation has a wide range because energy requirements are greatly affected by individual variation between animals and breeds, by age, neutering, ambient temperature and coat length. Energy requirements will also vary with the distance dogs travel during training. Rather than use a calculation to estimate how much an agility dog should be fed, therefore, it is best to adjust the amount fed to ensure that each dog maintains a lean body condition score (4-5 for most dogs or 3-3.5 for sight-hounds on a 9 point scale). A lean body condition is probably ideal because it will reduce the momentum that must be overcome when turning corners and will probably improve performance. It will also reduce pressure on joints and may extend the active life of those breeds of agility dogs which are prone to arthritis.

PROTEIN

Dogs use protein for making glucose for energy only after they have been running for about 30 minutes. As agility dogs run for less than a minute, they probably do not require increased protein for optimal performance. Only dogs undertaking endurance training would require more protein. Most commercial dry diets should contain enough protein but low protein therapeutic, 'lite' and 'senior' diets may not contain enough protein.

SOURCES OF ENERGY: CARBOHYDRATE VERSUS FAT

The energy for muscle contraction comes from (1) the high energy phosphate bonds in adenine triphosphate (ATP) and creatine phosphate stored in muscle, (2) from the metabolism of glycogen stored in muscle to glucose and then to lactic acid and pyruvate, or (3) from the oxidation of glucose and fat to carbon dioxide and water. The production of lactic acid from glucose does not require oxygen and is termed 'anaerobic glycolysis'. The oxidation of glucose and fat does require oxygen and, therefore, involves 'aerobic' metabolism.

High energy phosphate from ATP and creatine supports the highest rate of activity and provides much of the energy for acceleration but these substances are only present in small amounts and become rapidly exhausted. Anaerobic glycolysis supports the next highest rate of activity but build up of lactic acid in the blood limits the energy available from this source. Anaerobic glycolysis provides some of the energy required for fast sprints by Greyhounds but agility dogs do not run as fast. Anaerobic glycolysis is probably less important in agility dogs and it is unlikely that lactic acid should build up during an agility trial. Aerobic oxidation of glucose supports the next highest level of activity and provides some of the energy during long distance running. High speeds supported by glucose oxidation are only possible, however, while supplies of glucose from glycogen stored in muscle last. When supplies of glycogen are exhausted, only

lower speeds supported by fat oxidation are supported and athletes are unable to accelerate. The amount of glycogen in muscle can, therefore, affect stamina.

Unlike the other sources of energy, fat provides an almost inexhaustible source of energy. Dogs, in particular, are adapted to using the oxidation of fat as an energy source. All canine muscle fibers are able to use aerobic as well as anaerobic sources of energy and canine muscle, unlike human and cat muscle, does not contain type IIb fast twitch fibers that rely primarily on anaerobic sources of energy. Thus, glucose oxidation increases when dogs walk and run at low speeds but fat oxidation increases even more and provides most of the energy for sub-maximal aerobic exercise. Only when lactic acid builds up in dogs running very fast, such as Greyhounds, or in untrained dogs at lesser speeds, is fat oxidation less important. Aerobic metabolism of fat is likely, therefore, to provide most of the energy for agility dogs. Carbohydrate oxidation will, however, aid in supporting a higher rate of energy expenditure and a higher running speed than fat oxidation while glycogen stores last. The duration of an agility trial is so short that glycogen stores are unlikely to be depleted but should glycogen become depleted during several trials, fat oxidation would become the only available source of energy. Dogs may not be able then to turn, jump and accelerate as well as when glycogen was available.

WHAT TO FEED?

Dogs appear to run faster and have better stamina when fed a higher fat diet. Nevertheless, the ideal amount of fat that should be fed to agility dogs has yet to be determined. Very high fat diets have been reported to slow racing Greyhounds but increase the stamina of long distance running dogs. Low fat diets slow racing Greyhounds and decrease the stamina of long distance dogs. Intermediate (moderate to high) amounts of fat are, therefore, recommended for agility dogs. Inexpensive dry extruded dog foods are comparatively low in fat. These probably do not contain enough fat for agility dogs. More expensive dry dog foods tend to be higher in fat because fat is sprayed on to the kibble after it emerges from the extruder. These higher fat dry commercial diets probably provide enough fat for agility dogs. Alternatively, canned dog food can be added to the less expensive dry foods to increase the fat content of the diet. Training is important, however, to enable dogs to utilize the increased fat in their diet.

