Dog Owners and Breeders Symposium
July 28, 2001
University of Florida
College of Veterinary Medicine

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Dilated Cardiomyopathy in Dogs

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General

Dilated cardiomyopathy (DCM) is a very common form of heart disease in dogs. Only degenerative valve disease (also called mitral valve disease or endocardiosis or mitral insufficiency) and in some places heartworm disease are more common. It was not until the 1970s, when echocardiography began to be performed in veterinary institutions, that dilated cardiomyopathy could be diagnosed non-invasively with any certain degree of accuracy. The reported prevalence in dogs is approximately 0.5 percent.

Based upon a national database of dogs presented to veterinary schools in North America, we know that 5.8 percent of the Doberman Pinschers that were seen at a school had DCM, 5.6 percent of the Irish Wolfhounds presented had DCM. 3.9 percent of the Great Danes, 3.4 percent of the Boxers and 2.6 percent of the St. Bernards. The prevalence in purebred dogs in 0.65 percent whereas the prevalence in mixed breed dogs is 0.16 percent. In general, the dogs are middle age (4-10 years of age, with the exception of the Portuguese Water Dog (0.5 – 8 months)). Males are over-represented (2:1). Seventy to 80 percent of the Dobermans with congestive heart failure due to DCM are male and in a recent paper describing the disease in Dalmatians, all of the affected animals were male. This does not mean that female dogs are not affected by DCM, on the contrary, Dr. O'Grady at the University of Guelph found no sex predilection for Dobermans with subclinical (Asymptomatic or occult) DCM.

In most of the dogs that eventually die of DCM, the heart is very enlarged and appears flabby on examination. However, regardless of how symptomatic the dogs were before they died or how abnormal the heart looks upon "gross inspection," the microscopic appearance of the heart is not often very dramatic. So the microscopic appearance belies the severity of the problem.

There are many proposed reasons for DCM including viral infection, toxins, nutrition, small blood vessel disease, genetic abnormalities, etc., but there is not likely to be one cause for the disease in all affected dogs.

Familial and Genetic Influences

The general feeling is that most cases of DCM have a familial or genetic basis. This is based upon the observation of a higher prevalence in purebred dogs, in certain breeds and within certain families. However, in only rare instances have the genetics for the disease been described. For example, Boxers with cardiac arrhythmias have an autosomal dominant trait. There are also strong indications in other breeds such as the Doberman, where DCM can affect more than 50 percent of family members. In Portuguese Water Dogs DCM appears to be autosomal recessive and in English Cocker Spaniels it is associated with a particular immune deficit. An autosomal dominant mode of transmission has been proposed for the Newfoundlands with DCM.

Nutritional Influences

L-Carnitine L Carnitine deficiency has been identified in some dogs with dilated cardiomyopathy. Unfortunately we know from animal models that carnitine deficiency can be created by making the heart beat at very rapid heart rate (using a pacemaker); we also know that very rapid heart rates are common in dogs with DCM. Therefore the carnitine deficiency that was reported in earlier years may be the result of, rather than the cause of, DCM. So while we had all hoped that carnitine supplementation would resolve or prevent DCN in dogs, unfortunately it hasn't. Many people choose to supplement L-carnitine in the diets of dogs with DCM and it is not likely to cause them any harm, but it is costly and may not be of any real benefit. Boxers with DCM should get 2-3 grams every 12 hours. If you want to supplement a Cocker Spaniel with DCM, the recommended dose is 1 gram of L-carnitine every 12 hours. **DO NOT GIVE D-CARNITINE OF D,L-CARNITINE.**

Taurine In the late 1980s taurine deficiency was recognized as the cause of dilated cardiomyopathy in the majority of cats with DCM. Since then investigators in the field have taken a close look for taurine deficiency in dogs with heart disease. In a study of dogs with DCM, 13 of 76 dogs had low taurine concentrations and this included three of three Cocker Spaniels with DCN and four of six Golden Retrievers with DCM. Dobermans on the other hand had normal taurine concentrations. When Cocker Spaniels (in a larger study) were supplemented with taurine **and** L-carnitine, in MOST but not all cases, their heart function improved. Subsequent to this report some people have stopped using the L-carnitine and still had success in improving heart function in some cases. Once blood is drawn for taurine evaluation, Cockers with DCM should get 500 mg TAURINE every 12 hours.

Very recently investigators in Scotland have determined that many Newfoundlands with DCM had low plasma taurine concentrations. At this point they have not described whether supplementation of these dogs resulted in any improvement of clinical signs.

CoEnzyme Q There have been several studies of the use of CoEnzyme Q in human patients but none have been published in the veterinary literature. Some of the studies in humans have shown some benefit while others have shown no benefit. It is not likely that a CoEnzyme Q deficiency is the CAUSE of DCM in dogs but it is an antioxidant and is not likely to be harmful to dogs with heart disease. Unfortunately it is expensive and since there is no proven benefit, it is hard to recommend an appropriate dose. The current doses that are reported are not based upon any scientific evidence.

Breed Specific Differences

Doberman Pinschers People have known about sudden cardiac death in Dobermans since the 1940s. Most of the early male dogs imported into the US to form our current breeding stock died suddenly, most likely due to DCM.

DCM in Dobermans is manifested in several ways. The dogs may have arrhythmias or congestive heart failure as their primary presenting problem. Either of these may lead to death of the dog (arrhythmias (ventricular tachycardia, VPCs) are common in Dobermans who develop

symptoms for dilated cardiomyopathy. These arrhythmias are found in virtually 100 percent of the Dobermans that die from DCM. These arrhythmias can also be seen in dogs that are not symptomatic for DCM at the time (but these dogs often later on develop DCM). In one study, about 13 percent of Asymptomatic Dobermans had arrhythmias on an electrocardiogram.

Because of the high prevalence of this disease in Dobermans, several investigators have evaluated Dobermans who are not symptomatic in an attempt to define the early stages of the disease. Many of the Dobermans examined that did not have any symptoms of heart disease (e.g. the subclinical symptoms), were in fact found to have heart dysfunction. Despite these subclinical dogs not having symptoms at the time of their initial examination, 30 percent went on to develop heart failure or died suddenly, presumably due to arrhythmias. Based on this study, echocardiographic criteria have been developed to help identify Dobermans that are at substantial risk for developing symptoms. These criteria are:

- The internal diameter of the left ventricle during relaxation (LVIDD) > 46 mm
- The internal diameter of the left ventricle during contraction (LVIDS) > 38 mm

The current thought is that DCM is a slowly progressive disease in Dobermans that worsens over a period of two to four years eventually leading to more severe arrhythmias and in many cases, congestive heart failure. Once the dogs become symptomatic, standard therapies for treatment of the heart failure and/or cardiac arrhythmias should be instituted.

Nonetheless, the prognosis for Dobermans with DCM is poor. One study reported the **average** survival time is approximately three months once clinical signs of heart failure are seen. Another study reported the **average** survival time of 2.4 months.

Boxer Cardiomyopathy in Boxers was first described by Dr. Neil Harpster in the early 1980s. He defined three types. Type I was the Boxer with arrhythmias that was not symptomatic. Type II was the Boxer that had arrhythmias that was symptomatic (usually collapse) and Type III was the Boxer with congestive heart failure that may also have arrhythmias. Since then numerous Boxers have been evaluated for the disease and we now recognize that most of the Boxers fit into either the Type I or Type II categories. This has prompted some people to call the disease *Boxer Arrhythmia Disease* or *Boxer Dysrhythmia* rather than dilated cardiomyopathy. Cardiac arrhythmias (ventricular tachycardia, VPCs) are the common arrhythmia seen in the Boxers with DCM.

The disease in Boxers is now known to be an autosomal dominant trait with variable penetrance3. Most of the dogs on initial examination will have a normal physical examination but in some cases an arrhythmia is detected and in rare cases a murmur is heard. Most of the dogs (~ 90 percent) will have a normal echocardiogram (hence the reason for the suggested name change).

Holter monitoring (continuous ambulatory electrocardiography) is the good method to examine Boxers for the disease. Since we know normal dogs rarely have ventricular arrhythmias, finding of frequent arrhythmias suggest cardiac disease. What we do not know at this point is whether the information about the frequency or rate of arrhythmia we observe on the Holter recording will help establish an idea of severity or prognosis.

Irish Wolfhound Ventricular arrhythmias are not common in the Irish Wolfhound however arrhythmias originating higher up in the atria are common (atrial fibrillation). This is seen in up to 80 percent of the Irish Wolfhounds with DCM.

In a large study in Germany, 500 Irish Wolfhounds were examined. Abnormal cardiac findings were seen in 42 percent (n=209) of the dogs. Dilated cardiomyopathy was diagnosed in 24 percent (n=121) and was subclinical in about half these. Atrial fibrillation was present in 88 percent of the dogs with DCM. The median survival time was 5.1 months.

Cocker Spaniel Both English and American Cocker Spaniels have been reported to develop DCM. Some of them have low plasma taurine concentrations and show improvement with taurine supplementation (as well as other standardized treatment for heart failure). All Cocker Spaniels with DCM should have plasma taurine concentrations evaluated and while waiting for results the dogs should be supplemented. In some but not all cases, the other cardiac medications can eventually be discontinued.

In one breeding of English Cocker Spaniels there was a strong association between the DCM and a particular immune defect (C4 phenotype).

In a study where American Cockers with DCM that were supplemented with taurine and carnitine, dogs < ten years of age lived an average of 46 $\pm$ eleven months whereas dogs > ten years of age lived an average of 14 $\pm$ seven months.

Portuguese Water Dog There has been a report of twelve related Portuguese Water Dogs with DCM. What is unique in this case is that the dogs were young (average thirteen weeks). The dogs died quickly of congestive heart failure. An autosomal recessive pattern of inheritance is suspected.

Dalmatians All nine of the affected dogs in one study were male with an average age of 6.8 years. Eight of the nine dogs had been fed a low protein diet to prevent bladder stones. The presentation of the dogs was classic for congestive heart failure and the median survival was ten months. Three of the dogs were supplemented with carnitine without any substantial improvement. Four dogs were tested for taurine deficiency and all were normal. Six of the nine dogs were taken off the protein restricted diet; one improved. The report does not state the eventual outcome of this dog.

Great Danes Great Danes with dilated cardiomyopathy often present in congestive heart failure, with or without cardiac arrhythmias. Like the Irish Wolfhound, atrial fibrillation is the most common arrhythmia seen in Great Danes with DCM. Based on recent pedigree analysis, DCM in this breed is believed to be an X-linked recessive trait. So affected dogs should not be bred and males borne to affected females are at increased risk of developing DCM.

Clinical Signs:

- **NONE**
- **Fainting (Dobermans, Boxers)**

- **Difficulty breathing**
- **Coughing**
- **Distended abdomen (Ascites)**
- **Weight loss**
- **Cold extremities and ears**
- **Weakness and exercise intolerance**
- **Sudden, unexpected death**

Treatment

Treatment of dilated cardiomyopathy is quite variable and depends upon the severity of the problem as well as the predominating symptom (e.g. fainting vs. coughing). The treatment may include things such as a moderately sodium restricted diet, diuretics (such as Lasix®), digoxin, nitroglycerin, ACE inhibitors (such as Enacard®, Vasotec®, Lotensin®) and various antiarrhythmic agents (such as lidocaine, procainamide, sotalol, quinidine). Taurine and carnitine may also be added.

HEART CERTIFICATION by the Orthopedic Foundation for Animals:

Heart certification by OFA is aimed at gathering data regarding congenital heart disease. They state on their website:

> At this time the inherited, developmental cardiac diseases like subaortic stenosis and **cardiomyopathies** (emphasis added) are difficult to monitor since there apparently is no clear cut distinction between normal and abnormal. The OFA will modify the congenital cardiac database when a proven (echocardiography/Holter) diagnostic modality and normal parameters by breed are established.

Overview of Canine Dental Health and Disease

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Why is veterinary dentistry important?

- every pet has a moth
- 85 percent of pets over two years has some evidence of periodontal disease
- dental health contributes to overall health
- goal should be prevention rather than treatment

Dental Disciplines

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

Anatomy

Head

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

Dentition

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

Tooth Structure

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

Periodontium

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

Tooth Anatomy

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

Dental Formula

Deciduous

$$2 \{ \underline{3}\ 1\ 3\ C\ 1\ P\ \underline{3}\ 3 \}$$

Permanent

$$2 \{ \underline{3}\ 1\ 3\ C\ \underline{1}\ P\ \underline{4}\ 4\ M\ \underline{2}\ 3 \}$$

Eruption Times

Deciduous teeth

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

Permanent teeth

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

Normal Occlusion

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

Malocclusion

Class 0 occlusion: normal or normal for breed

Class 1 occlusion: jaw relationship normal but one or more teeth out of position

- Base narrow canines

- Anterior crossbite
- Lance canine (spear, tusk)
- Posterior crossbite

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

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

Pedodontics

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

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

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

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

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

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

Periodontal Disease
- Inflammation of the structures supporting the teeth
- Normal gingival: smooth, coral pink, well attached, sulcus 1-3 mm
- Plaque: accumulation of bacteria, glycoproteins, polysaccharides that adhere to tooth

- Tartar (calculus): plaque mineralizes within 48 hours, forms more readily in alkaline saliva

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

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

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

States of Periodontal Disease

Stage 1: gingivitis
- Only reversible state
- Gingival becomes swollen, rolled and red

Stage 2: early periodontitis
- Increased gingival pocket depth
- Up to 25 percent attachment loss
- Bacteria begins to change from aerobic to anaerobic
- Teeth stable

Stage 3: moderate periodontitis
- Deeper pockets
- More cirulent anaerobic bacteria colonizes

- Up to 50 percent attachment loss (bone and gingival)
- Slight mobility of teeth

Stage 4: severe periodontitis

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

Treatment of Periodontal Disease

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

Indications for Antibiotic Use

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

Antibiotics

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

Other Dental Abnormalities

Caries (cavities)

- Bacterial degradation of enamel, not common in dogs
- Maxillary 1st molar most common

Abrasions (external source)

- Excessive grooming
- Toys especially tennis balls
- Rocks, fences, cages

Attrition (wear from other teeth)

- Malocclusion especially level bite
- Increased risk of fracture if mid tooth (canine)

Enamel hypoplasia (reduced formation of enamel, enamel dysplasia)

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

Discolored teeth

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

Inapparent oral nasal fistula

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

Foreign bodies

- Wedged between upper 4th premolars
- Sticks, bones

Home Care

Brushing

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

Toothpastes

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

Rinses

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

Chew Toys

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

Diets and Treats

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

Nutrition for Working Dogs

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What factors are important?

