AgriLIFE EXTENSION
Texas A&M System

Veterinary Quarterly REVIEW

Extension Veterinary Medicine
College of Veterinary Medicine
& Biomedical Sciences

Volume 23, Number 4 Winter 2007-2008

Department of Large Animal Clinical Sciences, CVM,
TAMU, College Station, Texas 77843

This is number ninety-one of a continuing series of multi-species quarterly reviews and practice tips for veterinarians in Texas. Information in the Veterinary Quarterly Review is intended to be timely, concise and of practical value. Ideas and input from practicing veterinarians are encouraged Sources of abstracts, articles or practice tips will be credited. Questions/comments may be directed to blawhorn@cvm.tamu.edu.

Bruce Lawhorn
Visiting Professor, Swine Practice

F.C. Faries, Jr.
Extension Program Leader for Veterinary Medicine

Steve Wikse
Professor and Extension Veterinarian — Beef Cattle

BEEF CATTLE PRACTICE

Texas Becomes Cattle Brucellosis-Free

Texas beef and dairy cattle producers, cattle feeders and market operators achieved a long-sought victory on February 1, 2008, when the U.S. Department of Agriculture (USDA) announced that Texas has achieved cattle brucellosis-free status.

For nearly 50 years, Texas cattle producers battled brucellosis, or "Bangs Disease," the bacterial disease that is caused by *Brucella abortus.* The disease can cause cows to abort, deliver weak calves or produce less milk. Cattle brucellosis is a zoonotic disease but the reduction of the disease in cattle and the implementation of sanitary practices and pasteurization procedures reduced the transmission of the disease to humans.

"Texas is the last state to achieve the 'free' status," according to Bob Hillman, Texas' state veterinarian and head of the Texas Animal Health Commission (TAHC). "We have more herds and more cattle than any other state—14 million at last count. We also had more brucellosis infection to fight. In 1959, when Texas officially joined the national eradication program, we had more than 20,000 of the country's 100,000 infected herds. This victory for the cattle industry did not come easily or without hardship. For many cattle producers in the early days of the program, it meant losing a herd with only salvage value payment, or having the herd under quarantine and being unable to sell animals for long periods of time. Some producers' herds became reinfected. In recent years, infected herds

In this issue

were purchased from owners and depopulated whenever possible, to quickly wipe out infection," he said.

Dr. Hillman credited the 2006 Brucellosis Eradication Working Group, made up of 25 cattle industry members, with re-evaluating all aspects of the Texas brucellosis program in preparation for the Summer 2007 USDA review. During that review, cattle were tested statewide.

"Now we must ensure that the disease is not reintroduced, or if it is lingering undetected, we must find the infection and eradicate it quickly. If two infected herds are detected within the next two years, we could lose this hard-earned…status, so we must do everything to protect the health of our herds," said Hillman.

For the next two years, Texas will continue testing cattle at the first point of concentration and change of ownership. This procedure is part of the national brucellosis program standard. The brucellosis slaughter surveillance program also will continue for many years to ensure that the disease is completely eradicated.

"Decades of hard work are now paying off with this incredible accomplishment. I commend the cattle industry of the state and the Texas Animal Health Commission for working together to establish Texas as cattle brucellosis-free," Texas Agriculture Commissioner Todd Staples said.

"Hearing the words 'cattle brucellosis-free' is music to the industry's ears," said Ernie Morales, Chairman of the TAHC. "…there is a tremendous benefit for cattle producers to be able to market their cattle as being from a brucellosis-free state. This… designation will provide cattle producers and trading partners additional assurance that Texas cattle do not pose a disease risk."

Bruce Knight, Under Secretary for USDA's marketing and regulatory programs, said, "We must now focus our efforts on eradicating brucellosis from the free-ranging elk and bison populations in the Greater Yellowstone Area in order to protect our national cattle herd against future outbreaks of this disease."