WHEN TO FEED

Dogs do not need to be fed immediately before intense exercise. They show better stamina when they have not been fed. Dogs should therefore be fed after exercise has finished for the day. Ideally, dogs should not undertake intense exercise less than 8 hrs after a small meal or less than 16 h after a large meal to allow for the meal to pass through the intestinal tract.

WATER

Water is more important than food to exercising dogs and should always be made available for drinking free choice. Water is essential for dogs to maintain normal body

temperature in high ambient temperatures and when exercising. Dogs keep cool by panting which increases their water requirements. Heat is generated during exercise and unless heat is lost to the environment, rectal body temperature tends to rise. This exercise-induced hyperthermia limits how long dogs can exercise. Body temperature increases more during exercise in dehydrated dogs, whereas free access to water minimizes exercise-induced hyperthermia and markedly improves stamina. Body temperature can increase explosively even in sedentary animals in a warm environment when they become more than 10% dehydrated and the problem is exacerbated during exercise. Dogs should, therefore, be offered free access to water before, between and after agility exercise to reduce the risk of hyperthermia and to improve performance. It should be emphasized, however, that dogs are at even greater risk of hyperthermia when they exercise in high humidity. Even well hydrated dogs cannot control their body temperature if the humidity is high because water will not then evaporate to allow cooling. Under these circumstances, dogs should be cooled with cold water or ice or kept in an air conditioned environment before and after exercise to maximize performance.

The amount of water required by an agility dog will be affected more by ambient temperature than the amount of exercise. The water requirement can more than double in a warm environment. The water requirement during exercise, however, increases with the amount of energy expended by approximately 1 mL/kcal. An agility dog needs very little energy to complete each trial so it should require very little additional water for each trial (≈ 0.4 mL/kg BW for each 200 m trial). This increase in water intake is trivial compared to daily water requirements (≈ 50 mL/kg BW daily).

GLUCOSE

Glucose (0.2 to 5 g/kg BW) given before, during or after exercise helps to maintain blood glucose during exercise, promotes more rapid repletion of muscle glycogen after exercise and reduces the increase in body temperature that occurs during exercise. It does not improve endurance performance but could improve recovery between bouts of exercise. Any benefits are likely to be small in agility dogs, however, because glycogen is probably not greatly depleted during exercise in these dogs. It is not recommended to add any other nutrient to drinking water as some have been shown to reduce performance or to have detrimental effects when added to the drinking water.

CALCIUM AND PHOSPHORUS

There is no evidence that the dietary requirements for calcium and phosphorus are any higher in dogs that exercise than in sedentary dogs or dogs undertaking normal amounts of exercise. The bones of exercising dogs are 10% heavier than those of confined dogs but this increase develops over a long time and extra calcium should not be fed to exercising dogs. Excess calcium supplementation increases the risk of osteochondritis dissecans in growing large breed and in exercising dogs. Supplements are not necessary, therefore, when dogs are fed a balanced diet containing approximately 3 g/mcal calcium and phosphorus.

Calcium may need to be added to unbalanced diets, such as home-made diets containing lots of meat, however. Feeding foods without adequate calcium supplementation, such as meat can result in poor bone mineralization and increased risk of fractures in dogs.

SALT

Loss of salt in sweat does not occur in dogs as it does in humans during exercise. Sodium and potassium losses in saliva and urine are comparatively small. Most commercial diets contain adequate amounts of sodium, chloride and potassium ($\approx 1\text{g/Mcal ME}$) but very low sodium diets are not recommended.

Addition of salt to the drinking water is unnecessary and is not recommended. The osmolality (water absorbing tendency) of the blood increases in dogs when they become dehydrated. Dogs drink to correct this hyper-osmolality. Adding salt increases the osmolality of the drinking water and prevents correction of this hyper-osmolality. Dogs will, therefore, continue to crave and drink water when given salt-containing water, whereas dogs satisfy their deficit with one episode of drinking when given water to drink without any salt added.

TRACE MINERALS AND VITAMINS

There have been no studies of the effect of exercise on vitamin and trace mineral requirements in dogs. Vitamin B1 and B6 requirements probably increase in proportion to energy and protein consumption, respectively, but agility dogs require little extra energy or protein. Balanced commercial diets designed for dogs undertaking only moderate amounts of exercise contain plenty of vitamins and minerals and additional supplementation is almost certainly not required when agility dogs are fed such diets. Unbalanced home cooked diets may, however, require supplementation.

