September 2018

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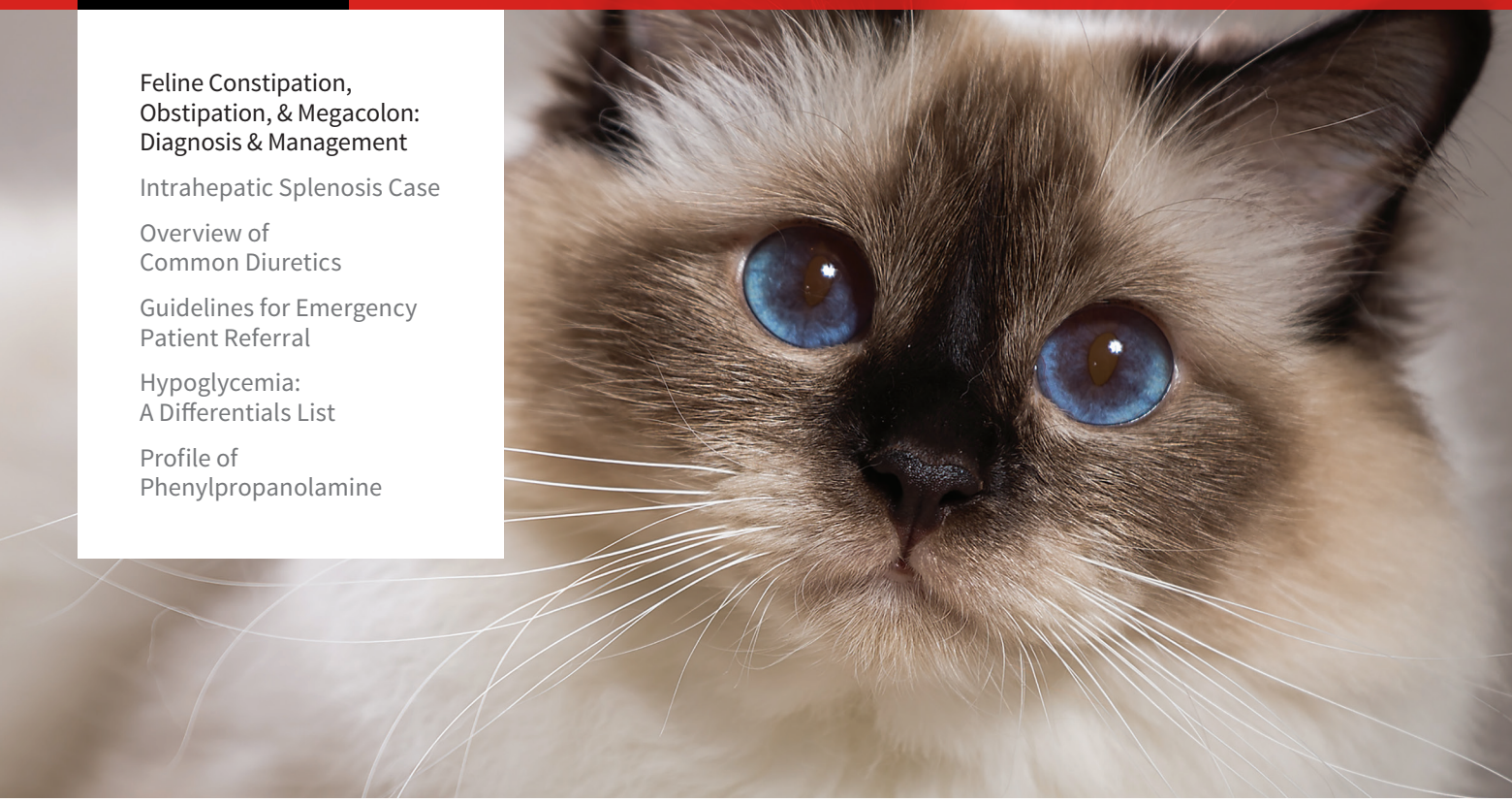
EOSINOPHILIA CAUSES IN CATS

IN THIS ISSUE



Volume 16 Number 9

WSAVA Global Veterinary Community

clinician's brief
THE OFFICIAL CLINICAL PRACTICE JOURNAL OF THE WSAVA

CANADIAN VETERINARY MEDICAL ASSOCIATION
L'ASSOCIATION CANADIENNE DES MÉDECINS VÉTÉRINAIRES

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Topical Solution

profender®

(emodepside/praziquantel)

CAUTION:
Federal law (U.S.A.) restricts this drug to use by or on the order of a licensed veterinarian.

Topical Solution for the treatment and control of hookworm, roundworm and tapeworm infections in cats and kittens that are at least 8 weeks of age and weigh at least 2.2 lbs (1 kg).