Texas Animal Health Commission, Box l2966, Austin, Texas 78711, (800) 550 8242, FAX (512) 719 0719. Bob Hillman, DVM, Executive Director. For more information, contact Carla Everett, information officer, at (800) 550 8242, ext. 710, or *ceverett@tahc.state.tx.us.*

New CD-ROM Can Help Cattle Producers Manage Herd Health

Texas AgriLife Extension has developed an interactive CD-ROM to help beef cattle producers manage their herds' health. The program was developed by Wayne Thompson, county Extension agent for agriculture in Harris County. It was based on the herd management practices and educational programs presented by "Buddy" Faries, Extension program leader for veterinary medicine. "This program will help producers select and schedule month-by-month health and production management practices," Faries said. "The calendar helps them to do the right thing at the right time."

The CD includes an interactive calendar that allows the beef producer to generate a management schedule to fit the length of his calving season and the month it begins. "A livestock producer should not manage herd health according to the calendar year or the coincidence of vacations when family members congregate," Thompson said.

The CD also has demonstration videos for each activity and a complete library of publications. Faries said, "The video demonstrations of health and production management practices help the producer do things the right way."

County Extension agents throughout the state are presenting the information in beef management workshops this year, where the CD-ROM will be available for $98. Producers should contact their county Extension offices for information.
The CD can also be ordered from Extension's online bookstore at *http://agrilifebookstore.org.* It is publication number SP-289.

Adapted from a November 8, 2007, news release by Lorri Jones, (281) 855-5620, *lv-jones@tamu.edu,* and Floron (Buddy) C. Faries, Jr., DVM, MS, Extension Program Leader for Veterinary Medicine, Texas AgriLife Extension, The Texas A&M University System, College Station, Texas, 77843. For more information contact Wayne Thompson, (281) 855-5600, *WHThompson@ag.tamu.edu,* or Floron "Buddy" Faries, (979) 845-4353, *FFaries@ag.tamu.edu.*

Update on Hypomagnesemia in Beef Cattle

Hypomagnesemic tetany (also called grass tetany and other names) is a fatal disease of cattle caused by inadequate dietary magnesium. It is characterized by hypomagnesemia, usually accompanied by hypocalcemia. Clinically affected animals have tonic-clonic muscular spasms and convulsions. It is most common in lactating beef cows, where it is known as lactation tetany.

Epidemiology

In the College Station area, we commonly see cases of hypomagnesemia in lactating beef cows grazing spring pastures as early as February. Lactating beef cows require a diet containing 0.22% magnesium (Mg) on a dry matter basis (DMB), while rapidly growing spring grasses commonly contain 0.10 to 0.12% Mg DMB. Wheat pasture is often associated with hypomagnesemia because wheat contains very low concentrations of magnesium, about 0.12% DMB.

Hypomagnesemia can also occur when young spring grass has adequate Mg and calcium (Ca) concentrations, but low sodium (Na) and high potassium (K) concentrations. Sodium deficiency increases the secretion of aldosterone, which causes

an increase in K in saliva and subsequently the rumen. High ruminal K reduces Mg absorption in the digestive tract. The tendency for hypomagnesemia increases markedly when the K/(Ca+Mg) ratio of the forage, expressed on an equivalent basis, exceeds 2.2. The high concentration of nitrogen (N) in rapidly growing grass also interferes with Mg absorption. Thus, the risk of hypomagnesemia is especially high when pastures are heavily fertilized with N and/or K.

Hypomagnesemia must also be on your list of differential diagnoses for sudden deaths of cows on hay diets. I have encountered hypomagnesemia in cows fed bermudagrass or wheat hays. These deaths occurred in January in beef cows fed bermudagrass hay containing only 0.11% Mg DMB.