Antioxidants

Oxidation reactions are an essential part of normal physiological processes but reactive oxygen containing substances and free radicals have the potential to cause oxidative damage to the fat in cell walls, to proteins and to DNA. The production of reactive oxygen containing substances and free radicals increases and the concentration of antioxidants in blood decreases during exercise. This may represent an oxidative stress. Nevertheless, this oxidative stress has not as yet been shown to cause any pathology in trained dogs during exercise. Furthermore, supplementation of antioxidant nutrients such as vitamins E, C or retinoids, have mostly failed to show any benefit in exercising dogs, whereas very high daily doses of vitamin E (35 IU vitamin E/kg body weight) and vitamin C (35 mg/kg body weight) slowed racing Greyhounds. Currently, therefore, there is no evidence that the requirement for antioxidant nutrients is greater in exercising dogs than in sedentary dogs and supplementation of agility dogs is probably not necessary if they are fed a balanced commercial diet. Nevertheless, the production of reactive oxygen species during exercise is increased during exercise in untrained dogs and there have been no studies examining the effect of antioxidant supplementation in untrained dogs. It

is possible, therefore, that there may be more benefit from antioxidant supplementation of the diet in untrained dogs that exercise only at weekends.

OTHER 'ERGOGENIC' NUTRIENTS

Arginine, tryptophan, aspartate, L-carnitine, creatine, dimethyl glycine, pangamic acid (vitamin B₁₅), inosine, co-enzyme Q, bee pollen, methylsulfonylmethane, caffeine, alcohol and vinegar have all been suggested to improve the performance of exercising dogs. To date, there is no evidence to support the addition of any of these substances to the diet of agility dogs or dogs undertaking any other form of exercise. Arginine, tryptophan and aspartate are all amino acids and there is no data to suggest that requirements for these amino acids increase more than the requirement for protein in exercising dogs. L-carnitine and creatine are synthesized by dogs and there is no evidence that this synthetic capacity is inadequate in exercising dogs. Addition of creatine to the diet, for example, does not appear to increase creatine concentrations in the muscle of exercising dogs as it does in some humans. The improvement in performance of racing Greyhounds given dimethylglycine and diisopropylammonium dichloroacetic acid can probably be attributed to dichloroacetic acid. Dichloroacetic acid is a potent drug which markedly curtails lactic acid production but dichloroacetic acid also has many toxic side-effects and a safe dose has not been established. Glucosamine, green lipped mussel, or chondroitin sulfate supplementation may be beneficial, however, in dogs with degenerative joint disease and dental chews may help to reduce the build up of dental tartar.

SELECTED SPECIFIC MUSCLE, TENDON AND LIGAMENT ABNORMALITIES AFFECTING DOGS

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INFRASPINATUS MUSCLE CONTRACTURE

This is an uncommon yet very characteristic lameness which typically occurs in hunting dogs. The history often includes a sudden onset of lameness sustained during vigorous exercise. The dog develops pain in one shoulder and is reluctant to bear weight on the affected limb. The lameness usually subsides in one or two weeks. Then several weeks subsequent to the initial episode a persistent lameness develops which is characterized by external rotation of the humerus. The lameness which develops several weeks after the original injury is not painful and results from fibrosis of the infraspinatus muscle.

The clinical signs that characterize the syndrome include:

Standing hyperextension of the limb

Adduction of the elbow

Abduction of the paw

Restricted shoulder movement
Marked scapular muscle atrophy
Lateral circumduction of the distal forelimb with a "carpal flip" while ambulating

EMG studies have shown that infraspinatus contracture is more likely due to a primary myopathy rather than a neuropathy. Histological findings show degeneration and atrophy of skeletal muscle with subsequent fibrosis. The possible pathogenesis may involve a traumatic event causing incomplete rupture, leading to fibrosis and contracture.

Treatment is by infraspinatus tenotomy. The approach is made just cranial to the deltoid muscle. Caudal retraction of the deltoid muscle allow visualization of the tendon of insertion of the infraspinatus muscle. Surgery offers an excellent prognosis and is generally curative.

BICIPITAL TENOSYNOVITIS

This is an inflammatory condition affecting the bicipital tendon and tendon sheath within the intertubercular groove of the humerus. It is assumed to be the result of a chronic strain or trauma. Disruption of the fibers of the tendon are typically observed on arthrography and/or ultrasound and at arthroscopy or arthrotomy. This results in synovial hyperplasia, chondromalacia of intertubercular groove, osteophytosis (particularly at the tendon's origin on the supraglenoid tubercle), and possibly calcification of bicipital tendon. Dogs usually present with a long standing history of chronic lameness. Pain is elicited when local pressure is applied to the tendon and associated tendon sheath.