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

What is unique about the exercising dog?

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

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

Dietary Protein (Percent Energy)	Dietary Fat (Percent Energy)	Time (Minutes) to Exhaustion	Distance (Miles)
20%	30%	100	15
20-40%	50-70%	140	20

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

How much protein?

7) **Dogs require at least 30 percent energy as protein** for endurance exercise to prevent anemia
8) Dogs do not require more than 24 percent energy as protein for sprint exercise

Types of pet foods:

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

food when they are working. Foods designed for growing dogs mostly contain increased protein and fat.

11) **Soft-moist** and **soft-dry** (e.g. Kibbles and Bits) are intermediate but mostly low fat and are not suitable for working dogs

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

Differences between pet foods

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

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

Supplements and treats

15) Unbalance balanced diets. Do not feed supplements. Treats should comprise < 10 percent of the diet
    a. Too much meat can result in thin bones and fractures
    b. Too much liver can cause stiff neck and joints
    c. Too much calcium can cause joint problems
16) Some treats and supplements may be beneficial
    a. Chews: may be beneficial for dental hygiene
    b. Antioxidants may be beneficial especially in dogs which are not properly trained or are eating a lot of extra fat. Some manufacturers are including increased antioxidants. If feeding a diet without increased antioxidants, 10-15 IU/kg vitamin E (as alphatocopheryl acetate) may be given daily of 100 IU/kg every week by mouth. Vitamin C may also be beneficial but the dose is less clear. It is probably best to give vitamin C immediately before exercise as it does not last long in the blood and 100-200 mg may be sufficient for the average medium to large sized dog.
    c. Glucosamine or green lipped mussel powder may help dogs with arthritis but should only be used in consultation with your veterinarian
    d. Fish oil may reduce inflammation of the feet in dogs working in snow. Some diets already contain fish oil and should not be supplemented.

Human food

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

Neutraceuticals and herbs

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

How much to feed?

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

Weigh your dog every two weeks and keep a record. Always use the same scale and do it before a meal but after urinating and defecating at the same time each day and not after exercise. Sudden changes in body weight are an indication of dehydration. Slow changes will give some indication of whether you are feeding too little or too much. Also keep a record of body condition score. Take a photo for comparison so you can see how things change over time. Your dog should have a body condition score of 5 on the 9 point Purina scale.

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

When and how often to feed?

There is little data on this subject. The custom of racing sled dogs is probably the best one to follow: dogs race for 4-6 hours then take a rest for 2-4 hours during which a high fat and protein snack is fed. Dogs then race for an additional 4-6 hours before eating a full meal. Exercise markedly affects stomach and intestinal function. It is wise, therefore, not to exercise until 2-4 hours after a large meal.

Water

Dehydration must be avoided. Offer water continuously during exercise. Pet dogs on average need 50 ml/kg per day (2-5 pints per day for a 45-100 lb working dog). Working dogs may need 4 or 5 times that amount (1-3 gallons/day). Adding three level tablespoons of table sugar to a liter of water may help water absorption and will increase the recuperation of dogs at rest stops.

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

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

Gastric Dilatation Volvulus: "Bloat"

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

Recommendation:

23) Feed a national brand pet food that says on the label that it has been tested using AAFCO approved feeding and is complete and balanced.
24) Feed a high fat dry food for maintenance. Add a canned food during periods of work.
25) Feed enough to keep dog lean.
26) Do not feed supplements such as meat, bones, calcium or vitamins
27) Give 100 IU/kg vitamin E once a week by mouth. Give 100 mg vitamin C one hour before exercise and repeat every 6-8 hours during exercise.
28) Keep treats to a minimum (<10 percent of the diet). Most of the diet should be pet food.
29) Give a dental chew once daily.
30) Make sure access to water at least every half hour during exercise.

31) Feed at least four hours before exercise and after exercise.
32) Rest dogs after 4-6 hours work: give sugar water and a high protein high fat snack.

Intervertebral Disc Disease

Cheryl L. Chrisman, DVM, MS, EdS

The **spinal column** begins at the base of the skull and ends at the tip of the tail creating the neck and backbone of the4 dog. The spinal column is formed by seven cervical, thirteen thoracic, seven lumbar, three sacral and usually five or more caudal (tail) vertebrae that are held together by ligaments and muscles. The vertebrae are each numbered for reference like C1-7, T1-13, L1-7, S1-3 and Cd1-5. This way problems can be localized along the spinal column as occurring at C5, T12, L7, etc. Each vertebra has a hole in the middle so as the vertebrae align with each other a channel call the **spinal canal** is formed and runs the entire length of the spinal column.

The **spinal cord** is a thick cable of nerves that begins at the base of the brain inside the skull and extends through the spinal canal. The relationship of the spinal cord and vertebrae are much like a string running through the beads of a necklace where the string is the spinal cord and the beads are the vertebrae. The spinal cord carries all the nerves going to and from the brain that make the legs and other structures function properly.

The **intervertebral (IV) disc** is a spongy cushion that is located between the vertebrae just below the spinal canal and spinal cord. The IV disc acts like a shock absorber and adds flexibility during movement of the spinal column. There are approximately 34 IV discs throughout the entire neck and back. Each IV disc consists of an outer tough ring that has a soft gelatinous center much like a day old jelly filled donut.

IV disc disease is a degeneration of the disc that can occur due to premature or normal aging specific to certain breeds of dogs. A degenerating IV disc can rupture and spill its contents into the spinal canal (Type I disc herniation). This is often referred to as a slipped disc and can irritate, bruise or compress the spinal cord causing sudden pain or paralysis. A degenerating IV disc can also enlarge and slowly push up into the spinal canal (Type II disc disease) and put pressure on the spinal cord or nerve roots which enter and exit the spinal cord causing a slow progressive staggering gait, weakness in the legs or complete limb paralysis. Type I intervertebral disc herniation will be discussed here.

Type I intervertebral disc degeneration is common in many breeds of dogs at any age such as Dachshunds, Pekingese, Poodles, Maltese, Cocker Spaniels, Shi Tzus, Lhasa Apsos, Pugs and Beagles. The diagnosis is suspected based on the breed, history and clinical signs but tests such as spinal radiographs, cerebrospinal fluid analysis, myelogram and computerized axial Tomography or magnetic resonance imaging scans may be necessary to confirm the diagnosis.

If pain or mild limb weakness is the only sign then muscle relaxants, steroids and rest are prescribed for 2-4 weeks. **The most difficult yet most important part of the treatment for mild IV disc disease is absolute rest.** The medication may make them feel great and it is easy for dog owners to think their problems are over and this can lead to sudden paralysis. Rest often requires a major change in the family's routine, as dogs must be confined to a crate or small room and not be able to play with their toys or other dogs. Further they must be taken briefly outside on a leash for urinations and defecations with no other exercise. Small dogs can be held and cuddled and carried, but no exercise.

Going up and down stairs, jumping on and off the bed or other furniture and sitting up to beg can cause the degenerating discs to rupture and paralysis can occur. Weight reduction may be necessary in obese dogs to reduce stress on the spinal column. With rest it is hoped that the degenerating IV disc will heal somewhat and the part that is extruded into the spinal canal will spread out and move away from the spinal cord relieving the pain or weakness without surgery. Many dogs with back pain will completely recover. They may have a recurrence of problems from the same or some other IV disc later in life. Dogs with neck pain usually have to have surgery as signs persist after rest and medications.

It is recommended that dogs that are at high risk for recurrence of IV disc disease be put on antioxidant drugs such as: Vitamin E 100-200 IU and Vitamin C 100-200 mg for a 10-30 lb dog. There has been some research done to show that antioxidants will protect the spinal cord to some degree in cases of injury. These vitamins can be found in any drug or health food store and a special dog formulation is not required. Going up and down stairs, jumping on and off the bed or other furniture, sitting up to beg, playing fetch or Frisbee and other games require sudden turns to the spinal cord or agility work should be avoided in the future to prevent recurrences. Sometimes no matter how well the weight and life-style are controlled other IV discs can cause problems.

Acute explosion of a degenerative IV disc can cause sudden paralysis of the legs, which is a surgical emergency. A rapid acting steroid such as methylprednisolone sodium succinate or prednisolone sodium succinate should be administered intravenously as soon as possible after the onset of paralysis and then immediate referral to a specialist who performs decompressive surgery is essential. Treatment within the first 25 hours is the most crucial and after that time all treatments will be less effective. Approximately 80 percent of all dogs with acute paralysis given immediate rapid acting steroids and surgery within 24 hours are eventually able to walk again. Preventative surgery may be done to reduce the incidence of future problems at the same time.

After surgery most dogs need a rehabilitation time of 1-6 months but will continue to get stronger for 12 months. Most dogs cannot walk immediately even after surgery because the spinal cord has been bruised from the exploding disc. Most paraplegic dogs canoe urinate for several days. Usually they have to stay in the hospital to have their bladders manually emptied otherwise the bladder could be permanently damaged. Once they can urinate they are returned home as most dogs heal faster at home. At home simple physical therapy such as bending and moving the limbs through their normal range of movement and gentle massage will help keep limbs supple until the nerve function returns. Movement and massage in shallow warm water in the bathtub or sink can be great physical therapy, if the dog owner has the time. Fifteen minutes twice daily can be helpful. By using a sling made from a wide belt or towel they can be exercised with support. Some movement begins to return to the limbs within four weeks after surgery.

In cases of severe spinal cord injury, paralysis can be permanent. Some owners may elect to try a cart with wheels that supports the hind legs for exercise purposes. I suggest they try the cart for a month and see how they think their dog's quality of life and how the household adjusts to a

paraplegic dog. Many dogs and families adjust fine. However dogs cannot live in the cart as they cannot lie down. The bladder must be emptied completely and urine samples monitored by a veterinarian for infections. Bladder infections, which then damage the kidneys is the most life threatening problem in paraplegic dogs. Most dogs with IV disc disease recover and with a few lifestyle changes lead happy lives.

Gastric Dilatation Volvulus – An Update

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Gastric dilatation Volvulus complex also know as bloat is a medical and surgical emergency which is know to primarily affect large and giant breeds of dogs. The disease has also been reported in smaller breeds such as the Pekingese and Dachshund. Mortality has been estimated as high as 30 percent. There are no reliable estimates of how many dogs develop bloat in the United States each year, but in certain breeds such as Irish Setters and Great Danes owners reported an incidence of seven and ten percent respectively. It does appear that purebred dogs are more likely to develop bloat than are mixed breed dogs.

Results of Retrospective Studies

Incidence Several recent reviews by Dr. Larry Glickman at Purdue University utilizing information from the veterinary medical database (VMDB) have discovered some interesting findings:

1) Amongst veterinary institutions the frequ3ency of bloat amongst all dogs ranged from 2.9-6.8 per 1000 dogs
2) Approximately 29 percent of the dogs with gastric dilatation and 33 percent of those with dilatation and volvulus died.
3) Aging of the dog increased risk. Dogs greater than seven years of age are more than twice as likely to have bloat as dogs 2-4 years of age.
4) Purebreds were three times as likely to have bloat as mixed breed dogs.
5) Males are twice as likely to bloat as females yet spaying or neutering has no effect on the risk of bloat.

Studies of Risk Factors

Several risk factors have been identified which may contribute to the establishment of bloat in purebred dogs.

Breed Bloat has been long reported to be more common in large and giant breeds of dogs yet until recently the prevalence of bloat was not compared to the dog population at large. When this data was analyzed statistically, it was found that the Great Dane, St. Bernard, Weimaraner, Irish Setter and Gordon Setter were breeds at greatest risk. An accompanying chart outlines the remainder of the breeds (Table 1).

Chest Conformation Although it is established that large and giant breeds are the breeds at greatest risk it has been shown there are profound differences in the risk of bloat within certain breeds. This possibility seems related to conformation of the naimal's chest. For instance, breeds such as Irish Setters which are at high risk may weigh approximately the same as some of the Retriever breeds yet the Retrievers are at much lower risk than Irish Setters for developing bloat. When looking at this more scientifically it was found that the depth and width of the chest may be the key in predicting which animals within a certain breed may develop bloat. It appears that the chest depth/width ration is highly correlated with risk of bloat, ie. Those animals with

deep, narrow chests within a certain breed are much more likely to develop bloat than those dogs with deep wide chests. In using external measurements of chest conformation it was found that within the Great Dane breed the depth/width ration may indeed be useful in identifying animals prone to bloat. Also, Great Danes with moderate and high abdominal height to width rations were approximately 5 ½ to 8 times as likely to develop bloat as those with low abdominal height to width rations. In Irish Setters the chest height to width ration also correlated with those dogs having a higher depth to width ration being much more likely to develop bloat than those animals with a lower depth to width ration. This information is obviously very significant in terms of selective breeding for the reduction of bloat in these breeds.