DESCRIPTION:
PROFENDER [1.98% emodepside/7.94% praziquantel] Topical Solution is a clear yellow ready-to-use solution packaged in single unit dosing applicator tubes for topical (dermal) treatment of cats 8 weeks of age and older and weighing at least 2.2 lbs (1 kg). The formulation and dosage schedule is designed to provide a minimum of 1.36 mg/lb (3 mg/kg) emodepside and 5.45 mg/lb (12 mg/kg) praziquantel based on body weight.

Emodepside, a semi-synthetic molecule, is a cyclic depsipeptide. The chemical name is Cyclo [D-2-hydroxypropanoyl-N-methyl-L-leucyl-3-[4-(4-morpholinyl)phenyl]-D-2-hydroxypropanoyl-N-methyl-L-leucyl-D-2-hydroxypropanoyl-N-methyl-L-leucyl-3-[4-(4-morpholinyl)phenyl]-D-2-hydroxypropanoyl-N-methyl-L-leucyl].

Praziquantel is an isoquinoline cestocide. The chemical name is 2-Cyclohexylcarbonyl-1,2,3,6,7,11b-hexahydro-4H-pyrazine-2,1-a-isoquinoline-4-one.

INDICATIONS:
PROFENDER Topical Solution is indicated for the treatment and control of hookworm infections caused by *Ancylostoma tubaeforme* (adults, immature adults, and fourth stage larvae), roundworm infections caused by *Toxocara cati* (adults and fourth stage larvae), and tapeworm infections caused by *Dipylidium caninum* (adults) and *Taenia taeniaeformis* (adults) in cats.

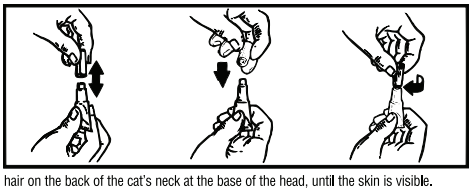
DOSAGE AND ADMINISTRATION:
The recommended minimum dose is 1.36 mg/lb (3 mg/kg) emodepside + 5.45 mg/lb (12 mg/kg) praziquantel as a single topical dose. A single treatment is effective and a second treatment should not be necessary. If re-infection occurs, the product can be re-applied after 30 days.

1. Select the package that correctly corresponds with the body weight of the cat. (See Table below.)

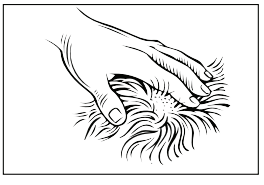
Cat Weight*	Profender Topical Solution	Volume (mL)	Emodepside (mg)	Praziquantel (mg)
2.2-5.5 lbs.	Small	0.35	7.5	30.0
>5.5-11 lbs.	Medium	0.70	15.0	60.1
>11-17.6 lbs.	Large	1.12	24.0	96.1

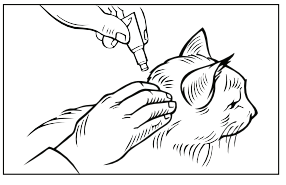
* Cats over 17.6 lbs should be treated with the appropriate combination of tubes.

2. Remove one unit dose tube from the package.
3. While holding the tube in an upright position, remove the cap from the tube.
4. Turn the cap over and place the other end of cap onto the tip of the tube.
5. Twist the cap to break the seal and then remove cap from the tube.



6. Part the hair on the back of the cat's neck at the base of the head, until the skin is visible.





7. To ensure the entire contents of the tube are administered, place the tip of the tube on the skin and squeeze the entire contents directly onto the skin. Lift tube away from the skin before releasing pressure on the tube.

Do not apply to broken skin or if hair coat is wet. Do not get this product in the cat's mouth or eyes or allow the cat to lick the application site for one hour. Oral exposure can cause salivation and vomiting. Treatment at the base of the head will minimize the opportunity for ingestion while grooming. In households with multiple pets, keep animals separated to prevent licking of the application site.

Stiff hair, a damp appearance of the hair, or a slight powdery residue may be observed at the treatment site. These effects are temporary and do not affect the safety or effectiveness of the product.

HUMAN WARNINGS:
Not for human use. Keep out of reach of children.

To prevent accidental ingestion of the product, children should not come in contact with the application site for twenty-four (24) hours while the product is being absorbed. Pregnant women, or women who may become pregnant, should avoid direct contact with, or wear disposable gloves when applying, this product. Studies performed in rats and rabbits suggest that emodepside may interfere with fetal development in those species.

PROFENDER Topical Solution may be irritating to skin and eyes. Reactions such as facial, tongue and hand swelling have been reported in humans in rare instances. Avoid contact with the application area while it is wet and wash hands thoroughly with soap and warm water after handling. People with known hypersensitivity to butylhydroxyanisole, emodepside or praziquantel should administer the product with caution. If the product accidentally gets into eyes, flush thoroughly with water. May be harmful if swallowed. In case of accidental ingestion or if skin or eye irritation occurs, call a poison control center or physician for treatment advice.