Initial treatment usually consists of limiting exercise and oral administration of anti-inflammatory drugs. An intra-articular injection of repository steroids (20 mg of depomedrol which potentially can be repeated in 4 weeks) may be tried if lameness persists. Surgical transposition of the bicipital tendon to the greater tubercle of the humerus has been advocated in chronic cases refractory to medical management. Some recent reports suggest the tendon may simply be transected and released via arthroscopy. Approximately half of the affected dogs are reported to respond to medical management and the majority, but not all of the dogs that are refractory to medical management, are reported to improve following surgery.

CARPAL HYPEREXTENSION INJURIES

Hyperextension injuries result in chronic subluxation of the carpus and is the most common form of carpal instability. The stability of the carpus is dependent on both the integrity of the complex system of carpal ligaments and the joint capsule. Traumatic injuries typically cause an acute non-weight-bearing lameness which may progress with time to a partially weight-bearing lameness. Other dogs develop hyperextension as a result of chronic degenerative changes. If the dog is weight-bearing the stance is usually palmigrade. Radiographs are indicated to detect concurrent fractures and assess the extent of degenerative joint disease. "Stress radiographs" are indicated to define the level(s) and extent of the instability. Instability can be to an antebrachiocarpal, intercarpal and/or

carpometacarpal joint. The level of instability has important implications when surgical intervention is done. Treatment options include:

- Splinting may be attempted initially but is rarely successful. The carpus is placed in flexion to relieve the stress on the healing supportive structures. Coaptation is maintained for 4-8 weeks.
- Primary open repair of ligamentous structures is described, but is rarely attempted due to the difficulty in identifying and repairing specific short and long ligaments (and the joint capsule). Numerous techniques have been described utilizing various autogenous and synthetic prostheses.
- Resolution of lameness often requires arthrodesis. Pancarpal arthrodesis is generally done; however, if instability is isolated to one level (antebrachiocarpal, intracarpal or carpometacarpal) as defined on stress radiographs, then a selective partial carpal arthrodesis can be done. Plates are most often used to stabilize pancarpal arthrodeses. The plate is placed on the dorsal surface of the carpus because it is readily accessible. This places the plate on the compression, rather than the tension, surface of the bone. Thus, the plate is generally protected for 1-3 months, with an appropriate form of external coaptation. It is preferable to stabilize carpometacarpal arthrodeses with small transarticular pins placed in retrograde fashion from the metacarpal bones. If a plate is used to stabilize partial (intracarpal-metacarpal) arthrodesis, the plate tends to interfere with radiocarpal joint function. Again, the fixation is generally protected with a coaptation splint for 1-3 months following surgery. Dogs have a slightly altered gait, but can often return to working or competition following successful arthrodesis.

GRACILIS/SEMITENDINOSUS MYOPATHY

Myopathy of the gracilis and semitendinosus muscles has been reported as an infrequent cause of hindlimb lameness in dogs. Myopathy of the gracilis and semitendinosus muscles is a reported cause of hindlimb lameness in dogs. Nearly ninety percent of the dogs reported with this condition are German or Belgian Shepherds with young adult male dogs most often affected. Bilateral involvement is reported in 20-39% of affected dogs. Although the gracilis muscle is most commonly affected, the semitendinosus muscle can also be affected. Ipsilateral gracilis and semitendinosus muscle involvement has been reported in two dogs and contralateral involvement of the gracilis and semitendinosus muscles in another dog.

The lameness associated with myopathy of either the gracilis or the semitendinosus muscle is distinctive and has a similar appearance regardless of which muscle is affected. The two muscles have similar insertions and functions, differing primarily in their origins. The gracilis muscle arises from the pelvic symphysis, while the origin of the semitendinosus muscle is located more lateral on the caudoventral portion of the ischiatic tuberosity. In the caudomedial stifle region, both muscles have broad flat tendons which pass over the head of the medial gastrocnemius muscle in the popliteal space and insert on the cranial border of the tibia. Both muscles also have distal aponeuroses which extend from their caudal borders, blending with the crural fascia and contributing a well-

developed reinforcing band to the calcaneal tendon. The distinctive gait abnormality is the result of functional shortening of the affected muscle which limits abduction of the coxofemoral joint and extension of the stifle and hock. On physical examination it is possible to determine which muscle is affected by carefully palpating the proximal location of the taut muscle band: the semitendinosus muscle originating more lateral on the ischiatic tuberosity, while the origin of the gracilis muscle is located more medial on the pelvic symphysis.