Many factors influence the occurrence of hypomagnesemia:

- Cows in late gestation or early lactation are most susceptible because their Mg and Ca demands are greatest.
- Thin cows on low-energy diets are more susceptible than cows in good body condition. Energy intake positively affects the absorption of Mg.
- Heavily parasitized cows are more likely to develop hypomagnesemia.
- Old cows are more susceptible than young cows.
- Brahman and Brahman-cross cattle are less susceptible than non-Brahman cattle.
- Winter storms sometimes precipitate an outbreak of hypomagnesemia because during a storm, metabolic demands increase while feed consumption decreases and urinary excretion of Mg may increase. Magnesium is not readily released from the bones of adult cattle. Lactating beef cows must ingest their required 20 grams of Mg every day.
- Outbreaks of hypomagnesemia are more common during winters that have above-average numbers of cloudy days, rainy days and days with temperatures below 40 °F.
- The excitement of being moved to a new location sometimes initiates hypomagnesemia. Epinephrine causes a precipitous fall in serum Mg.

Clinical Signs

Often a cow is found dead with no warning because death can occur within hours in severe cases. Clinical disease can present as an acute form, subacute form or milk fever-like form. Signs progress rapidly in the acute form, which begins with a change in behavior to an alert, belligerent attitude. Cows are nervous, have a wild look in their eyes, and hold their ears erect and heads high. They often charge people. Their muscles tremble, their ears twitch, and they show protrusion of the third eyelid. They vocalize more and urinate more often than normal. Salivation is prominent. They may high-step or walk with a stiffened gait and stagger. Then they fall to the ground, exhibit nystagmus and have seizures. A series of severe tetanic contractions causes legs and feet to thrash and carve curved grooves in the ground. Death occurs within hours once seizures begin.

In the subacute form, cows remain in a state of hyperexcitability, are high-headed and have muscle tremors. If a cow at this stage is stressed, she will often become more aggressive, collapse, develop convulsions and die.

The third form of grass tetany resembles milk fever. Affected cows are found recumbent and depressed. These cases have hypocalcemia as well as hypomagnesemia.

Diagnosis

Diagnosis can be made antemortem based on clinical signs, dietary history and tests to demonstrate a low Mg status. Analyzing the concentrations of minerals in forages consumed by affected cows can aid diagnosis. Hypomagnesemia is likely when the K/(Ca+Mg) ratio (equivalent basis) of the forage exceeds 2.2. Use these equivalent values in your calculations: K:39, Ca:20, Mg:12. The following ratio is calculated for a forage made up of 0.30% Ca, 3.0% K and 0.15% Mg. Make calculations based on 100 g of forage (DMB):

- 100 g forage has 0.3 g Ca (100 g X 0.003), 3.0 g K (100 g X 0.03) and 0.15 g Mg (100 g X 0.0015)
- Change to <u>equivalent basis</u> by dividing the amount of each mineral by its equivalence value.

$$\frac{\frac{3.0\ g}{K\ 39}}{\frac{0.3\ g}{Ca20} + \frac{0.15\ g}{Mg12}} = \frac{0.077}{(0.015 + 0.013)} = \mathbf{2.75}$$

Forages with a K/(Ca + Mg) ratio of 2.75 are definitely high risk for hypomagnesemia. We would like the ratio to be less than 2.2.

Serum or urinary Mg concentrations can be used for diagnosis in clinical cases. The onset of clinical signs of grass tetany correlates most closely to Mg concentrations in cerebrospinal fluid (CSF), but CSF is difficult to collect from a nervous animal. Serum Ca concentrations are generally also subnormal in cases of grass tetany. If an animal is dead, a diagnosis of hypomagnesemia can be made from serum Mg concentrations of a subset of live cows at risk or from Mg concentrations in the vitreous humor, urine or CSF of the dead animal. Concentrations of Mg in the vitreous humor, urine and CSF are considered to be valid 12 to 48 hours after death, depending on ambient temperature. Diffusion of Mg between the intracellular and extracellular compartments of dead animals negates the use of serum Mg after death.