Diet Exact determinations of types of diet on risk for developing bloat still cannot be made. Although cereal-based diets have incriminated, it is difficult to compare groups since almost all large and giant breeds are fed cereal-based diets. Therefore, further controlled studies will be necessary to determine if cereal-based diets are in fact a risk factor. However, several interesting findings have come to surface with regard to the diet and nutritional management of breeds predisposed to bloat. For instance, it has been shown that dogs who eat one meal a day are almost twice as likely to develop bloat as those fed twice a day. The rate of eating is also very important. Those dogs characterized as slow eaters have the lowest incidence of bloat whereas those dogs characterized as moderately fast eaters have about 2 ½ times the chance of developing bloat and those characterized as fast eaters have almost five times the chance of developing bloat as those being characterized as slow eaters.

Body weight may also be of some significance. Being overweight actually reduced the incidence of bloat compared to dogs that were optimum weight. However, those animals characterized as significantly underweight were about three times as likely to develop bloat as those animals characterized as optimum weight.

Gender It has been shown that males are approximately twice as likely to develop bloat as females. Neutering does not seem to have an effect on the incidence however.

Personality and Environment There does seem to be a direct correlation of the animal's temperament relating to its tendency to develop bloat. Those animals being characterized as unhappy or fearful were about 2 ½ times as likely to develop bloat as those animals characterized as happy. In addition, the environment may play a role. Stress appears to also significantly increase the chance of the animal developing bloat. Therefore, animals who may undergo significant stress traveling to show, etc. are tow to three times as likely to bloat than those animals who are not significantly affected by the transport. Also activity level may be important with those animals characterized as hyperactive and those animals being categorized as less active were twice as likely to develop bloat than those animals characterized as having a normal activity level.

Summary and Conclusions

With regard to known epidemiologic factors affecting bloat some of the following recommendations can be made. It appears there is a correlation with chest and abdominal height/width ration with those animals having tall thin chests and abdomens more likely to

develop bloat than those with lower, wider chests and abdomens, there selective breeding may possible be recommended to diminish the incidence due to conformation. With regard to diet and nutrition changes in feeding relating to twice a day feeding versus once a day feeding may be recommended. Also, changing the time of the meal is significant with those animals having constant changes in meal time being approximately 2 ½ times as likely to develop bloat as those being fed at regular intervals. In addition, those animals undergoing a sudden increase in food intake are almost three times as likely to develop bloat as those animals kept on a regular food intake. Therefore, recommendations may be made to keep consistency and times of feeding regulated and to feed the moderate amount of food without sudden increases in the amount fed. It also appears that reducing the amount of stress on the animal will decrease the chances of bloating. Although no specific recommendations can be made about tranquilization knowing the temperament of your dog may help you in minimizing the amount of stress encountered during travel to and from shows. It also appears that keeping physical activity to a moderate amount that is what the animal is used to, will be more helpful in reducing the chances of bloat than allowing extra activity than normally expected.

Etiology

The exact etiology of GDV is unknown, but it is most likely a multifactorial disease. Ingestion of cereal based diets and water followed by exercise is reported in some but not the majority of cases. Stretching of the hepatogastric or hepatoduodenal ligaments from chronic overeating may allow transposition of the stomach. Gastric outlet obstruction by foreign bodies has been observed in some cases but delayed gastric emptying caused by pyloric hypertrophy is not conclusively proven as a cause of GDV. Splenic torsion or displacement occurs secondarily to DGV rather than initiating it, as was once believed. Gas production secondary to bacterial fermentation by clostridial organisms is a postmortem finding and is not a source of gas in live animals. Aerophagia is a likely cause since gas composition of the gastric lumen resembles atmospheric air and the onset of GDV often follows vigorous exercise, excitement and barking. Recently there is evidence that gastric motility disorders may induce or follow GDV.

Clinical Signs

Dogs usually demonstrate hypersalivation, retching or unproductive vomiting on presentation. Cranial abdominal distention is apparent and gastric tympany is usually present on blunt percussion of the right anterior quadrant. Hyperpnea or dyspnea accompanied by open mouth breathing indicates hypoxia due to reduced diaphragmatic excursions. Shock is evidenced by pale or injected mucous membranes, prolonged capillary perfusion, tachycardia and weak rapid femoral pulse.

Mechanisms of Rotation

A lack of coordinated gastric contractions due to gastric myoelectric dysrhythmias may slow gastric emptying and contribute to the development of gastric dilatation volvulus (GDV). Food and fluid distension from overeating or gaseous distension from aerophagia causes intra-abdominal angulation of the gastroesophageal junction that prevents belching or vomiting. Gastric dilatation results. Volvulus occurs when the dilated gastric fundus becomes displaced

from a left dorsal to a right ventral position. The pylorus concurrently shifts from its right ventral position to a left, caudal and dorsal position. When viewed from the rear a clockwise rotation occurs in the majority of the animals. The spleen follows the greater curvature to the right. The gastrosplenic ligament and short gastric arteries are often torn during the volvulus.

Initial Management of GDV

Initial patient management involves shock therapy, and gastric decompression followed by management of cardiac arrhythmias. Shock therapy involves fluid loading with 90 ml/kg of lactated Ringers solution of the first hour. The use of hypertonic saline may also be beneficial, as it has been shown to be beneficial in increasing gastric arterial perfusion. Treatment for acid-base status is controversial with one study indicating normal pH and another indicating the presence of metabolic acidosis in cases of GDV. However, with mild metabolic acidosis Na bicarbonate infusion is not necessary as long as adequate volume replacement with lactated Ringers solution is achieved. Hypokalemia is a common finding associated with GDV and potassium replacement is sometimes warranted. Corticosteroids are administered after initial treatment with intravenous fluids. They cause vasodilation and improved tissue perfusion if fluid volume is adequate. Cardiac dysrhythmias are commonly seen and require careful pre- and postoperative management. Paroxysmal vent4ricular tachycardia, and premature ventricular contractions are most commonly seen.

Gastric decompression is accomplished using a pre-measured, well lubricated PVC plastic foal nasogastric tube. Ability to pass the tube into the stomach does not mean that gastric volvulus is not present. If intubation is not possible in the prone position it is attempted in a sitting upright position. Sometimes trocharization is necessary to reduce distension and facilitate tube passage. The character of the fluid is sometimes important in predicting the status of the gastric lining. Black fetid smelling fluid with flecks of devitalized mucosa indicates that mucosal ischemia is present and often predicts the presence of gastric wall necrosis. After decompression, the stomach is lavaged with 4-5 liters of water using gravity flow, dose syringe or stomach pump.

Radiography

Radiography is always postponed until after patient stabilization. With gastric dilatation the stomach appears as a grossly distended gas and fluid filled structure that occupies the cranial abdomen displacing all remaining viscera posteriorly. The spleen is usually not visible in its normal left ventral location and is often located in a right dorsal position. Gastric volvulus is suspected when the pylorus is located dorsal, cranial and to the left of the midline. After decompression it may take a classic “upside down” appearance. Left and right lateral views are recommended. On the right lateral view gas can be seen in the pylorus whereas on the left lateral view gas may be seen in the fundus. If stomach position is questionable barium sulfate is administered to identify the pylorus.

Surgical Management

Definitive management of GDV involves 1) repositioning of the stomach with resection of any devitalized gastric wall and 2) a prophylactic gastropexy technique to prevent recurrence. UP to

80 percent recurrence of GDV is reported with gastric decompression or repositioning alone. We now advocate laparotomy as soon as the patient is a reasonable anesthetic risk. This allows early derotation that increases circulation and allows assessment of gastric wall viability. Areas of necrosis are detected early and resected if possible. With 270º to360º clockwise gastric volvulus the dilated stomach is covered on its ventral aspect by omentum. Reduction is accompanied by passing the had down the left abdominal wall, grasping the pylorus in its left dorsal position and rotating it in a caudal and counter-clockwise manner to its normal right sided location.

Gastrectomy Techniques

Standard methods for gastrectomy involve ligation of branches of the left gastroepileploic arteries and veins allowing areas along the greater curvature of the stomach to be resected. The stomach is resected back to areas of healthy bleeding. Spillage is likely and prevented through the use of Babcock forceps or stay sutures. After resection is complete the stomach is closed in two layers. The mucosa and submucosa are closed with a continuous inverted Cushing pattern of 2-0 or 3-0 PDS or Maxon. The serosa and muscularis are then closed with a similar pattern. Recently we have relied heavily on the autostapling equipment for rapid gastrectomy procedures with minimum risk of spillage. The TA90 autostapler is used with the green (4.8 mm) or blue (3.5mm) cartridge. Often several end-to-end staple lines have to be placed since each staple line is only 9 cm in length. The surgeon needs to overlap the staple lines by a few mm to prevent leakage between the staples.

Rationale for Gastropexy

By definition gastropexy describes the fixation of the stomach to nearby structures or body wall as a means of preventing recurrence of GDV. Although gastropexy procedures reportedly diminish the recurrency rate of DGV, their reliability in producing permanent adhesions between the stomach and abdominal wall is not well documented.

Most North American surgeons use an antral gastropexy procedure to fix the gastric antrum to the right abdominal wall. The three major categories of "permanent" antral gastropexies used in North America are the tube gastrostomy described by Parks (1976); the incisional gastropexy described by MacCoy (1982); and the circumcostal gastropexy described by Fallah (1982). In addition, two modifications of muscle flap techniques, one using a "muscular flap" from the abdominal wall (Shulman, 1986) and another using a "belt-loop" from the gastric muscularis (Whitney, 1989), have recently been described.

Clinical Results

Potential advantages of the tube gastropexy are that 1) the surgery is rapid and easy, 2) the tube not only creates a permanent adhesion of the gastric antrum to the abdominal wall preventing recurrence of volvulus but also 3) allows for continued gastric decompression in the early postoperative period and 4) slurried food or medications can be offered through the tube. The main disadvantages of the technique are 1) the nursing care and long hospital period required for

tube management and 2) t6he potential for fatal peritonitis secondary to leakage around the tube or early removal by the dog.

Clinical studies of the tube gastrostomy have yielded encouraging results. Flanders (1984) reported recurrence of volvulus in only one of 29 dogs treated with tube gastrostomy for a follow-up time ranging from 14 to 40 months. However there was a mortality rate of 31 percent during the first week after surgery. Johnson (1984) reported on 76 cases where this technique was used with only a five percent recurrence rate. Older studies describe a recurrence rate as high as 29 percent (Walshaw, 2976) as well as a 17 percent complication rate (Fox 1985) including premature dislodging of a tube, peritonitis, subcutaneous cellulites and persistent stoma drainage.

Advantages of the incisional gastropexy are that 1) the procedure is rapidly done, 2) the stomach lumen is not entered and 3) fibrous connective tissue enters the abdominal rectus muscle and stomach wall to form a strong mature adhesion. The potential disadvantage is that the gaseous decompression is not provided in the postoperative period. The incisional gastropexy is popular among many North American surgeons but unfortunately no good retrospective studies are available to determine its clinical efficacy.

The circumcostal technique has become popular for use in academic medicine because it probably forms a stronger adhesion. It is reported to be more difficult to perform than the other techniques but the author disagrees with this statement. Potential advantages include a 1) viable muscle flap adhesion as well as 2) a more proper anatomic placement of the stomach. Potential disadvantages include a prolonged surgical time, potential for rib fracture and potential for pneumothorax because of the close proximity to the diaphragm. Lieb (1984) reported on 39 dogs with circumcostal gastropexies to have a slightly lower recurrent rate (2.6 percent at 13.7 months) than dogs with tube gastrostomy.

Belt loop gastropexy offers similar advantages to the circumcostal and incisional gastropexies in that the gastric lumen is not entered and the risk of peritonitis is minimal. The technique is easily performed by an unassisted surgeon. Although the belt loop gastropexy has not been evaluated biomechanically one would suspect that breaking strengths would be superior to incisional or tube gastrostomy techniques but not quite as secure as circumcostal techniques since the base of the flap is narrower than the latter technique.

Postoperative Management

Diligent postoperative care is mandatory for successful outcome of the gastric dilatation volvulus patient. Most dogs that die in the postoperative period will do so within the first 3-4 days after surgery. After major gastric resection the animal is kept NPO for a period of 24-48 hours. Maintenance fluid, electrolyte and acid base status is critical during this period. Maintenance fluid should be given at a rate of 40-60 ml/kg per day. Although many dogs maintain normal serum potassium levels following gastric dilatation volvulus a total body potassium deficit may exist because of the NPO status, vomiting, oral gastric innervation and removal of gastric secretions. Therefore, supplementation of 20 mEq of potassium chloride is usually added to each

liter of fluids to help maintain a total body potassium. Hypokalemia can also contribute to the development of cardiac arrhythmias, and gastrointestinal ilius.

The Role of Reperfusion in GDV

Mortality associated with gastric dilatation volvulus is most often due to gastrointestinal ischemia secondary to the large twisted stomach. It has been estimated that mortality can increase to 60 percent in the presence of gastric necrosis. Tissue ischemia to the gastric wall occurs due to reductions in arterial perfusion and venous stasis within the stomach wall. When the stomach is decompressed via the stomach tube and derotated via surgery there is rapid reperfusion of this ischemic tissue. Paradoxically, tissue ischemia followed by reperfusion with oxygenated blood may further increase tissue damage due to a phenomenon known as reperfusion injury.

Reperfusion injury is thought to be mediated through the activity of oxygen-derived free radicals that is based on an iron dependent mechanism. These free radicals result in cellular lipid peroxidation and cell death. Since GDV is associated with high mortality and since most deaths occur within 96 hours of surgical intervention it is plausible that treatment directed toward preventing or moderating reperfusion injury may improve survival following the correction of GDV. Studies in experimental dogs with GDV have shown that xanthene oxidase inhibitors such as allopurinol and iron chelators such as deferoxamine have been helpful in reducing the amount of free radical production consequently minimizing cellular damage due to reperfusion injury and potentially decreasing mortality associated with GDV. Although experimental results are preliminary, it is likely that within five years some of these now experimental drugs will be utilized in emergency centers for the clinical management of GDV.