The lameness associated with this condition has been described as strictly mechanical, although nearly half the owners of dogs in one study noted that their dog's activity level decreased as the lameness progressed and increased following surgery, suggesting that the lameness may, at least in some dogs, have some associated pain. This is supported by the observation that digital pressure exerted on the affected muscle(s) and abduction of the ipsilateral coxofemoral joint elicited a pain response in the majority of dogs in one study.

Although descriptions of the gross pathologic abnormalities observed is often limited by the extent of the muscle exposed at surgery, the distal myotendinous portion of the affected muscle consistently was thickened and fibrotic. The distal myotendinous portion of the affected muscle can be visually apparent and is palpable as a thick taut band located on the medial aspect of the stifle. Increase in the cross sectional area of the distal myotendinous portion of the affected muscle can also be apparent on radiographs and ultrasound. In several dogs in which the entire length of the muscle was examined at surgery or necropsy, a thick fibrous band was associated with the caudolateral border of the gracilis muscle. The fibrous band had the gross and histologic appearance of a tendon with its origin on the pubic symphysis and merging with the thickened tendon of insertion of the gracilis muscle proximal to the stifle. Necropsies of several normal dogs have failed to reveal an analogous structure. We feel this band is a fibrous remnant of the caudal portion of the gracilis muscle. The remaining cranial portion of the gracilis muscle can have a normal or slightly mottled appearance.

Several theories have been proposed regarding the etiopathogenesis of this condition: 1) a single traumatic event or the end result of repeated microtrauma; 2) an autoimmune process; 3) a primary neuropathy; and 4) an ischemic phenomenon based on the perivascular distribution of myofiber degeneration and fibrosis in several dogs. The exact etiopathogenesis, however, has yet to be determined.

Any procedure which disrupts the continuity of the affected muscle (simple transection, partial excision, and complete resection) results in immediate improvement in abduction of the coxofemoral joint, increased extension of the stifle and hock, and temporary resolution of lameness. Recurrence of lameness following surgery; however, has been reported in all dogs with adequate follow-up evaluations. Recurrence of lameness is the result of fibroplasia in the wound reuniting the severed ends of the affected muscle. Adjunctive surgical procedures (inverting the incised margins of the muscle or tendon, or implanting free autogenous fat grafts in an attempt to prevent scar tissue from reuniting the severed segments), and/or administration of glucocorticoids or lathyrogenic agents

(D-penicillamine and colchicine) following surgery have not prevented recurrence of lameness. Although there is no known effective treatment, affected dogs appear to be very functional pets and some affected dogs continue to work or compete.

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Osteosarcoma: The Nemesis of Large Breed Dogs

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The format of this seminar will take the shape of frequently asked questions and answers we encounter when dealing with clients at the Veterinary Medical Teaching Hospital.

To start, what is an Osteosarcoma?

Osteosarcoma is a cancer of bone, which has its origin in the bone forming cells called osteoblasts.

What causes these cells to become cancerous?

A number of reasons have been found. However, essentially osteosarcoma is a genetic disease and the events that lead up to the cell becoming cancerous are related to changes in the genetic structure of the cell. These changes can be due to inherited causes (e.g. certain large breeds of dogs are prone to this cancer), environmental causes (e.g. radiation), and biological causes (e.g. viruses).

Is it malignant?

By malignant we mean cancer spreading to the local gland and /or other organs. The osteosarcoma is considered a highly malignant cancer with about 100% mortality if not treated. The osteosarcoma cells have genetic changes, which allow them to escape from the “mother cancer” and invade distant sites. The cancer cells use enzymes called “matrix metalloproteinase’s” or MMP’s to invade the healthy tissue. These enzyme digest membranes and connective tissue allowing invasion. The cells also have the ability when

they reach a certain size to recruit blood vessels to help them grow bigger; we call this “angiogenesis.” Most deaths from malignant cancer, even though the “mother cancer” is treated successfully, are because of spread to distant organs. In the case of osteosarcoma, that organ is the lung; other organs can also be affected but less often.

What breeds are affected and what is the risk to my dog?