Magnesium concentrations in various tissues of normal and hypomagnesemic animals are listed in the following table:

Sample	Normal value	HypoMg tetany
Serum	1.8 to 3.0 mg/dl	< 1.0 mg/dl
Urine	1.0 to 20.0 mg/dl	< 1.0 mg/dl
CSF	> 2.0 mg/dl	< 1.20 mg/dl
Vitreous humor	1.8 to 2.7 mg/dl	< 1.5 mg/dl

Most textbook discussions state that there are no specific findings at necropsy of cows that have died from hypomagnesemia. However, astute clinicians have found certain gross lesions associated with hypomagnesemia. At least 50 percent of cows that die will leave signs of struggling in the ground due to tetanic convulsions. There is usually a bluish color to the udder, nose and mucous membranes of dead animals. Death is caused by respiratory failure and anoxia results in cyanosis. Striking lesions can often be found in the pleural cavity. The violent death caused by respiratory failure causes alveoli to rupture, which leads to varying degrees of interstitial emphysema of the lungs, accompanied by hemorrhage and edema. These lesions can sometimes be mistaken for acute bovine pulmonary emphysema and edema (ABPEE). In addition to the lung lesions, streaks of hemorrhage are usually present on the pericardium and large areas of diffuse hemorrhage are present in the submucosa of the trachea. The bladder usually is empty. There are often lesions of chronic disease such as liver flukes, stomach worms or internal abscesses that predisposed the victim to hypomagnesemic tetany.

Differential diagnoses include diseases characterized by sudden death or central nervous system signs. These include anaplasmosis, dallisgrass staggers, acute lead poisoning, rabies and tetanus, as well as nitrate or prussic acid toxicity.

Treatment

Treatment must be given promptly with a minimal amount of excitement. Cows affected in the pasture often die from the stress of being caught and restrained. Tranquilization may be needed. Treatment consists of intravenous and subcutaneous administration of solutions containing Mg and Ca, followed by oral supplementation with Mg paste for several days. The key to success is administering enough Mg and Ca. Commercial preparations for treating milk fever do not contain high enough concentrations of Mg for cases of hypomagnesemia. Administering a 20% Mg sulfate solution has a higher success rate. Even with adequate treatment, some cows do not respond favorably, especially thin cows suffering from protein-energy malnutrition.

My treatment of choice is intravenous administration of 500 ml of 20% Mg sulfate solution (supplies 20 g Mg), followed by subcutaneous injection of 500 ml of 25% calcium borogluconate/15% Mg gluconate (a commercial milk fever preparation). Large cows such as Charolais or Simmental breeds often need an extra bottle of the milk fever preparation. Heart rate and rhythm must be monitored during intravenous administration to avoid potential cardiac arrest. I once worked with a practitioner who always treated grass tetany cases with intravenous administration of a 20% Mg chloride solution. Mg chloride is much more available to the body, but it is even more toxic to the heart than Mg sulfate. I've never used Mg chloride, but my colleague certainly got those grass tetany cows to hop up fast! It's very important to give daily oral doses of 60 g Mg oxide in paste form or 10 pounds of alfalfa hay once a day for 5 to 7 days to prevent relapses.

A rectal enema of 200 ml 30% Mg chloride (60 g Mg chloride in 200 ml water) is recommended in some publications as an alternative method of treatment that could be carried out by a producer. This supposedly causes serum Mg concentrations to return to normal within 10 minutes of administration. The enemas may cause some mucosal sloughing. Administering larger volumes of more dilute Mg chloride may prevent that adverse effect. I have not tried enema treatments.

Prevention

There are several ways to prevent hypomagnesemia by supplementing cows with adequate amounts of Mg. It's very important to begin the supplements about 30 days before tetany is generally observed (usually late winter or early spring). The most common preventive measure is to offer cows a commercial salt/trace mineral mixture containing about 10% magnesium oxide. Mineral mixtures designed to prevent grass tetany usually are labeled Hi-Magnesium or Green Pasture minerals. It's very important that the product be the only source of minerals in the pasture. Unfortunately, cows often do not consume enough because magnesium oxide is bitter. Cottonseed meal (CSM) or molasses can be mixed with the mineral to increase palatability. The ratio of CSM to mineral needed to obtain recommended amounts of consumption (usually 4 ounces per head per day) varies from one ranch to another and often must be found by trial and error. Ten pounds of CSM per 100 pounds of mineral often boosts consumption to desired levels.