Bloat Risk Ranking for 24 Purebreeds, Compared with Risk for All Dogs Combined *

Breed	***Affected***	***Not Affected***	***Risk Rank***
Great Dane†	299	37	1
St. Bernard†	81	19	2
Weimaraner†	49	13	3
Irish Setter†	180	65	4
Gordon Setter†	24	10	5
Standard Poodle†	47	33	6
Basset Hound†	39	34	7
Doberman Pinscher†	139	130	8
Old English Sheepdog	27	29	9
German Shorthaired Pointer	25	28	10
Newfoundland	13	15	11
Airedale Terrier	12	15	12
German Shepherd Dog†	202	246	13
Alaskan Malamute	23	29	14
Chesapeake Bay Retriever	10	14	15
Boxer	28	39	16
Collie	39	71	17

Labrador Retriever	72	182	18
English Springer Spaniel	18	45	19
Samoyed	13	42	20
Dachshund	26	81	21
Golden Retriever‡	37	158	22
American Cocker Spaniel	14	115	23
Miniature Poodle‡	10	159	24

*Rank based on unadjusted odds ration (an estimate of relative risk) in purebreeds for which there were ≥ ten cases and ≥ eight controls. All dogs combined (pure and mixed breeds) included 1934 cases and 3868 controls.

†Risk significantly higher than for all dogs combined.

‡Risk significantly lower than for all dogs combined.

From Glickman LT[2]. Epidemiologic Studies on Bloat in Dogs. Purina Veterinary Previews, 1992; 2: 10-15.

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Canine Geriatric Medicine

Michael Schaer, DVM

Geriatrics – The branch of medicine dealing with the problems of ageing and diseases of the older animal.

How old is old?

Giant:	> 7 years
Medium:	> 9-10 years
Small:	> 10 years

The Big Baddies
- Cancer
- Renal failure
- Heart failure
- Chronic progressive lung disease
- End stage liver
- Necrotizing pancreatitis
- Intestinal volvulus

Signs of Chronic Illness
- Weight loss
- Increased thirst/urination
- Cough
- Weakness
- Persistent vomiting/diarrhea
- Altered mental state

Signs of Acute Illness
- Sudden vomiting/diarrhea
- Mental depression
- Anorexia
- Difficulty breathing
- Pain
- Weakness
- Seizures/fainting
- Fever

Vomiting – DDX
- Gastric irritation
- Pancreatitis
- Major organ dysfunction
- Intoxication
- GI obstruction: foreign body/tumor

- Metabolic disease: DKA, hypercalcemia

Vomiting – Data Base
- CBC
- Serum chemistries
- Radiographs: chest/abdomen
- Ultrasound: abdomen

Fever – DDX
- Infection
- Immune
- Neoplasia
- Metabolic
- Excitement/environment
- Necrosis
- Drug reaction

Fever – Data Base
- Thorough history
- CBC
- Serum chemistry
- Urinalysis and/or culture
- Radiographs: chest/abdomen
- Serology – where indicated
- Cytology and culture – where indicated

Weakness – DDX
- Neurological
- Muscular
- Orthopedic
- Metabolic
- Cardiovascular
- Anemia
- Any chronic debilitating illness

Weakness – Data Base
- Thorough Hx
- Meticulous PE
- CBC
- Serum chemistries
- ECG
- Others as needed

Increased Thirst/Urination – DDX

- Diabetes mellitus
- Diabetes insipidus
- Chronic renal disease
- Pyometra
- Cushings
- Chronic liver disease
- Psychogenic PD
- Hyperthyroid
- Diuretic drugs/others
- Hypercalcemia

Data Base for PD/PU

- CBC
- Serum chemistry profile
- Urinalysis and/or culture
    - And/or radiographs
    - And/or abdominal ultrasound

Labored Breathing – DDX

- Pleural space – fluid, masses
- Heart – congestive failure, dysrhythmias
- Lung – edema, inflammation, infiltrate

Minimal Data Base for Dyspnea

- Chest radiographs
- CBC
- Serum chemistries
- Urinalysis
- Heartworm test; other serology
- ECG and/or echocardiogram
- Cytology or biopsy

Adverse Drug Reactions

- More common than most think
- Difficult to predict
- Individual variation
- Can occur with any single drug
- Drug interactions also common

Phenothiazine tranquilizers:	Severe hypotension, prolonged sedation
Ace inhibitors:	Renal failure
Metronidazole:	Neurotoxicity
Cimetidine:	P450 inhibitor
Theophylline:	Restlessness, GI, neuro

Common Sense Rules
- Keep blind pets away from swimming pools
- Keep deaf pets away from traffic
- Provide roughened floor surface for weak animals
- Second guessing can jeopardize your pet
- Don't mess with Mother Nature
- Avoid excesses

The Do's
- Do tend to their needs
- Do feed appropriately
- Water free choice
- Extra opportunities for nature's ways
- Yearly veterinary visits

The Don't's
- Don't restrict water
- Don't ignore acute signs of illness
- Don't force exercise in hot weather
- Don't be your own doctor

Things to Avoid with the Geriatric Pet
- Forced exercise in the unconditioned pet
- Obesity
- Water deprivation
- Environmental temperature extremes
- Unhealthy teeth
- Ignoring abnormal signs or behavior
- Mental stress

Old is NOT Dead
- Animals die from disease or organ failure
- They rarely die from "old age"
- Age brings out the best in all of us – right?

Research Priorities Identified by AKC Parent Clubs*

C. Richard Dorn, DVM, MPH** and Erika Werne, MIM***

The Goal of the AKC Canine Health Foundation is to effectively integrate the observations and knowledge of dog owners, breeders, veterinarians and other scientists in advancing the health of dogs. The Foundation conducts an annual Canine Health Survey of the AKC Parent Breed Clubs. Each Club's health committee is asked to list the major diseases that should receive priority for research support. The most frequently identified research priority diseases in 2000 were: hip dysplasia, epilepsy, cancer, allergies, hypothyroidism, bloat, progressive retinal atrophy, autoimmune disease, heart disease and cataracts.

From the year 2000 Parent Club Health Survey and other communications from the Breed Clubs, a listing of specific breed interests was compiled and distributed with the Foundation's Request for Research Proposals to researchers. This listing, containing 85 diseases with corresponding breeds, is included with this abstract as an attachment. The number of breeds associated with a specific disease varied widely. For example, Basenji was the only breed listed with Fanconi Syndrome (kidney defect), but there were 25 breeds listed with hip dysplasia.

Even though a specif breed interest list has been prepared, it may not include a disease or diseases affecting your dogs. Also, a research proposal directed at a disease affecting your dogs may not be submitted. What can an individual owner or breeder do to attract research to their breed? The following are factors to consider in building the case for attracting researchers to health problems in your dogs:

1. Basis of diagnosis of disease(s) in my dogs
    a) Physical examination
    b) Clinical procedures
    c) Laboratory tests
    d) Disorders versus definitive diagnoses
    e) Manifestational versus causal diagnoses
2. Kennel versus breed problem
    a) Continuous new cases in kennel/breed:
    b) Continuous new cases in other breeds?
    c) Frequency versus relative risk (odds ration) of developing the disease
    d) Cause(s) known
    e) Causal factors present
    f) Who determines whether it is a kennel or breed problem
3. Breed club surveys
4. Matching funds with AKC Canine Health Foundation
5. Long term versus short term support
6. Feedback from Foundation and researchers

The selection of researchers to receive grants from the Foundation is based on the outcome of a hierarchical review process. The researchers' responses to the Request for Pre-proposals are reviewed by the Foundation's Grants Committee members who examine them for scientific promise and likelihood of controlling or preventing major disease problems. The principal

investigators of the successful pre-proposals are then contacted and invited to submit a Full Application using a standard format. The submitted Full Applications are then distributed to three scientific peers for review. The peer-reviews are then examined by the Foundation Board of Directors and the final selection for grant fund distribution is made.

Six annual grant cycles have been completed. Three hundred fifty-six pre-proposals (six cycles only) have been received. Two hundred fifteen Full Applications (six cycles) were received and peer-reviewed. At present, there are 69 active grants and 15 pending grants. Thirty-one research projects have been completed.

In addition to research, the Foundation provides financial support for education programs such as this Dog Owners and Breeders Symposium. Other Foundation supported education program topics have been: canine genetics, cancer research, immunological diseases and reproductive health. The website www.akcchf.org is available for persons wishing more information about the Foundation.

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3. Padgett GA: Control of Canine Genetic Diseases. 1998 Howell Book House, NY.
4. Wilkie PJ: Future Dog: Breeding for Genetic Soundness. 1999 Minnesota Agricultural Experiment Station, St. Paul, MN.
5. Ackerman L: Guide to Health Problems in Purebred Dogs. 1999 American Animal Hospital Association Press, Lakewood, CO.
6. Casey D: Primer on Molecular Genetics. Web site www.bis.med.jhmi.edu/Dan/DOE

*For presentation at the Fourth Annual Dog Owners and Breeders Symposium, University of Florida, Gainesville, Florida on July 9, 2000

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Current Concepts in Veterinary Ophthalmology

Dennis E. Brooks, DVM, PhD
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What do dogs and cats see?

1. Small monocular visuals fields of peripheral vision in dogs and cats (~80°) as compared to the horse (146°).

2. Large **frontal binocular** visual field of 85° in dogs and cats

3. Dogs have cones that are receptive at 429 and 555 nm and are dichromats. All evidence suggests that the cat is dichromat with vision similar to a human who is red-green color blind. Dogs and cats appear to respond to the blue and yellow short-wave length colors the best, but appear to have trouble with green and red. Dogs and cats are also rod-dominant animals. As rods do not function in daylight these animals are dependent on their few cones for spatial and temporal visual resolution, which probably means that their blue and yellow visual world is a fuzzy blue and yellow world. What appears red to us is simply dark to the dog and cat, and a part of the green spectrum is indistinguishable from white. Colors that would appear very rich to us a more pastel-like to the cat. The cat sees a green, grassy lawn as a whitish lawn, and a green rose-bush as a whitish bush with dark flowers.

4. Most dogs are emmetropic with a tendency for mild myopia in German Shepherds (-9.86D), Rottweilers (-1.77D) and Miniature Schnauzers (-0.66D). Myopia is associated with nuclear sclerosis in older dogs. Refractive errors are rare in cats, but when they do occur, they tend to be myopic.

5. Acuity is 30 cycles per degree (cpd) for humans, 18 cpd for horses, 12 cpd for dogs and 6 cpd for cats. Acuity in dogs in 0.4 times that of people, 0.67 times that of horses, and 2 times that of cats. Acuity in cats is 0.2 times that of people, 0.33 times that of horses, and 0.5 times that of dogs. If normal human vision is 20/20, then that of the dog is 20/50 and that of the cat is 20/100.

6. Aphakic condition in dogs is hyperopic. Streak retinoscopy for air or spectacle correction is +14D in the dog and +10.5D in humans. Pseudophakic emmetropia requires +41D intraocular lens to correct the hyperopic aphakic condition in the dog.

7. The preponderance of large diameter axons and large ganglion cells indicate that the dog should have good motion detection and high temporal contrast capabilities. Acuity is less than humans and horses, but greater than the cat. Dogs have few alpha ganglion cells (GC) in the temporal quadrants of the retina. Alpha GC have large receptive fields, large, fast-conducting axons, and are associated with motion detection. The effect of the loss of such cells to the binocular nasal visual fields of the dog is not known.

8. Siamese cats have fewer ganglion cells, little stereopsis, less acuity and too much crossing over of optic nerve axons at the optic chiasm. This results in misrouting and suppression of the visual input from the nasal visual fields at the LGN. Cross-eyedness may be an adaptation to move the nasal retinas (and temporal visual fields) more anteriorly, as the more esotropis that is present the more misrouting of axons that has occurred.

9. The vertical stenopaic slit pupil of the cat allows for maximum light control and optically decreases astigmatic scatter in the horizontal meridian.

10. The lens of dogs and cats has weak accommodative ability and therefore they have limited near focus capability. The corneal curvature of both species (u.9 mm in dog, 9.1 mm in cat) is much greater than the human (7.9mm) to compensate for this.

Common Ocular Disorders of Dogs and Cats

Traumatic Proptosis

May result in enophthalmos, exophthalmos, or even complete proptosis or luxation of the eyeball. Infections, inflammation, blindness and/or strabismus may be sequelae. Brachycephalic breeds are at higher risk.

Pupils dilated – guarded to unfavorable prognosis

Treatment for traumatic proptosis:

1. Keep the eye moist. Gently remove debris with copious sterile saline flushes.
2. Under general anesthesia replace the eye, performing canthotomy if necessary
3. Temporary tarsorrhaphy
4. Maintain medical treatment with systemic antiobiotics, topical antibiotic ointments and atropine. May use systemic anti-inflammatory drugs.

Corneal Ulcers

Ulcerative keratitis is the **most common** corneal disease in our practice. A corneal ulcer is a lesion in which epithelium and a variable amount of stroma have been lost. Small acute ulcers in normal cornea should heal rapidly.

Corneal ulcers classified by depth

Superficial (simple) ulceration	→	Deep Ulceration	→	Descemetocele	→	Iris Prolapse	→ Endophthalmitis

Corneal ulcers classified by etiology. Abrasions, Foreign bodies, Entropion, Eyelash disease, Infectious, KCS

Treatment – the phases in treatment of a corneal ulcer are as follows:

- Determine etiology – remove or eliminate specific cause (e.g. KCS, correction of entropion).
- While most corneal ulcers are secondary, concurrent bacterial involvement must be treated by antimicrobial therapy.
- Broad – spectrum antibiotics are usually administered; culture and sensitivity tests can guide selection.

Prevent progression:

1. **Acetylcysteine (5 percent)** is used topically for its collagenase and protease inhibiting properties.
2. **SERUM.** Serum is thought to act when used topically or subconjunctivally because it contains an alpha-2 macroglobulin with antiocollagenase activity.
3. Topical **atropine** therapy (1 percent TID) is also instituted to relieve pain due to secondary anterior uveitis, and to decrease the formation of synechiae from the miotic pupil (as a result of uveitis).