We use three terms to describe the occurrence of a cancer, these are: incidence, prevalence and risk. By incidence, we mean the number of new cases occurring during a specific time interval such as one year. By prevalence, we mean the total number instances of old or new cases in one year. By risk, we mean a risk factor that increases the likely hood of osteosarcoma. We know from research that large and giant breeds of dogs weighing greater than 40kg (88lbs) accounted for 29% of osteosarcoma cases. Dogs less than 15kgs account for less than 5% cases. It is obvious from the data that weight is certainly a risk factor for developing osteosarcoma.

Another obvious risk factor would be breed; Table 1 lists the dogs at risk although weight appears to be more important. Other less important risk factors include: surgical implants, chronic bone infections. The incidence in the USA is estimated to be about 8,000 new cases per year. This could be higher as most are not reported. True prevalence is unknown.

How will I know my dog has osteosarcoma and where will it most likely occur?

The most important sign is bone pain. If the legs are affected the early sign are generally lameness, this can even precede

radiographic changes of osteosarcoma. Follow-up X-rays for a non-resolving lameness are very important especially in large breeds. Approximately 75% of cancers occur on the long bones of the legs. In the back leg, the most common sites are around the knee (stifle), and some around the hock. In the front leg, the shoulder area (proximal humerus) and the distal radius (corresponds to the wrist area in humans) are the most common sites. The tumor in the more advanced stage is recognized a a swelling over the affected area that is painful and feels “hot to touch”.

Breeds	% Incidence for the breed
Irish Wolfhound	4.88
Rottweiler	4.80
Greyhound	4.27
Great Dane	4.26
Irish Setter	3.95
Samoyed	3.39
Akita	1.57
Doberman Pinscher	1.47
Siberian Husky	1.38
Golden Retriever	1.11
Labrador Retriever	0.94
Basset Hound	0.79
Australian Shepherd Dog	0.46
Mixed Breed	0.44
Boxer	0.32

¹ Data compiled from 1996-2004 case records seen at the UF VMTH and analyzed by Julie Rosenberger (Class of 2007) and Dr Cynda Crawford, University of Florida

To make the diagnosis, veterinarians need to do radiographs (x-rays) of the affected bone and the lungs. The cancer looks very typical on radiographs, but to confirm the diagnosis the vet must do a biopsy of the tumor. At the University of Florida, we typically use a biopsy needle called a Jamshidi. The biopsy does require an anesthetic as the procedure is painful and require good pain medication at this time as the cancer pain seems to flare up after the biopsy. The diagnosis is then confirmed by a pathologist. It is not uncommon for a biopsy to come back negative and the biopsy needs to redone. It is a delicate balancing act between taking enough tissue for diagnosis, but not so much that the bone is weekend further. X-rays of the lungs are vital to identify spread of the cancer to the organ, caution should be noted here as x-rays only start picking up spread after the cancers reach a certain size (about 2mm in diameter).

So you have given me this terrible news what are the treatment options for my dog?

When we treat osteosarcoma in our pets we generally have two goals in mind. The first, which is more by way of a question, can the cancer be cured? Generally, most malignant cancers in dogs cannot be cured and osteosarcoma is no exceptions. If that is the case, our next step would be to provide palliative care. With osteosarcoma we generally focus on two aspects of palliative care, the first being pain control. Effective treatment in the early stages of the cancer is to put the dog on an anti-inflammatory drug such as carprofen, deracoxib, or piroxicam, or other non-steroidal anti-inflammatory. These drugs should not be combined with cortisone (prednisone) or other steroids as they can lead to severe gastrointestinal bleeding. At the time of biopsy, additional narcotic painkillers are also needed. Probably the most effective method in controlling pain is to amputate the leg. In a sizable number of cases this choice is not exercised because of medical and/or owners preferences. In that case what we do radiation treatment of the primary cancer, or limb sparing surgery (replacing the affected bone with a surgical implant or prosthesis), or a radiopharmaceutical treatment. Owners are also justified in considering euthanasia as osteosarcoma is a very painful cancer and cure is unlikely.

The second aspect of palliative care is to treat the primary cancer and suppress spread. A

number of the treatments for pain (e.g. limb sparing, amputation and radiation) form the basis of primary tumor control. Choices for treatment must take into account cost versus benefit, stage

Treatment	% Alive 1 year	% Alive 2 years
Amputation <u>without</u> chemotherapy	10%	
Amputation with chemotherapy	30-62%	7-21%
Limb sparing with chemotherapy	63%	
Radiation (SRS) and chemotherapy (UF) ²	50%	38%

of the cancer, owners concerns, and expertise of the clinician. Table 2 gives a list of survivals for various treatment types. The purpose of chemotherapy adding to primary treatment such as surgery or radiation is to control distant spread, which in most cases is to the lungs. Chemotherapy is only moderately effective in delaying the spread of the cancer.