Feeding legumes, which contain high concentrations of Mg, will also prevent grass tetany. Cattle can be supplemented with legumes by feeding alfalfa hay or by overseeding pastures with clover.

Other methods of prevention include dusting pastures with Mg, injecting round bales with Mg solutions, or spraying a solution of Mg and molasses onto hay in windrows as the hay is baled. It is also important not to over-fertilize pastures with K and N. Fertilization should be based on periodic soil analysis to ensure that the amount of K, N and Mg applied

results in optimum grass production without increasing the danger of grass tetany.

During an outbreak of hypomagnesemia, more intensive efforts, such as feeding alfalfa hay or a high-Mg grain mixture, must be made to ensure an adequate daily intake of Mg. These measures can also help prevent the problem. Feeding 10 pounds of alfalfa hay per cow per day will supply 15 g of Mg and help quell an outbreak of grass tetany. Similar benefit will result from the daily feeding of magnesium oxide (MgO) mixed with grain. The amount of MgO recommended per ton of grain depends on the number of pounds of grain fed daily as listed in the following table:

Lbs. grain/cow/day	Lbs. MgO/ton grain*
2	100**
3	70
4	50
5	40
6	35

* Products with added fat should be avoided because fat will tie up Mg and Ca in the digestive tract and interfere with their absorption.
** More than 100 pounds of MgO per ton of grain is unpalatable.

Bottom Line

Hypomagnesemia of beef cattle can reach epidemic proportions when multiple risk factors work together. In Texas, the disease occurs most often in thin, recently calved cows on a low-Mg diet (unsupplemented grass pasture or grass hay) during prolonged cold, wet and windy weather.

Serum, urine or vitreous humor Mg concentrations are diagnostic. The K/(Ca + Mg) ratio (equivalent basis) can identify pastures or hays that are high risk for hypomagnesemic tetany. This information can be used for diagnostic or preventive purposes. At necropsy, some cases may have lung changes that could be confused with gross lesions of ABPEE.

Administering a 20% Mg sulfate solution intravenously to tetanic cows results in higher recovery rates than using commercially available preparations. The most commonly used control practice is to offer a high-Mg salt/trace mineral mix to the cattle several weeks before calving starts and continue throughout the calving season.

From Steven E. Wikse, DVM, DACVP, Professor and Extension Veterinarian, Food Animal Section, Department of Veterinary Large Animal Clinical Sciences, Texas AgriLife Extension Service, College of Veterinary Medicine and Biomedical Sciences, The Texas A&M University System, College Station, Texas, 77843-4475.

GENERAL PRACTICE

Popular Brochure B-1601, "Reference Guide for Texas Ranchers," Still Available

Effective ranch management requires a tremendous amount of information. At any given time, a ranch manager may be making decisions about the care and management of livestock, control of noxious plants, use of fire to improve forage quality, or reseeding of native pasture. "Reference Guide for Texas Ranchers" consolidates the basic information ranch managers need into an easy-to-read and practical reference. Common Conversions, Livestock Husbandry, Grazing Management, Rangeland Weed and Brush Control, Prescribed Burning, Seeding Rangeland and References are major topics covered in the 34 pages, 8 charts and 18 tables. Examples of specific information included are: guide to use of saline waters for livestock; average composition of common feeds; commonly used grazing systems; sprayer calibration instructions for herbicide application; and seeding rates for grasses.

While some of the information represents "expert opinion" rather than data from quantifiable research, all of it was subjected to professional review. This is a must-have, handy guide for ranch managers. Go to *http://agrilifebookstore.org* to order your copy.