Provide corneal support: In the treatment of deep corneal ulceration, coverage with one of the various kinds of flaps should be maintained for 10-21 days. Types of conjunctival flaps: 360˚, pedicle, and bridge.

Topical and Subconjunctival Corticosteroids are Contra-Indicated When the Cornea Retains Fluorescein Stain (i.e. Corneal Erosion or Ulcer is Present).

Treatment of Deep Stromal Corneal Ulcers, Descemetoceles, Iris Prolapses, Corneal Lacerations

1. Aggressive therapy
2. Surgical repair is needed
3. Topical and systemic antiobiotics
4. Topical atropine
5. Conjunctival flap, corneoscleral transposition, or partial – thickness corneal graft

Entropion

- Inward rolling of eyelid margin
- Entropion is most common in dogs
- Uncommon in cats (primarily in Persians)
- Medical treatment: Ocular lubricant ointments – Lacrilube, Dura Tears, or Duralube
- Surgical treatment: Temporary vs. permanent

Eyelid Neoplasms

- Sebaceous gland adenoma
    - Most common
    - Frequently found in older dogs
    - Should be removed

- Melanomas
    - Frequently darkly pigmented
    - Early surgical resection is recommended
- Squamous cell carcinoma
    - Rare in the dog
    - Most common lid tumor of cats, cows and horses
    - Early biopsy and wide surgical excision are imperative
    - Radiation therapy decreases the chance of recurrence

Keratoconjunctivitis sicca (KCS)

Keratoconjunctivitis sicca (KCS): aqueous deficiency of the precorneal tear film (PTF) causing progressive inflammatory changes of the cornea and conjunctiva.

Diagnosis
Schirmer tear test
- Normal 15-25 mm/minute
- Suspicious 8-10 mm/minute
- Low < 8 mm/minute

Incidence
Breeds: English Bulldog, West Highland White Terrier, Lhasa Apso, Pug, Cocker Spaniel, Pekingese, Yorkshire Terrier Shih Tzu, Miniature Schnauzer, Boston Terrier

Clinical Signs
Acute: Blepharospasm, conjunctivitis, mucoid discharge, corneal ulcers, dry appearance
Chronic: owners frequently complain of "chronic eye infection," copious, mucoid-mucopurulent discharge, dull cornea with neovascularization, pigmentation of cornea, improvement with any topical medication

Treatment
Medical
- Always attempt 1-2 months of medical treatment because the problem may be transient. Owner compliance may be difficult.
- Goals to remove pain and maintain vision:
    - Replace tears – Hypotears (Cooper Vision); Tears Naturale (Alcon), Lacrilube (Allergen); Duratears (Alcon); Lacriserts (Merck)
    - Stimulate production of tears with 2 percent cyclosporin (OPTIMUME); dose: 1 BID; may take 3-4 weeks before increasing tear production
    - Control bacterial flora – topical broad spectrum antibiotic (e.g. triple antibiotic ointment)
    - Control inflammation – topical corticosteriods, may combine with topical antibiotic, use only if not ulcerated.

Conjunctivitis
The eye gets red or has conjunctivitis in nearly all types of eye disease. The eye has limited ways to react to injury!

Diagnostic tests:
- STT – routine on all conjunctivitis cases
- Culture/sensitivity – fornix
- Cytology – topical anesthetic, spatula

Etiologies:
- Bacterial
- Viral
- Allergic – frequent cause

Hypertrophy and Prolapse of Nictitans Gland (Cherry Eye)

1. Primarily seen in young dogs, less than two years
2. Most common in Beagles, American Cocker Spaniels, Pekingese
3. Gland protrudes above free border of the TE, becomes inflamed and enlarged. May see epiphora, mucoid discharge and conjunctival inflammation
4. Histologically plasma cell and lymphocyte infiltration
5. Treatment
    a. Medical – physically replace gland, topical Corticosteroids
    b. Surgical – Repositioning of gland to normal location

Cataracts

Definition: Opacity of the lens or lens capsule. Lens fibers are disrupted.

Cataract Classification: to help evaluate etiology and prognosis. Classification of cataracts by degree of maturation:
- Incipient: earliest lens changes
- Immature: fundic reflex still present (usually present peripherally); vision is impaired to a variable extent
- Mature: lens is totally opaque, the fundic reflex is absent, and vision is lost
- Hypermature: wrinkling of the anterior lens capsule; this resorption may cause a lens-induced uveitis

Nuclear Sclerosis: Age related change due to compaction of the lens fibers. Not a true opacity therefore not a true cataract.

Surgical Techniques for Cataracts
- Extracapsular cataract extraction (ECCE)
- Phacofragmentation (preferred therapy at the moment)

Canine and Feline Glaucomas

Aqueous Humor Dynamics and Intraocular Pressure

By definition, glaucoma is increased IOP with associated visual deficits. In most cases in dogs and cats, glaucoma is caused by obstruction or stenosis of the aqueous humor outflow pathways.

Pathologic Effects of Glaucoma

All ocular tissues are eventually affected by the elevated IOP. The presence, individually or as a group, of a "red eye," corneal edema, mydriasis, belepharospasm, blindness, and buphthalmos can be explained by the increased IOP. If the IOP cannot be reduced, an overall increase in the size of the globe may result (buphthalmos). This change may occur more rapidly in young dogs and cats. Zonular disinsertion results in lens luxation.

Types of Glaucoma

Primary glaucoma in dogs is a breed-related, hereditary condition. Predisposition to primary open-angle glaucoma in the Persian and Siamese cat breeds has also been noted, but in the author's experience, domestic short-hairs are more often affected. In both dogs and cats, affected animals may present with only one eye involved, but the risk is very high for development of glaucoma in the other eye.

Secondary glaucoma is more commonly encountered than primary glaucoma in dogs and cats. The elevated IOP results from other disease processes within the eye. The glaucoma may be open or closed angle, and in some instances is associated with pupillary block. The condition tends to be unilateral without an inherited basis.

Clinical Signs of Acute and Chronic Glaucoma

The presentation of a patient with a painful, red eye requires that glaucoma be ruled out among the possible diagnoses of conjunctivitis, uveitis, or keratitis. The onset of clinical signs in cats is often insidious, as cats are less likely to demonstrate the acute intense corneal edema and episcleral congestion exhibited in dogs. Signs of chronic glaucoma are dramatic. The include combinations of the early signs with buphthalmos, lagophthalmos, exposure keratitis, luxated lens, corneal striae, optic nerve atrophy with cupping, and retinal atrophy.

Tonometry

IOP must be accurately measured to diagnose glaucoma. The normal canine and feline IOP is 15 to 25 mm Hg. An IOP greater than 30 mm Hg is considered pathologic and diagnostic for this condition.

Medical Therapy

Multiple drug therapy to decrease IOP by reducing production of aqueous humor and diminishing the resistance to aqueous humor outflow is the most effective approach. Carbonic-anhydrase inhibitors reduce ciliary-body production of aqueous humor independent of diuresis. Topical parasympathomimetic drugs act primarily to cause ciliary muscle contraction, increasing the outflow of aqueous humor. B-adrenergic antagonists decrease production of aqueous humor.

Oral and intravenous hyperosmotic agents lower IOP rapidly and are used in the emergency treatment of acute glaucoma.

Surgical Therapy
Cyclocryotherapy has been found to be effective in decreasing production of aqueous humor by the transcleral freezing of the ciliary body with nitrous oxide. The most effective surgical or medical therapy for glaucoma is the gonioimplant. Enucleation or evisceration with prosthetic silicone implants is indicated when vision is lost in uncontrolled glaucoma. The source of pain is removed, and no further medication is necessary. The cosmetic appearance of the prosthetic implant is sometimes preferred to that of enucleation.

Inherited Retinal Disease

Collie Eye Anomaly
- Congenital (present at birth)
- A developmental anomaly of the choroids, ± optic nerve and sclera
- Inherited as a simple autosomal recessive, but probably is much more complex than this
- Stationary (non-progressive – except for retinal detachments and intraocular hemorrhage)
- Bilateral asymmetry of the signs
- Collies (rough/smooth), Border Collie, Shetland Sheepdog
- In USA approximately 85 percent of Collies are clinically affected but this number is declining (approximately 5-10 percent of Shelties). Obviously with a recessive condition this means that most "normal" dogs are carriers
- Signs
    - Choroidal hypoplasia: A "pale" area of varying size of depigmentation temporal to superotemporal to the optic disc. This may be an area of RPE and choroidal hypopigmentation, tapetal hypoplasia and choroidal dysplasia. Choroidal hypoplasia is seen in all cases of CEA.
    - Colobomas of posterior pole (often of optic nerve; seen in 30 percent of cases) – may be associated with visual deficit
    - Retinal detachment (5-10 percent of cases)
    - Retinal or vitreous hemorrhage of hyphema
    - Blindness (3-4 percent of cases); many dogs with this disease show no visual abnormalities, unless severe colobomas or retinal detachment are present. Other anomalies seen in Collies may include retinal folds (vermiform streaks) and subepithelial corneal opacities. These dogs are invariably microphthalmic. Retinal dysplasia (folds) and corneal dystrophy may also be seen in "normal-eyed" Collies; i.e. they are not necessarily part of the CEA syndrome. Tortuosity of retinal vasculature is not per se a feature of the disease although may accompany microphthalmia.
- Some dogs have only minor choroidal hypoplasia. These pale areas may become masked with pigment as the dog's fundus matures. Thus by twelve months of age, although genotypically affected, may appear clinically normal and are termed "go-normals."
- Diagnosis can be made at 4-8 weeks of age. Severely affected puppies should be identified at this time. Due to the prevalence in the population, eradication is very difficult. Breeding minimally affected dogs does not necessarily avoid severely affected offspring.

Retinal Dysplasia

- Not really a specific disease – an anatomic retinal abnormality that may occur alone or in conjunction with other ocular abnormalities. This is a separate entity from photoreceptor dysplasia seen in progressive retinal atrophy
- Congenital – seen in puppies
- Stationary (non-progressive usually)
- Can be inherited (recessive and maybe dominant forms) or acquired due to intrauterine inflammation and retinal necrosis (canine herpes virus or irradiation)
- Signs
    - Retinal folds – seen as streaks, dots or Y or V shapes in the tapetal fundus, often around the superior retinal vessels; may be green, grey or dark in color or grey or black streaks in the nontapetal fundus
    - Retinal rosettes: histiologic description of dysplasia
    - Focal retinal degeneration
    - Retinal detachments $\pm$ hemorrhage, if dysplasia is extensive
    - Leukocoria
    - Microphthalmia
    - Cataract
    - Nystagmus
    - Blindness if dysplasia is sever
- Localized retinal dysplasia seen in American Cocker, Spaniel, English Springer Spaniel, Beagle, Labrador Retriever, Rottweiler, Samoyed, Golden Retriever and potentially in other breeds
- In the Sealyham and Bedlington Terriers the disease is a vitreo-retinal dysplasia
- In the Labrador Retriever at least one form of retinal dysplasia is associated with skeletal dysplasia
- Diagnosis of retinal dysplasia at 4-8 weeks of age by ophthalmoscopy – avoid breeding affected dogs.

Retinal Dysplasia with Multiple Ocular Anomalies

- In the Australian Shepherd retinal dysplasia occurs with:
    - Microphthalmia $\pm$ microcornea
    - Heterochromia irides
    - Cataract
    - Choroidal hypoplasia
    - Colobomas/Staphyloma
    - Retinal detachment
    - Superficial cornea opacity
- Similar abnormalities have been reported in the Great Dane, Merle Collies and Old English Sheepdogs
- Inherited in the Merle Australian Shepherd. Etiology unknown in other breeds.

Progressive Retinal Atrophy (PRA)

- Inherited retinal photoreceptor dysplasia (young animals) or degeneration (mature animals)

- Usually not congenital
- Not stationary (eventual blindness – in some forms of the disease)
- Generalized progressive retinal atrophy; blood tests becoming available for some dog breeds
    - Inherited in most cases as simple autosomal recessive
    - Progressive loss of vision – night vision first, then day vision
    - Breeds affected: Irish Setter, Collie, English/American Cocker Spaniel, Miniature/Toy Poodle, Norwegian Elkhound, Afghan Hound, Border Collie, Cardigan Welsh Corgi, Beagle, Borzoi, Dachshund, Cairn Terrier, English Springer Spaniel, English/Gordon Setter, Greyhound, Labrador Retriever, Miniature Pinscher, Pointer, Saluki, Siberian Husky, Samoyed, Swiss Hound, Shetland Sheepdog, Tibetan Terrier
    - Variable age of onset, e.g. in the Irish Setter and Collie PRA can be diagnosed by ophthalmoscopy by 6-8 months of age. In Miniature Poodles PRA may develop anywhere between 3 and 12 years of age. Other breeds tend to be in between these extremes
    - Signs: Night blindness progressing to total blindness; slow and incomplete pupillary light reflexes (maybe); tapetal hyper-reflectivity (retinal thinning); nontapetal depigmentation and pigment clumping; retinal blood vessel narrowing and loss, especially arterioles starting at the periphery; optic nerve atrophy; cataracts form due to release of toxins from the diseased retina
    - Diagnosis: behavioral signs, maze test, ophthalmoscopy, electroretinogram
    - Etiology: in the dogs with early onset (Collies and Irish Setters) PRA, it is due to a deficiency of enzyme cGMP phosphodiesterase: allows accumulation of cGMP which is toxic to photoreceptors. Other types of PRA are photoreceptor dysplasia or degeneration.
    - No treatment at present

Sudden Acquired Retinal Degeneration (SARDS) or "Silent Retina Syndrome"

- Sudden loss of vision (DDX optic neuritis, retinal detachment)
- No ophthalmoscopic signs at first; later (months) see typical hyperreflectivity, etc., of retinal degeneration
- ERG extinguished from the onset (hence "silent retina")
- Especially middle-aged, slightly obese female dogs that are also PU/PD (? "Cushingoid"); seen in Poodles and Dachshunds
- Etiology unknown, toxic degeneration or metabolic disorder; may be related to imbalances of MSH and ACTH and the resulting metabolic disturbances of the RPE; vitreal glutamate levels are high

Advanced Reproduction Symposium
Robert V. Hutchison, DVM

Maximizing Neonatal Survival

Multiple studies have indicated that 20-30 percent of all puppies of normal gestation length die before reaching six months of age. Of the many causes of neonatal death, prolonged labor, hypoxia and dystocia are among the primary causes. To avoid puppy loss the breeder and the veterinarian need to be aggressive when signs of labor problems appear. A Caesarian section is a commonly performed surgical procedure that can be rewarding to both the client and the veterinarian.