² Early not published data

What's in the pipeline for new treatments?

At the University of Florida, we are continuing our research in stereotactic radiosurgery (SRS), see Table 2. It promises to be a viable alternative to amputation. We have found that pain control is good and that long-term control of the primary tumor is possible. Other areas of research include blocking MMP's and angiogenesis to slow down secondary spread of the cancer to vital organs such as the lung. In addition the use of bisphosphonates (human drugs used to treat osteoporosis) seem promising in controlling bone pain and preventing spread of the cancer. Arguably, the most important development will be an immune stimulant or vaccine that will help the body block the spread of the cancer to vital organs.

Diagnosis and Management of Urinary Incontinence

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Key points

Urinary incontinence is a common problem, occurring with an incidence of 10-20% of female dogs after ovariohysterectomy.

Complete diagnostic evaluation of urinary incontinence involves imaging studies to rule out anatomic abnormalities and functional studies to rule out sphincter mechanism incompetence.

Medical therapy with alpha adrenergic agonists is successful in approximately 70% of dogs with urinary incontinence.

Current methods for surgical therapy are highly successful in the short term, but long-term success has been unsatisfactory.

Introduction

Urinary incontinence occurs with alarming frequency in dogs, particularly in spayed females. Retrospective studies have reported that the incidence of urinary incontinence in dogs following ovariohysterectomy ranges from 13.6% to 20.1%. The significance of urinary incontinence in an indoor pet cannot be underestimated, as the problem can often lead to euthanasia due to repeated house soiling. Thus, urinary incontinence is a common problem with serious consequences; it is our responsibility as veterinarians to be familiar with its diagnosis and treatment.

Causes for urinary incontinence may be divided into two categories: anatomical abnormalities and functional abnormalities. The anatomical abnormality that is most often associated with urinary incontinence is ureteral ectopia. Suspicion of ectopic ureter is highly dependent upon history and physical examination of the animal, as this problem occurs almost exclusively in female dogs and is characterized by constant dribbling of urine since birth. Functional abnormalities causing urinary incontinence may be congenital or acquired, but most commonly involve urethral sphincter mechanism incompetence (USMI), otherwise known as “hormone responsive incontinence”. In contrast to ureteral ectopia, USMI typically occurs in middle-aged spayed female dogs and is characterized by intermittent incontinence during recumbency and sleep. Other factors such as polyuria/polydipsia, urinary tract infections and vaginal strictures can exacerbate pre-existing incontinence, but in my opinion, are rarely primary causes.

One purportedly simple method of diagnosing USMI is by response to treatment (typically with phenylpropanolamine, see later section). This method is not as simple as it seems, because a lack of response to treatment does not rule out USMI- up to 30% of dogs do not respond to pharmacologic therapy. In addition, many dogs require dosage adjustments and up to 4 weeks of drug therapy before a response is noted. Due to these issues, I believe that it is preferable to perform a proper diagnostic evaluation before pursuing therapy.

Diagnostic evaluation of urinary incontinence is focused primarily on identifying these anatomical or functional anomalies. Anatomic abnormalities may be identified through a number of imaging techniques. Initial screening examination is typically performed by use of survey radiography or abdominal ultrasonography. Abdominal ultrasound findings consistent with ureteral ectopia include hydronephrosis or hydroureter on the affected side. In chronic cases, parenchymal and renal pelvic changes may be suggestive of pyelonephritis. Intravenous contrast urography is the classic method for identification of ectopic ureters. However, the technique is time consuming and can be difficult to interpret. We have switched to the use of computerized tomography (CT) after intravenous contrast administration. This technique is less labor intensive than performing staged radiographs, does not require removal of feces from the colon and allows specific localization of the entry point of ectopic ureters. Disadvantages of CT excretory urography are that it has limited availability, adds to client expense and requires general anesthesia. A final method for definitive identification of ectopic ureters is direct visualization via cystoscopic evaluation. This technique is considered the “gold standard” and has high sensitivity and specificity, but requires expensive equipment and a high skill level to obtain a complete examination of the urinary tract.