From Allan McGinty, Professor and Extension Range Specialist, Texas AgriLife Extension Service, The Texas A&M University System, San Angelo, Texas 76901.

The Home Edition of "The Merck/Merial Manual for Pet Health" Recently Released

The first edition of "The Merck/Merial Manual for Pet Health," (Home Edition, 1,345 pages; $22.95 US, $27.95 Canada) was recently released (first printing September 2007). It is written in everyday language, and is touted as the easy-to-read, complete health resource for companion animal owners. The species-specific format covers dogs, cats, horses, birds and exotics (amphibians, chinchillas, ferrets, fish, gerbils, guinea pigs, hamsters, mice, prairie dogs, potbellied pigs, rabbits, rats, reptiles and sugar gliders). Special subjects include emergencies, diagnostic testing and imaging, infections, zoonoses, drugs and vaccines, poisonings, pain management, travel with pets, health and the human-animal bond, and cancer and tumors. This book would be a great reference to have in the reception areas of veterinary clinics. Go to *www.merckbooks.com* to order.

From Merck & Co., Inc, Whitehouse Station, New Jersey, which publishes "The Merck/Merial Manual for Pet Health" on a not-for-profit basis in an educational partenership with Merial Limited, a Merck and Sanofi-Aventis Company.

New Publication Highlights Proper Use of Sulfonamides

When administering a drug to a show animal, the owner must be certain there will be no detectable level of the drug in the animal's urine when it is tested at a show. One class of drugs, the sulfonamides, are of special concern because even though they may be administered correctly and

the proper withdrawal times observed, residues could remain in the urine. This can happen if the owner fails to give the animal adequate amounts of water, without which there may be crystallization in the kidneys. It can also happen if the animal continues to be exposed to the drug through the environment, even after the drug is no longer being administered. For example, the soil may contain sulfonamide excreted in urine and manure, or medicated drinking water may spill and accidentally contaminate the soil. If an animal ingests contaminated soil while grazing or feeding, its exposure to the drug continues.

A new publication, L-5495, "Proper Use of Sulfonamides in Market Show Animals," is available from Texas AgriLife Extension at *http://agrilifebookstore.org.*

EQUINE PRACTICE

Horse Gastric Ulcer Syndrome Can Be Controlled with Diet

Research from Texas A&M University showed that feeding alfalfa to horses that have the potential to be high performers either prevented or was therapeutic in treating stomach ulcers.

"Something in alfalfa hay tends to buffer acid production," said Dr. Pete Gibbs, Extension horse specialist. "Thirty percent of the 1 million horses in Texas are used in racing, showing and competitive performance. Up to 90 percent of racehorses and more than 50 percent of arena performance horses have gastric ulcers of varying severity. When they have ulcers, horses don't eat as well, work as well, or feel as good," Gibbs said. It is thought that ulcers are caused by feeding grain, confinement, exercise and overall environmental stress. Studies have shown that horses will heal if given less acidic diets.

The recent research project in the department of animal science's equine science program was part of Travis Lybbert's master's degree thesis in collaboration with the College of Veterinary Medicine. Dr. Gibbs served on Lybbert's academic research committee.

In the research, 24 quarter horses from 12 to16 months old were separated into two treatment groups. One group was fed bermudagrass hay and the other was fed alfalfa hay to meet the daily roughage needs. The yearlings had forced exercise during the study. The horses were examined internally with an endoscope at the beginning and end of two 28-day trials. In this study, ulcer scores increased when alfalfa was removed from the horses' diets and they were turned out on pasture.

It is commonly thought that horses turned out on pastures are better off than those that are confined. However, if grass hay is the only hay they are fed, horses can still get gastric ulcers, said Gibbs.

Horse owners—especially those with performance horses—have two options, Gibbs said. They can give their horses a pharmaceutical product that will decrease acid production, or they can manage their horses' diets. The second option does not stop acid production but buffers the acid, Gibbs said. Further study of horses with varying degrees of ulceration is needed. This would help determine the extent to which alfalfa or alfalfa-based products might help, and possibly reveal the properties of alfalfa that lessen the occurrence and severity of ulcers.