Caesarian sections are performed for a number of reasons:

1. **Puppies are not deliverable**: pelvic shape or size, overly large puppy or a mal-positioned pup necessitates quick action to save the litter

2. **Uterine inertia**: the progesterone level dropping below 2.5 ng and the pre-whelping temperature drop (a monitoring of the progesterone level dropping) returning to normal (101º-102º) should initiate active labor. Treatment with oxytocin at6 this time may help to actuate labor. Any signs of fetal stress should suggest a more aggressive approach.

   Oxytocin will stimulate uterine contraction and initially increase the blood flow to the uterus thereby increasing the oxygen level to the feti. If oxytocin is continued to be given the uterine blood vessels dilate and the blood pressure to the uterus drops, subsequently robbing the puppies of oxygen. The only factors affecting the oxygenation of the puppies in utero are the uterine blood pressure and the fetal heart rate.

   A good "rule of thumb" when using oxytocin to aid delivery is to give one dose either intramuscularly or subcutaneously. An average dose should be two units/ten pounds body weight (i.e. a 40 lb bitch would receive eight units). Most oxytocin is 20 units/ml. If no response is noted in the bitch within 20 minutes a second injection of oxytocin is given. If after the second injection there is still no response, a C-section should be performed. Further injections of oxytocin can be detrimental.

3. **Signs of in-utero fetal stress**: Black, red or green vaginal discharge before any puppies are delivered signifies placental separation and blood leakage. The puppies should be immediately evaluated for stress by monitoring the fetal heart rates. Normally the fetal heart rate should be twice that of the bitch. The use of ultrasound and Doppler will evaluate the strength and rate of the fetal heart. A slowing of the fetal heart can be the deciding factor as to whether a Caesarian is needed. The evidence of fetal bowel movements in utero can also indicate fetal stress.

4. **Convenience**: A planned C-section can be a method for the breeder to assure the most live puppies. Timing of the Caesarian section is critical to assure fetal health.

Puppies need a full gestation period to be ready to survive outside the womb. A bitch's anticipated due date can be calculated as 63 days post ovulation of 65 days post LH surge. Breeding date cannot be used to calculate the whelping date of a bitch.

Timing for a planned Caesarian section:

- Calculated due date based on progesterone of LH spike at breeding time
- Body temperature of bitch below 99.5º
- Progesterone level below 3 ng
- Active labor

Anesthesia for a Caesarian Section

As all anesthetics cross the placenta to some degree, it is a challenge for the veterinarian to choose an appropriate drug for maximum control and comfort for the bitch but not so depressive as to cause puppy lethargy, hypoxia or even death.

For an anesthetic to be considered for a Caesarian it must:

- Cause rapid induction
- Be safe for the bitch
- Be safe for the puppies
- Allow for rapid recovery
- Not have prolonged after effects

An anesthetic given for a C-section must allow for endotracheal intubation. Gastrin, a hormone secreted by the placenta causes a lowered PH (increased acidity) of the stomach acid. If a pregnant bitch vomits while undergoing or recovering from surgery and aspirates into her lungs the results could be fatal.

Many anesthetic protocols exist for use in veterinary medicine. However most do not meet the criteria set forward for safety in a C-section.

Local/Epidurals: Do not allow for intubation of the bitch and also provide no oxygen source
Tranquilizers: Depress blood pressure resulting in fetal hypoxia; also depresses pups
Dissociatives: Increase blood pressure to the uterus, but must be used with a tranquilizer causing cardio-vascular and respiratory suppression
Opiates: May require up to 4-6 days to totally clear the puppies' systems causing depression in the puppies
Barbituates: Small doses cause minimal depression, but larger doses can be very depressing to neonates and the bitch
Inhalants: Isoflurane and Sevoflurane are minimally absorbed and clear the bitch and puppies very quickly
Propofol: Crosses placenta, but clears puppies and bitch very quickly

Currently no perfect anesthetic for Caesarian section exists. The better protocol currently available includes the use of Propofol for induction and intubation of Isoflurane and Sevoflurane

for maintenance of the bitch. These drugs cause minimal suppression, quick induction and recovery and no lingering side effects.

Preparation for C-section

Maternal stress, pain and anxiety are detrimental to the feti. An essential element in preparing a bitch for a C-section is for the owner, veterinarian and medical staff to avoid mental or physical stress to the bitch.

The bitch is pre-treated with Atropine sulfate, which crosses the placenta, and helps maintain the fetal heart rate. The bitch should have an intravenous catheter and fluids running before and during surgery to maintain normal blood pressure throughout the surgery. Pre-oxygenation of the bitch before anesthesia will help to avoid fetal acidosis should the bitch become apnic (stop breathing) during induction of the anesthesia.

Surgery

Once the bitch is in an appropriate surgical anesthetic plan, she is placed in dorsal recumbency. Care sho8uld be taken not to tip the table as is often done in abdominal surgery, as this forces the gravid uterus against the diaphragm, inhibiting respiration. An incision from the pubis to the umbilicus should initially be made. The incision may need to be extended if the uterus is overly distended with puppies. Care should be taken to not expose too large a surface area of the uterus to the air, as this can cause hypothermia, especially in small bitches.

Incisions in the uterus are made to allow quick removal of the puppies. The surgeon should be very sure that all puppies have been removed from the uterus. Any placental tissue still left in the uterus should be removed before closing.

An inverting pattern of the surgeon's choice, with an absorbable suture material, is used to close the incision sites. If the uterus is slow to contract, an injection of oxytocin should be given intra-muscularly. The muscle and skin closure is routine.

The bitch is removed from the table and once the abdomen is cleaned, the puppies are allowed to nurse. If proper fluid perfusion and blood pressure have been maintained, the milk flow is not compromised by the surgical procedure and future breeding is not comprised.

Suggested Reading

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Isoflurane: Abbott Laboratories, North Chicago, IL 60064
Propofol: Schering-Plough Animal Health Corporation, Union, NJ 07083
Sevoflurane: Abbott Laboratories, North Chicago, IL 60064

Maximizing Conception Rates

Since the American Kennel Club's recognition of litters conceived from frozen semen in 1981 and the subsequent acceptance of fresh chilled semen, practitioners are being asked more frequently to assist clients with maximizing conception rates. The gratification one feels when successful is one of the great rewqr4ds in veterinary medicine.

The bitch is uni2que when compared to other domestic animals. The ova are ovulated in a progesterone "bath" as opposed to the estrogen environment of other species. The uterus is not easily accessible due to the cervix being difficult to reach, small in diameter and irregularly shaped (pointing caudal ventral into the vagina and cranial-dorsal into the uterus).

The bitch ovulates an immature egg that requires a further meiotic division before fertilization can occur. All this process takes place in an estrous cycle averaging 21 days of which the fertile period is approximately 72 hours.

Ovulation timing in the bitch has been attempted in many methods including:

- **Physiological Signs**
- **Estrogen Monitoring**
- **Lutenizing Hormonal Spike**
- **Progesterone Rise**

Physiological Signs: Flagging*:* softness of the vulva, and color of discharge have historically been used to determine the proper time to successfully breed a bitch. The fact that fresh semen lasts for a number of days in the bitch allows for "rough guesses" of ovulation to be successful in achieving conception. Multiple breedings also cover a wide range of time making conception likely

The recent popularity of fresh-cooled extended semen, frozen semen and limited availability of desired males has made a more specific method of ovulation timing mandatory.

Estrogen Monitoring: The use of estrogen for anticipating the ovulatory timing o9f the bitch is not reliable. The most frequently used method of estrogen monitoring has been through the use of vaginal smearing. By taking a cotton swab and sampling the cells lining the vaginal tract one can gauge the development of the ovarian follicles.

As the estrogen level rises, the vaginal wall thickens. As the serial samples are taken, one compares the cellular hydration, cytoplasmic/nuclei ration and the staining of the chromatic material. As the estrogen level continues to rise, the vaginal wall will reach a 25-30 cell thickness. The cells dehydrate and become "cornified."

The dehydration of the vaginal epithelial cells does not, however, reach a peak but rather stays in a greater than 80 percent cornified state for a period of six to ten days. This does not equate with ovulation timing or indicate that ovulation has actually occurred. Many other methods of estrogen timing in the bitch have been tested, but due to the uniqueness of the bitch's ovulatory process, estrogen is not an acceptable nor a consistent method of timing the bitch's breeding pattern.

Lutenizing Hormone (LH): Lutenizing hormone (LH) from the pituitary gland triggers the release of the ova from the follicles. The release of the LH occurs for a 12-24 hour period. Urinary testing for LH was unsuccessful due to the necessity of collecting the first morning urine from the bitch and the fact that the LH is metabolized into different fractions, some were detectable in the urine by commercial testing, other fractions were not.

The serum testing for lutenizing hormone has been used successfully to anticipate the release of the ova. An initial rise of progesterone from the ovarian follicle triggers the pituitary to release the LH. The short duration of the LH in the bloodstream requires daily testing that can be time consuming to the client, expensive and uncomfortable to the patient. LH is species specific and levels cannot be confirmed by sending the serum to an outside lab. No laboratory in the United States is currently running canine LH on a commercial basis.

Progesterone Testing: The release of progesterone from the ovary and its subsequent rise to an average of two to three nanograms (ng) signals lutenizing hormone release from the pituitary gland and denotes the start of the ovulatory process. The rise of progesterone to greater than five ng (5-8 ng) indicates that ovulation has occurred. Once the ovulation day has been confirmed, insemination timing can be planned depending on the anticipated life of the semen being used.

The bitch needs to be tested every 48-72 hours to anticipate the prime breeding time. Progesterone rises and continues to stay elevated for approximately two months in the bitch, whether she is pregnant or non-pregnant.

The normal time interval between LH release and ovulation is 48 hours. Cortisol release needs to be a concern when doing progesterone testing as stressed bitches may have a delayed period between a rise of two to three ng and the time that the progesterone rises greater than five ng. Some bitches under stress have been shown not to ovulate even after a rise of progesterone above

two ng. It is essential, when using progesterone testing, to confirm that the bitch has risen above five ng and has indeed ovulated.

Reported problems have made the in-office use of progesterone Elisa tests of questionable value. Hopefully these problems can be corrected and these valuable tests can be returned to the veterinary laboratory.

Methods of Breeding

Natural: If the male is proven, local and available, a natural breeding is the preferred method. Progesterone testing is still necessary to confirm that the breeding corresponds with the ovulation.

Vaginal Artificial Insemination: If timing is correct, but a natural is not possible nor desired, a vaginal insemination will enable the semen to be deposited at the external cervical opening. This method can also be applicable when a natural breeding can be a risk for disease, injury to the dogs or an inconvenience to the owner.

The drawback to a vaginal insemination is that the semen is deposited in the vagina and must be properly positioned for it to be drawn into the uterus so that the sperm cells can swim to the fallopian tubes (around the ovaries) where conception occurs.

When proper timing of the estrous cycle is performed and proper semen handling and delivery is accomplished, conception rates should rival those of natural breedings. Delivery technique problems such as improper placement of the insemination rod, improper semen placement, or semen damaged due to mishandling, have unfortunately convinced many dog breeders and veterinarians that artificial inseminations should be only used as a last ditch measure.

The bitch is positioned with her rear elevated either manually or on a breeding ramp. Care should be taken to avoid pressure on the bitch's abdomen. The semen is drawn from the collection tube through an insemination rod of proper length to reach the cervical opening. It is important that the semen be deposited at the entrance to the cervix so that the semen can be drawn into the uterus. With gloved hands, the veterinarian gently inserts the insemination rod through the lips of the vulva at tan upward 45º angle. The rod is gently passed over the pubis and along the dorsal median fold until it is parallel with the lumbar spine and localized in the area of the cervix. If resistance is encountered, the rod should be gently twisted or withdrawn a short distance, then advanced again.

When the insemination rod is properly positioned, the semen should be gently inseminated. The syringe is then removed from the rod. It is not necessary to push large amounts of air into the rod or normal to get semen back flow if the rod is properly positioned and the bitch is in the proper stage of estrous. Excessive air "bubbling" through the semen is detrimental to the fragile plasma membrane of the head of the spermatozoa.

The bitch is then "feathered" digitally for one minute; the rear of the bitch is maintained in an elevated position for a few minutes to allow gravitational feeding of semen to the anterior

vagina. The bitch owner is instructed to confine the bitch or restrict her activity for one to two hours post-insemination.

Intra-uterine Deposition of Semen

A technique for surgical insemination in the bitch was first described in 1974. The intra-uterine deposition was initially used to increase poor conception rates in the use of canine frozen semen. Since that time, deposition of semen into the uterus has become a routine technique used in numerous situations encountered in canine reproductive me3dicine, resulting in dramatically improved conception rates.

Situations where this can be helpful include:

Frozen Semen: Due to the lack of spermatozoa energy, buffer chemical makeup, or cervical resistance, the conception rates from cryo-preserved canine semen have been historically low when used with a vaginal insemination. Deposition into the uterine lumen has resulted in conception rates equal to those from natural breeding.