If anatomic abnormalities are ruled out, functional evaluation of the continence mechanism may be performed. Urethral pressure profilometry (UPP) is a method for quantification of the pressure exerted by the urethra as a catheter is placed into the bladder and slowly withdrawn out of the urethra. Urinary continence is dependent upon maintenance of a maximal urethral pressure that exceeds intravesicular pressure, thereby resisting the leakage of urine. Documentation of decreased maximal urethral pressure

remains one of the few methods to objectively diagnose USMI in dogs. Unfortunately, equipment for performing UPP is expensive (\$10,000 to \$25,000) and results vary depending upon the position of the animal, size of the catheter used, speed of withdrawal, rate of fluid infusion and the use of sedatives or analgesics. Another technique related to UPP may correlate more closely with clinical incontinence by evaluating the entire continence mechanism. This test, called leak point pressure, involves placement of a catheter into the bladder or rectum to allow measurement of the intravesicular or intraabdominal pressure. The abdomen may then be compressed until leakage occurs from the vulva. The pressure at which leakage occurs is termed leak point pressure. Currently, these urodynamic tests are performed only in large referral institutions and the value of their application in practice is limited by difficulties in standardizing results between institutions.

The pathophysiology of USMI in spayed female dogs has been the focus of many research studies, though it remains controversial. Two mechanisms are likely to contribute to incontinence after ovariohysterectomy. One factor that has been implicated is caudal movement of the urogenital tract that occurs after ovariohysterectomy. The normal position of the bladder and proximal urethra is intra-abdominal so that increased abdominal pressure causes compression of both the bladder and urethra, preventing urine leakage. If the bladder moves caudally, the urethra may enter the pelvic canal (pelvic bladder) and intra-abdominal pressure will be transferred only to the bladder, predisposing to urine leakage. A second contributing factor may be the hormonal changes that occur after ovariectomy. Estrogen is thought to sensitize the smooth muscle of the proximal urethra to the effects of catecholamines, increasing urethral tone. Certainly, many dogs improve when treated with estrogen replacement. However, spayed dogs with USMI have similar estrogen levels to spayed dogs without USMI and hormone replacement is not 100% effective in curing incontinence in all dogs. Thus, a combination of predisposing anatomical and hormonal factors may contribute to USMI after ovariohysterectomy.

Pharmacologic therapy is the mainstay for treatment of urinary incontinence in dogs. The most commonly used agent is the alpha agonist phenylpropanolamine or PPA. This drug has an efficacy of approximately 70% in resolving urinary incontinence due to USMI in female dogs. Side effects of restlessness and anorexia are somewhat predictable for a sympathomimetic drug and are dose related. The effects of life-long administration of PPA have not been evaluated and the drug has not been consistently available. Despite these issues, PPA is currently the first line therapy for USMI in dogs due to its efficacy and minimal side effects.

Estrogenic compounds have also been demonstrated to have some efficacy in the treatment of USMI. Unfortunately, estrogens are associated with a low rate of serious side effects, such as prolonged bone marrow suppression, endocrine dermatopathies and reproductive disorders. Due to these side effects, Estrogen is not typically used unless a dog has proven non-responsive to PPA therapy, or PPA is not available.

Tips for pharmacologic therapy:

I begin with PPA at a dose of 1mg/kg BID

In dogs that are refractory to initial dose of PPA, I increase the dose to 1mg/kg TID
In dogs that do not respond to PPA given three times per day, estrogenic compounds (Diethylstilbesterol) may be used in combination with PPA and may have synergistic effects.

Surgical intervention is an option in dogs that fail to respond to pharmacological therapy. Techniques described in dogs with USMI are similar to those used in human beings and have accomplished static resistance to urine leakage by altering the anatomic position of the bladder neck and pelvic urethra, by urethral bulking, or through urethral sling procedures. The most commonly used surgical technique, colposuspension, was adapted from a technique used in women with hypermobility of the urogenital tract. Sutures are placed into the vaginal wall through a caudal midline abdominal approach and are passed cranially and ventrally around the prepubic tendon, pulling the urogenital tract into the abdomen. Though colposuspension has good short-term efficacy, surgery alone has produced poor long-term results, with restoration of continence in only 14-56% of dogs in large clinical studies. Many dogs that do not respond to surgery will benefit from continued administration of PPA.

Summary

Urinary incontinence is a common and serious problem in dogs. Primary rule outs include ectopic ureter and urethral sphincter mechanism incompetence. Diagnostic evaluation should include both imaging and functional studies. Pharmacologic therapy is successful in the majority of dogs and surgery is only offered in cases that are refractory to phenylpropanolamine. Surgery is simple to perform and has a high initial success rate, but incontinence will often recur within the first year.