"Based on what we know right now—for horses that are kept in con-

Veterinary Continuing Education Seminars, College of Veterinary Medicine and Biomedical Sciences Texas A&M University, 2008

*February 28-March 1, 2008	Texas Veterinary Medical Association Winter Conference
*April 25-27, 2008	Annual Feline Conference (Dr. John August)
*June 6-8, 2008	17th Annual Food Animal Conference (Dr. Lewis Dinges)
*July 18-20, 2008	Practical Veterinary Dentistry (Dr. Bert Dodd)
*August 1-3, 2008	2nd Annual Dermatology Conference (Dr. Christine Rees)
*October 4-5, 2008	Canine Conference (Dr. Mike Willard)
*October 24-26, 2008	Annual Equine Conference (Dr. Jeff Watkins)
*November 21-23, 2008	Clinical Neurology Conference (Dr. Jonathan Levine)
*December 5-6, 2008	Annual Exotic Pets Conference (Dr. Sharman Hoppes)
*December 12-14, 2008	Annual Equine Reproduction Symposium (Dr. Dickson Varner)

*Confirmed

Calendar is subject to revision.

For more information on these programs of self-study and personalized continuing education opportunities, please call (979) 845-9102; fax (979) 862-2832; or e-mail *ceoffice@cvm.tamu.edu.* Visit our Web site at *http://www.cvm.tamu.edu/vtce.*

From the Office of Veterinary Continuing Education, Texas Veterinary Medical Center, College Station, Texas.

finement, eating feed and getting forced exercise—it makes sense to consider some alfalfa as part of their diet," he said. Until further research is done, he recommends that horses weighing 1,000 to 1,300 pounds be fed about 1 pound of alfalfa after a grain meal.

Adapted from a December 13, 2007, article, "Horse Gastric Ulcer Syndrome Can Be Controlled with Diet," by Edith Chenault, (979) 845-2886, *echenaul@ag.tamu.edu.* For more information contact Dr. Pete Gibbs, (979) 845-3579, *PGibbs@ag.tamu.edu.*

CANINE PRACTICE

New Genetic Test Available for Centronuclear Myopathy of Labrador Retrievers

Hereditary myopathy, type II muscle atrophy, and Labrador muscular myopathy are all names used to describe the same hereditary muscular disorder seen in Labradors since the 1970s. This disease results in rapid exercise intolerance because of a deficiency in the number of fast twitch or type II muscle fibers.

Clinical signs usually appear by the age of 10 to 16 weeks, although some females may not show signs at up to 5 months of age. Signs are a rapid onset of exercise intolerance, generalized weakness, a choppy or stilted gait, difficulty in holding the head upright, and collapse. All signs will abate with rest, but when exercise is resumed, affected dogs get progressively weaker. Veterinary neurology texts give a comprehensive review of clinical signs.

This disorder is inherited as an autosomal recessive trait, so in the past, the only way to detect carriers was to look back through the family tree/pedigree of an affected dog. This did not allow carriers to be identified before they were used for breeding.

Now there is a simple DNA test that will detect carriers before they reach reproductive age. The test is performed on a cheek swab, so sampling is easy. The test is very accurate, specific and sensitive, with very few false positives or negatives. The test is performed at Alfort School of Veterinary Medicine in Paris, France. The project communications coordinator is Marilyn Fender. The cost of testing a single dog is about $80, which includes the cost of the test, brush kits and shipping. There are different rates for litters.

This test is a major breakthrough in the control of inherited myopathy because carriers can now be eliminated from the breeding pool. Go to *www.labradorcnm.com* for information, submission forms and the certification of results.

From Dr. Mark D. Neer, Diplomate, ACVIM, Veterinary Teaching Hospital Director, Oklahoma State University (OSU) College of Veterinary Medicine, as reported in *OSU Animal HealthUpdate,* Spring 2007.