Fresh Cooled Extended Semen: Shipment of semen rather than shipping of the bitch has become commonplace. Due to shipment time and spermatozoa energy depletion, numerous clients have chosen intra-uterine semen insemination for their bitches in hopes of increasing conception rates.

Bitches with suspected uterine or ovarian disease: The ability to access uterine and ovarian health at breeding time is advantageous to clinicians presented with bitches with histories of reproductive failure. This can be accomplished doing a surgical insemination.

Giant and Toy Breeds: Individuals on the extreme ends of size have historically been recognized for conception difficulties. The ability to overcome anatomical barriers and inseminate directly into the uterus has increased litter numbers and relieved client frustration.

Males with lowered or compromised spermatozoa numbers: Little work has been done in the canine to definitively define minimum semen parameters necessary for conception. By directly depositing the semen into the uterus and bypassing the cervix and vaginal vault, conception can be achieved with lesser sperm numbers and lesser overall semen quality.

Introducing the semen into the uterus can be accomplished using two different methods:

1. **Trancervical Insemination (TCI)**: This technique entails the use of a cysto-urethroscope using fiber optics to visualize the external opening of the cervix. A flexible catheter is maneuvered through the external cervical opening and subsequently into the uterus.

   The TCI requires no anesthesia and minimal restrictions for the bitch. This technique allows the semen to be deposited into the uterus but does not allow visualization of the uterine lining by the veterinarian. This technique is most useful with compromised

semen life, but a suspected normal uterus in the bitch. The technique has shown to not be useful for endometrial biopsy.

2. **Surgical Intrauterine Deposition of Semen**: A pre-surgical evaluation is performed on the bitch. Anesthetic induction consists of coning smaller individuals with gas anesthesia (i.e. Isoflurane, Sevethane). Larger bitches may require an injection of a standard, short acting anesthetic (i.e. Propofol) to achieve relaxation for intubation and maintenance with gas anesthesia.

   The bitch is prepared in the same manner as a bitch undergoing an ovario-hysterectomy. The ventral abdomen is clipped and the bitch is placed in the surgical theater in dorsal recumbency. Prepping of the surgical site is done in a routine manner. The abdomen is draped in preparation for the surgery.

   A four to six centimeter incision is made midway between the pubis and the umbilicus. The incision is made in the skin, subcutaneous fat and through the linea alba. The uterus is identified and elevated to the surface though the incision. The uterus is draped with a saline moistened laparotomy pad as the semen is prepared for the injection procedure.

   A volume of semen varying between .5 ml and four ml is prepared for insemination. If the volume is greater than four ml, the semen should be centrifuged for five minutes. The supernatant is decanted and disposed. The semen pellet is gently re-suspended with the remaining supernate or with a semen extender.

   The semen to be injected is gently drawn into a six ml syringe through an insemination rod. The rod is removed and a 22-gauge ¾ inch needle is attached to the syringe.

   The surgeon inserts the needle into the lumen of the uterine body at a 45º angle with the bevel of the needle up. The semen is slowly injected into the uterus. The semen should flow easily with obvious distention of the uterine horns. If the injection cannot be achieved or is difficult, the needle should be repositioned.

   Saline moistened gauze is held over the injection site after the needle is withdrawn. After one minute, the gauze is removed and the uterus is replaced into the abdomen. Closure of the fascia muscle, subcutaneous tissue and skin is the routine procedure.

   The bitch's rear is elevated as she recovers from the anesthesia. The intra-uterine pressure during the surgical deposition of the semen may cause mild back flow through the cervix into the vaginal cavity. This appears to be of no concern. If the surgeon has doubt as to the semen being placed in the uterine lumen, a post-operative vaginal smear will confirm spermatozoa, if the technique has been performed properly.

Feeding Dogs for Life Stages

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Life Stages: Much overlap in requirements
Maintenance
Gestation (Pregnancy)
Lactation
Growth
Old Age

Maintenance

Types of Pet Food

1. **Dry food** contains very little moisture so as feed analysis reported on bag is similar to dry matter analysis. They are formed by an extruder and are **mostly low fat** (~25 percent energy as fat) because the extruder requires a low fat mixture. These are fine for dogs which are couch potatoes but may not contain enough fat for gestation, lactation or growth, or for dogs that undertake a lot of exercise. They **may not give optimum coat quality**. More expensive dry diets have fat sprayed on after extrusion and tend to contain more fat (40 percent energy as fat). They are packaged in special grease proof bags and are greasy to the touch.
2. **Canned foods** contain 75 percent moisture and are more expensive but usually contain more fat and protein. Multiply the analysis on the bag by four to compare with a dry diet.
3. **Soft-moist and Soft-dry** e.g. Kibbles and Bits. These are intermediate but mostly low fat

Differences between pet foods

Foods with different names do not necessarily differin composition. Marketin gstrategies aim to increasemarket share often by increasing number of brands and increasing shelf space. Differences between pet foods are often small because new brand names are created with small changes in composition as a method of increasing the number of brands and because the final composition is restricted by the nutrient requirements of the animal, the need to restrict cost and to maintain palatability. Some terms such as “premium” and “super-premium” have no definition and do not guarantee better performance. The major differences are:

- **Dry vs. Canned**: Canned usually contains more fat and protein than dry diets
- **Generic vs. proprietary**: Generic diets are usually made with poorer quality ingredients and better ingredients and are usually tested on animals. “Generic” diets are inexpensive private label of local or regional manufacturers. There is no policing of label claims if only sold within the state of Florida.
- **Life stage and therapeutic diets**: Sometimes have different compositions but diets for puppies and diets for adult maintenance are often very similar in composition.

Supplements and treats unbalance balanced diets so should be avoided or restricted to less than ten percent of the diet. Chews may be beneficial for dental hygiene.

Human food is not complete and balanced so must have supplements such as vitamins and minerals added if more than ten percent of the diet. Uncooked meat represents a likely source of infection especially in young and pregnant animals. Bones, especially spiky bones such as the vertebrae found in chicken necks can get lodged in the esophagus especially in small breeds of dogs. Too many bones can also cause constipation.

Neutraceuticals and herbs: Quality, consistency, absorption, potency and efficacy are uncertain. Toxicity and therapeutic indices have not been established; more of a risk in young and pregnant animals. Nevertheless, likely that some of these will prove beneficial in the future.

How much food?
Adjust food intake to maintain optimum body weight and condition. Do not feed too much. The slim-line model is best. *Ribs should be felt but not seen. There should be a waist visible from the side and from above.* The recommendation on the back of the packet can provide a guide but there is much individual variation. Reproductive performance may be sub-optimal if animals are too fat or thin.

Pregnancy (9 weeks)
First five to six weeks: bitch should maintain an ideal body condition score during mating and early pregnancy. There is no need for increased food intake above normal. Reasons for unsuccessful mating are usually poor timing, not diet.

Last three to four weeks: Most fetal growth occurs during the last trimester so increase food intake by ten to 15 percent per week. Protein requirements are high during this period sop do not feed a low protein food. A higher fat high protein canned diet can be added to the usual dry diet. Growth diets or therapeutic diets do not always contain enough protein. **Do not feed supplements.** Only exception would be to add folate to the diet of Bulldogs and Rhodesian Ridgebacks. There is plenty of folate in pet foods so supplementation may not be necessary but in humans additional folate minimizes the risk of spina bifida. Supplementation may minimize the risk of spinal defects in breeds where these defects are more common.

At the end of pregnancy: Body weight should have increased ten to 15 percent and intake should be 40 to 50 percent more than usual. The gravid uterus often limits intake, however, so feed energy dense (high fat) food in small frequent meals. Most of the additional extra body weight should then be lost when she gives birth.

Eclampsia: Hypocalcaemia is observed occasionally in dogs **around parturition** leading to tetanus, seizures and poor uterine contraction. The cause has not been established. Nevertheless, it seems wise to follow similar practices as those used to prevent milk fever in cattle (e.g. calcium supplements should not be given before parturition as it will reduce the ability of the body to respond to the calcium requirements of parturition.) **This is an emergency. Treatment consists of slow intravenous administration of ten percent calcium gluconate** and limiting pups suckling (raise pups with milk replacer to minimize milk needs).

Lactation: Food intake increases with milk production to a peak three to four weeks after whelping. Water intake increases proportionately so free access to water is essential. Amount of increase varies with size of litter to as much as three or four times normal. Frequent meals of an energy dense (high fat) high protein food are recommended. Diets should also contain some carbohydrate for optimum lactose production.

Bitches milk is best!

- Colostrum contains antibodies so must ensure pups get colostrums or severe risk of overwhelming infection is present.
- Bitch's milk is twice the energy density and contains more protein, fat, calcium and less lactose than cow's milk.
- Breast milk varies in composition during lactation (early milk contains almost lo lactose). The enzymes and transporters in the puppy intestine that digest and absorb nutrients like lactose are switched on and off in a programmed fashion, so diarrhea and/or constipation are possible if milk replacer or pet food is different from that expected by the pup at any particular age.
- Milk also contains other substances such as epidermal growth factor that may influence the development of the puppy intestine.

Orphan Rearing

- Feed with warm milk replacer (e.g. Esbilac or using homemade recipe: ½ cup cow's milk, 2 egg yolks, ½ tablet Centrum Junior, 1 tsp dicalsium phosphate).
- Volume per day is about 15 percent of body weight during first week then 25 percent during subsequent weeks assuming the food contains approximately 1 kcal/mL.
- Monitor body weight daily. Puppies must not lose weight.
- Keep pups warm and humid (85-90ºF, > 50 percent humidity).
- Establish feeding frequency from pups crying. Do not wake to feed. Feed often first day (every two to four hours) and then can reduce to four times daily and can leave overnight if kept warm.
- Stimulate to defecate and urinate after feeding.
- If possible, foster pups on another bitch. Alternatively, divide litter in two and alternate during day between bitch and orphan feeding.

Neonatal Feeding

- Days 1-3: suckle 40 times daily for total of ten to 15 hours per day.
- Day 4: suckling more efficient so only eight hours per day.
- Days 10-12: Eyes open, start to examine solid food.
- Week 3: Offer moist palatable energy dense food four times daily. Remove any left after an hour.
- Gradually increase time away from dam and then wean at seven to eight weeks of age. Reduce bitch's food for two days prior to weaning to cut milk production.

Growth

- **Energy requirements**: from three to four months on, feed about the same amount of food as that required for maintenance by parents. Amount of food required by Great Dane pups and adults is greater than that required by other breeds of similar weight.
- **Rate of growth**: very variable depending on size. Larger breeds take longer to reach adult weight (Yorkshire Terriers: ~ eight months; Newfoundlands: ~ 18 months to two years). Limit rate of growth in large breeds to minimize hip dysplasia and growth deformities. Keep lean. Restricted intake does not affect final height, length, bone size or muscle mass.
- **Puppy Food Composition**:
    - Feed an increased **protein** and increased fat growth diet up to four to six months of age. "Large breed dog diets" contain less fat to limit the rate of growth. There is currently no evidence that these diets cause less orthopedic problems than higher fat diets if intake is restricted to prevent rapid weight gain.
    - **Calcium should be close to one percent DM (3 mg/kcal) with a CA:P ration of 1:1 to 2:1**. Pups fed all meat diets and insufficient calcium develops osteoporosis and pathological fractures. Large breed dogs fed too much calcium can develop osteochondritis dessicans. Most commercial puppy foods including those marketed for large breeds contain this amount of calcium. The calcium content in adult maintenance diets are sometimes closer or above two percent DM. Amount of protein has no effect on orthopedic problems.
    - Do not feed supplements especially in large breed dogs. Large dogs eat more food so eat more vitamins and minerals. Increased calcium and vitamin D may be detrimental.

Neutering and Spaying

Increased risk of obesity because reduces energy requirement by one third and increases appetite.

Old Dogs

- Keeping a dog lean is the best method for prolonging age
- Antioxidants may be beneficial; doses are uncertain however and some diets have these added so that additional supplementation should only be administered under veterinary supervision
    - Vitamin E up to 10-15 IU/kg daily or 100 IU/kg weekly by month
    - Vitamin C needs to be given three to four times daily to increase blood levels
- Energy needs decline with age
- If have disease then may need special diet; most require extra protein and not less
- If thin needs extra calories, if obese needs less
- Needs to maintain intake of essential nutrients

Recommendations:

- Feed a national brand pet food that has been tested using AAFCO approved feeding and is complete and balanced for the particular life state (e.g. reproduction or growth or adult maintenance).
- Do not feed supplements. A pet food that is complete and balanced does not require supplements. Do not feed extra meat, calcium or vitamin supplements especially in growing or pregnant animals.

- Treats are ok but try to keep to a minimum. The bulk of an animal's diet should come from pet food.
- If feeding mostly human food, make sure it is cooked and balanced for the particular life stage.
- Coat quality can be poor if dogs are fed a low fat inexpensive dry food. To improve fat content of the diet, do not add meat to the diet. It is safer to use a more expensive high fat dry diet or to add a canned diet to the dry diet. High fat is here defined as greater than 40 percent energy which is equivalent on the label to greater than five percent fat for a canned diet, greater than 13 percent in a semi-moist diet and greater than 18 percent fat in a dry diet.
- Do not restrict protein or other essential nutrients unless specific disease necessitates this.
- Keep animals lean!!!

An Introduction to Acupuncture

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Acupuncture represents an ancient art of medical practice which has its roots in antiquity. Perhaps the earliest record of acupuncture dates to the "Ice-Man" found in the Italian Alps, who had a tattoo over a know acupuncture point which had to have been placed there by another person. On MRI (Magnetic Resonance Imaging), the Ice-Man had an intervertebral disc herniation which should have been painful and which might have responded to acupuncture treatment at the tattooed point.

Acupuncture is most closely associated with ancient China where it remains part of Traditional Chinese Medicine (TCM). The first written record of acupuncture is found in the 4,700 year-old Huang Di Nei Jing (Yellow Emperor's Classic of Internal Medicine), one of the oldest medical textbooks in the world. It contains the earlier theories by Shen Hung, the father of TCM. Shen Nung documented theories about circulation, pulse, and the heart over 4,000 years before Western medicine had any co9mcept about them. The first veterinary text was written in China around 450 BC, dealing with the treatment of horses with acupuncture and TCM herbal medicine.

Interest in acupuncture in the United States exploded in the 1970s following President Nixon's historic trip to China, where demonstrations of surgical analgesia using acupuncture were made. Reportedly, one of Nixon's aids was ill and responded almost immediately to acupuncture treatment, even though Western medicines were not working. This led to a great increase in scientific interest in acupuncture in the West, producing over 8,000 scientific references to acupuncture since that time. In animals, the cited references make up about ten percent of the total publications.

While acupuncture in human beings and domestic livestock are referenced in China for over 2,000 years, recently some have criticized that acupuncture in dogs and cats is perhaps only 25 years old. This may be true, since dogs and cats were not considered important animals in ancient China and certainly were not an integral part of the household as they have become in the West until recently. On the other hand, the principles of TCM and acupuncture for pet species does have its roots in olden times.

Acupuncture (Greek: *acus*: needle; *pungare*: to pierce, to puncture the skin with a needle) may be defined as the insertion of needles into specific points on the body to cause a desired healing effect. In TCM, acupuncture includes using fine-needle (dry needles) acupuncture, hemoacupuncture (blood letting), moxibustion (burning of the herb, *Artemisia vulgaris*, over acupuncture points), pneumoacupuncture (insertion of air under the skin), acupressure, and firing (counter-irritation with heat). In modern times, constant stimulation of the needles has been replaced by electrical acupuncture. Acupuncture (injection of dilute solutions into acupuncture points) and gold-bead implants (insertion of small metallic particles at certain acupuncture points) have also become popular. Low-power lasers and static magnets can also be used to stimulate certain superficial acupuncture points.

Successful application acupuncture depends upon three important factors: the acupuncture point; the method of stimulation; and the response observed. Modern experiments have shown that acupuncture points occur where nerves penetrate tissue planes or where nerves, themselves, divide. As such, there are four major types of acupuncture points. Type I (motor) points are the most common and exist where nerves penetrate muscles. Type II points are located where nerves intersect on the dorsal and ventral midlines of the body. Type III points are located where superficial nerves branch. Finally, Type IV points are located where nerves penetrate tendons (Golgi tendon organ). Most acupuncture points are located at areas of low electrical resistance and high electrical conductance of the skin. Deep in the point, there are accumulations of free nerve endings; small arterioles, veins and lymphatics; and tissue mast cells. Stimulation of these points results in degranulation of the mast cells, activation of the inflammatory cascade, alterations of blood and lymph flow, and conduction of nerve impulses to the central nervous system. This results in a local response which spreads over time throughout the entire neural axis evoking numerous biochemical changes within the nervous system and eventually the whole body. The initial sensation is termed, "deQi," meaning "the arrival of "Qi." In this context, Qi can be interpreted as "energy."

As the basis of ancient acupuncture, Shen Nung theorized that the body had an energy force running throughout it. This energy force is known as Qi (roughly pronounced Chee). The Qi consists of all essential life activities that include the spiritual, emotional, mental, and the physical aspects of life. A person's health is influenced by the flow of Qi in the body, in combination with the university forces of Yin and Yang. If the flow of Qi is insufficient, unbalanced or interrupted, Yin and Yang become unbalanced, and illness may occur. Qi travels throughout the body along "Meridians" or special pathways. The Meridians (or Channels) are the same on both sides of the body (paired). There are fourteen main meridians running vertically up and down the surface of the body. Out of these, there are twelve organ meridians in each half of the body. There are also two unpaired midline meridians. The acupuncture points are specific locations where the meridians come to the surface of the skin, and are easily accessible by acupuncture, moxibustion, and acupressure. The connections between them ensure that there is an even circulation of Qi, a balance between Yin and Yang. Energy constantly flows up and down these pathways. When pathways become obstructed, deficient, excessive or just unbalanced, Yin and Yang are said to be thrown out of balance. This causes illness. Acupuncture is said to restore the balance.

Yin and Yang is an important theory in the discussion of acupuncture treatment, in relation to the Chinese theory of body systems. Qi is prevalent throughout all of nature and is comprised of two parts, Yin and Yang. Yin and Yang are opposite forces, that when balanced, work together. Any upset in the balance will result in natural calamities, in nature; and disease in human beings. Yin is signified by female attributes: passive, dark, cold, moist, that which moves medially, and deficient of Yang. Yang is signified by male attributes: light, active, warm, dry, that which moves laterally, and deficient of Yin. Nothing is completely Yin or Yang. The most striking example of this is man himself. A man is the combination of this mother (Yin) and his father (Yang). He contains qualities of both Yin and Yang forces. Within Yin, there is Yang, and within Yang, there is Yin.

As the ancient Chinese looked upon their world and observed nature, they found universal relationships in all things. They descr4ibed these events and used them to develop TCM principles of health and disease. From these observations, a number of medical theories emerged. The most commonly used are five-element theory, eight principles of disease, and ZangFu (organ) physiology. TCM today incorporates aspects of each of these theories in determining the disease pattern and treatment plan.

Nature is made up of combinations of wood, fire, earth, metal and water. Each element represents an energy, direction, season, color, taste, feeling, ZangFu organ and tissue. Wood is warm, east, spring, green, sour, anger, liver-gall bladder, and tendons and ligaments. Fire is hot, south, summer, red, bitter, joy, heart-small intestines/pericardium-triple heater, blood vessels and tongue. Moreover, individuals tend to have certain characteristics that can be explained by their elemental constitution. Wood animals are the generals. They want to be the leaders, want to be in control and want to be around other animals. They are the alpha dogs. They are strong, loud and easy to anger, yet whom you want around in a pinch. Fire animals are exuberant. They are the emperor and are the leaders. They are bouncy and always on the go. They seem to never run out of energy and are always joyful. Earth animals are laid-back and easy going. They need constant companionship and seek to please, desiring praise for their loyalty. They worry too much about pleasing others. Metal animals are the ministers. They hold themselves aloof and are loners. They are frequently sad, yet competent when called upon to perform. Water animals are fearful and timid, often hiding when around strangers. Their bark is usually worse than their bite. Knowing the constitution of the animal can help in determining how to help maintain health and minimize complications as the animals age.

According to TCM theory, there are two major cycles: creation (shen) and control (ko). The creation cycle states “wood feeds fire and is consumed to produce earth which is compacted to make metals giving rise to mountains who capture water which flows to feed the wood.” This relationship means that wood is the mother of fire and fire is the child of wood. Wood nourishes fire that benefits from this relationship while draining the resources of wood. The control cycle states, “wood sends roots to hold the earth which builds up to dam the waters which quench the fires that melt the metals which can be formed into axes to chop down the woods.” As such, wood is the grandparent of earth and restrains if from action.

The eight principles of disease determine whether the disease is superficial (exterior) or deep (internal) which describes the location of the disease. The disease is then characterized as hot or cold which determines the nature of the disease pathogen. Finally, the disease is classified at excess or deficient which explains the relative strength of the pathogen and body’s defense mechanisms as they battle. These are called the six roots: exterior-interior; hot-cold; and excess-deficient. They are further described as toying or Yin. Yang diseases are usually exterior, hot and excessive, while Yin diseases are interior, cold and deficient.

There are six ZangFu organs (two are fire elements, presumably since thee emperor’s functions were important enough to include additional components to carry out the emperor’s duties) and include a husband-wife pair (Fu-Zang). In TCM, the wife always comes first,

hence ZangFu. These are Liver (Yin) – Gall Bladder (Yang); Heart (Yin) – Small Intestines (Yang) plus Pericardium (Yin) – Triple Heater (special Yang organ that regulates exchange of functions between the upper, middle and lower parts of the body); Spleen (Yin) – Stomach (Yang); Lung (Yin) – Large Intestines (Yang); and Kidney (Yin) – Urinary Bladder (Yang). Each of the ZangFu organs has superficial meridians which connect them to the organ functions and are accessible through acupuncture. In fact, recent scientific studies have shown that these connections do exit and are the basis for the somatovisceral reflexes and referred pain. Even though ancient Chinese did not perform anatomical dissections, they did uncover significant understandings of the inner workings of the body./ We have interpreted these findings in translations of their works, but it is astounding how intuitive many of their insights were, particularly when the basic tenets of their understanding of ZangFu physiology were laid down over 2000 years ago.

By describing the pathology of disease in terms of these theories, acupuncture treatment plans can be developed. In the West, we like to use formulas to determine what treatments should be used, following only the application of points which have been previously shown to work in that disease. This, however, limits the use of acupuncture and TCM. It reduces the art of acupuncture to the science of Western thoughts. We do this too much, as it is. On the other hand, it is still important to study and characterize the effects of acupuncture both when it is successful and when it fails, since even ancient wisdom must continue to grow and refine. In general, local conditions can be treated locally. All local points act locally. In addition, constitutional points can help support the patient's struggle to return to health. Certain points have special functions which can be added as needed. Finally, 5- element points can be used to support and strengthen ZangFu organs to help with internal medicine problems.

Acupuncture has come a long way and we are now beginning to put scientific methodology to it, defining its principles in modern terms and clarifying its past. As we scrutinize acupuncture, it is remarkable how much of the old knowledge stands up to the pressure. Personally, I think the pressure is on modern science. Think of the challenge it is for science to explain how a single, small needle appropriately placed can lead to rapid resolution of signs of disease. There is now doubt that this happens, whether explained in terms of Yin or Yang or in terms of endorphin release or neural stimulation.

Acupuncture has reached an all time high for popularity and acceptance, but there is still a long way to go in defining how to integrate acupuncture with modern Western medicine. While they can exist together for the mutual benefit of the patient, some Western drugs can alter the responses to acupuncture and vice versa. We should all be on pins and needles awaiting the day when acupuncture is fully integrated into mainstream medicine.

Management of Dogs with Chronic Atopic Dermatitis: What's New?

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Concept of pruritic threshold and co-factors involved in the pathogenesis of atopic dermatitis (AD)

- According to the theory of threshold a given individual can tolerate a variety of stimuli without developing clinical signs as long as the threshold is not reached. All these stimuli have an additive effect and once the threshold is reached itching and other clinical signs become evident
- Most dogs with AD also have other allergies thus control of concurrent allergies is crucial to decrease the severity of clinical signs.
- Dogs with AD are prone to secondary skin infections that significantly contribute to the level of discomfort. Resolution of skin infections may dramatically decrease the level of pruritus.

Immunotherapy

- For sever cases or for cases with clinical signs all year round, Immunotherapy (IT) is the best long-term therapy.
- IT is effective in 60 to 80 percent of cases and is especially indicated in young animals. Vaccine should be made including the positive results of the allergy test.
- Choice of allergens is based on the history of each case (e.g. seasonality, plants are more prevalent in the patient's environment).
- More than one vaccine may be necessary in cases with numerous allergies.
- Side effects are not common and include increased pruritus after the injection, urticaria, and rarely anaphylaxis.
- Response to IT is not usually evident of the first three to four months and it may take up to a full year before full efficacy is evaluated. If adverse effects are seen, schedule may need to be adjusted. It might be useful to pre-medicate the patient with antihistamines one hour before the injection.
- Once the maintenance dose is achieved the interval of administration is judged based on the individual case.

Pharmacotherapy: New Therapies

Cyclosporine

- Cyclosporine A (CsA) is very effective in severe cases of AD. In one study, CsA was administered PO to the dogs at 10-20 $mgkg^{-1}$ and decrease of pruritus was evident within a few days. After remission was noted, the dose was tapered to the lowest every other day dose.
- In another study, Cs was given orally SID at 5 $mgkg^{-1}$ for six weeks. At the end of tr4ial, pruritus decreased by 78 percent according to owners and skin lesions decreased by 58 percent according to the investigator. No difference in efficacy was noted between prednisolone and CS therapy.

- CsA is available in capsules (25 and 100 mg) or in an oral solution (100mg/ml). CsA is fairly pricey (1 bottle of 50 ml oral solution costs around $300 and 30 capsules of 100 mg cost from $170-$190).
- Soft gel capsules (Sandimmune®) and microemulsion formulations (Neoral®) are not interchangeable with the latter having a higher bioavailability. Studies reported for canine AD utilized the microemulsion formulation.

Misoprostol

- Misoprostol (Cytotec®, Searle, Skolkie, IL, USA) is a PGE1 analog with anti-allergic properties.
- In an open study of 20 dogs with AD, significant improvement was detected when misoprostol was given at 3-6 $mcgkg^{-1}$ TID for 30 days. Pruritus decreased by half in 56 percent of the dogs and skin lesions improved similarly in 61 percent of cases. Improvement was seen after one week of administration. Adverse effects consisted in mild vomiting and diarrhea in some patients.
- In another blinded, placebo-controlled trial 20 dogs with AD were given either three weeks of placebo of misoprostol at 5 $mcgkg^{-1}$ orally TID. Both pruritus and skin lesions significantly improved. Misoprostol may be combined with antihistamines to improve efficacy and decrease the need for glucocorticoids.

Pentoxifylline (PTX)

- PTX, Trental® (Hoechst-Russel Pharmaceutics, Trenton, NJ, USA) is a phosphodiesterase inhibitor that has multiple immunomodulatory properties.
- PTX ($10mgkg^{-1}$ BID) has been used for canine AD in a double-blinded, placebo controlled, clinical trial for four weeks. Clinical signs were evaluated and scored by investigator and owners. During PTX treatment, both pruritus and skin lesions improved significantly but not resolved completely. No adverse effects were reported.