WILDLIFE AND EXOTIC PRACTICE

Publication E-98, "Integrating Deer, Quail and Turkey Habitat," Available

Managing for deer, quail and turkeys in South Texas requires careful planning. To be successful, the minimum needs of each species must be met while minimizing the grazing competition between deer and cattle. This publication covers the habitat needs of white-tailed deer, bobwhite quail and Rio Grande turkey (6 pages, 2 tables, 6 photos). Order online at *http://agrilifebookstore.org.*

From Robert K. Lyons, Professor and Extension Range Specialist, The Texas A&M University System.

SWINE PRACTICE

Practice-Building Opportunities with Surgical Procedures in Show Swine

These surgical procedures are commonly performed in a busy show swine practice:

1. Castration
2. Cryptorchid castration
3. Castration wound abscess removal
4. Vasectomy
5. Removal of preputial diverticulum (urine pocket removal)
6. Removal of persistent penile frenulum
7. Diagnostic skin biopsy
8. Tumor/lump removal
9. Umbilical hernia repair
10. Umbilical stalk abscess removal
11. Inguinal hernia repair
12. Prolapsed rectum replacement (purse-string suture)
13. Prolapsed rectum resection
14. Prolapsed vagina/cervix replacement
15. Claw amputation
16. Removal of inflamed bursa of sternum
17. C-section

Practitioners interested in expanding their show swine practice should develop their skills in performing these procedures.

From Bruce Lawhorn, DVM, MS, Visiting Professor, Swine Practice, Food Animal Section, Department of Large Animal Clinical Sciences, College of Veterinary Medicine and Biomedical Sciences, The Texas A&M University System, College Station, Texas 77843-4475.

SHEEP AND GOAT PRACTICE

Donkeys Becoming Popular to Protect Herds From Predators

More than one-third of U.S. lamb and sheep losses in 2004 were caused by predators. In four of five U.S. regions, coyotes and dogs accounted for 60 to 73 percent and 6 to 12 percent of lamb losses, respectively. In the fifth region, coyotes and dogs were responsible for 38 and 30 percent of lamb losses, respectively. Coyotes and dogs accounted for 52 and

23 percent of sheep losses, respectively, in all areas in 2004. It is obvious that livestock predation by coyotes and dogs needs to be reduced.

In addition to guard dogs and other methods of protection, donkeys are increasingly being used to protect livestock herds. About 9 to 16 percent of Texas' sheep and goat producers and 99 percent of Colorado sheep producers use guard donkeys.

Donkeys, mules, horses and zebras generally are intolerant of coyotes, wolves and wild dogs. Donkeys may be the most intolerant; they will run after and attack coyotes and dogs, which often deters them from further predation. Jennies are thought to make better guards than jacks because of their maternal, protective characteristics. Donkeys bond better with livestock if they are raised from a young age with the livestock they will protect. One to three guard donkeys per herd may be best, because if there are too many donkeys they will tend to form their own herd and not protect livestock as well. Exposing a prospective guard donkey to a dog will reveal whether it is aggressive toward canines; if it is not aggressive, it cannot be expected to protect a herd.

Several publications on controlling coyotes are available from the Texas AgriLife Extension Service. These are: B-1664, "Coping with Coyotes;" L-1917, "Controlling Coyotes with Snares;" L-1908, "Trapping Coyotes;" and L-5473, "Managing Suburban Coyotes." They can be ordered at *http://agrilifebookstore.org*.

Adapted from "Sheep and Lamb Predator Loss in the United States, 2004," at *http://nahms.aphis.usda.gov/sheep/sheep_pred_deathloss_2004_pdf*; "Ranchers Use Donkeys to Protect Herds from Predators," *Sheep Industry News,* Volume 11, Issue 12, page 6, December 2007; and Jon Gersbach, Milam County Extension Agent, Texas AgriLife Extension Service, The Texas A&M University System.