The Material Safety Data Sheet (MSDS) provides additional occupational safety information. For customer service or to obtain product information, including the MSDS, call 1-800-633-3796. For medical emergencies or to report an adverse reaction, call 1-800-422-9874.

PRECAUTIONS:
Safe use of this product has not been evaluated in cats less than 8 weeks of age or weighing less than 2.2 lbs (1 kg), in cats used for breeding, during pregnancy or in lactating queens. The effectiveness of this product when used before bathing has not been evaluated.

Use with caution in sick or debilitated cats. Oral ingestion or exposure should be avoided. Use with caution in heartworm positive cats. The cats enrolled in the field study were heartworm antigen and antibody negative prior to entering the study. In a laboratory study, cats artificially infected with adult heartworms and treated with PROFENDER Topical Solution had fewer worms recovered than the placebo control group. (**See ANIMAL SAFETY.**)

ADVERSE REACTIONS:
Field study: In a controlled, double-masked field safety study, owners administered PROFENDER Topical Solution to 606 cats. Adverse reactions reported by the cat owners included licking/excessive grooming in 18 cats (3.0%), scratching treatment site in 15 cats (2.5%), salivation in 10 cats (1.7%), lethargy in 10 cats (1.7%), alopecia in 8 cats (1.3%), agitation/nervousness in 7 cats (1.2%), vomiting in 6 cats (1.0%), diarrhea in 3 cats (0.5%), eye irritation in 3 cats (0.5%), respiratory irritation in 1 cat (0.2%) and shaking/tremors in 1 cat (0.2%). All adverse reactions were self-limiting.

Laboratory effectiveness studies: One cat died 10 days after receiving PROFENDER Topical Solution. The necropsy showed chronic active cholangiohepatitis. While the use of the drug did not appear to be the direct cause of death, treatment with the drug cannot be ruled out as a contributing factor (**See PRECAUTIONS**). One cat treated with a vehicle placebo (formulation minus the active ingredients) showed salivation, gagging, lethargy and a swollen tongue.

Foreign Market Experience: The following adverse events were reported voluntarily during post-approval use of the product in foreign markets: application site reaction (hair loss, dermatitis, pyoderma, edema, and erythema), salivation, pruritus, lethargy, vomiting, diarrhea, dehydration, ataxia, loss of appetite, facial swelling, rear leg paresis, seizures, hyperesthesia, twitching, and death.

Post-Approval Experience: The following adverse events are based on post-approval adverse drug experience reporting. Not all adverse events are reported to FDA CVM. It is not always possible to reliably estimate the adverse event frequency or establish a casual relationship to product exposure using this data. The following adverse events are listed in decreasing order of reporting frequency in cats: Application site reaction (hair loss, dermatitis, pyoderma, edema, and erythema), hypersalivation, lethargy/depression, vomiting, ataxia, anorexia, trembling/twitching, diarrhea, mydriasis, fever, hyperactivity/nervousness. In some cases, death has been reported as an outcome of the adverse events listed. For a complete listing of adverse reactions for Profender Topical Solution reported to the CVM see: http://www.fda.gov/ADEreports.

The listing includes Adverse Events reported to CVM for products , such as Profender, that contain the combined active ingredients emodepside and praziquantel. Listings by active ingredient may represent more than one brand name.

To report suspected adverse events and/or to obtain a copy of the MSDS or for technical assistance, call Bayer Animal Health at 1-800-633-3796.

For additional information about adverse drug experience reporting for animal drugs, contact FDA at 1-888-FDA-VETS or online at http://www.fda.gov/AnimalVeterinary/SafetyHealth.

EFFECTIVENESS:
In a total of 13 controlled laboratory studies to establish effectiveness, 149 cats were treated with PROFENDER Topical Solution. In the field study conducted at 13 veterinary clinics/hospitals, 837 purebred or crossbred cats from single and multi-cat households were enrolled to evaluate safety and effectiveness under field conditions of use. Of those, 606 received a single treatment with PROFENDER Topical Solution. Cats ranged in age between 2 months and 17 years and weighed between 0.8 lbs (0.36 kg) and 21 lbs (9.62 kg). Data from these studies demonstrated PROFENDER Topical Solution is safe and effective for the treatment and control of hookworm infections caused by *Ancylostoma tubaeforme* (adults, immature adults, and fourth stage larvae), roundworm infections caused by *Toxocara cati* (adults and fourth stage larvae), and tapeworm infections caused by *Dipylidium caninum* (adults) and *Taenia taeniaeformis* (adults).

ANIMAL SAFETY:
In a field study, PROFENDER Topical Solution was used in cats receiving other frequently used products including: analgesics, anti-fungals, non-steroidal anti-inflammatories, anthelmintics, antimicrobials, flea and tick products, sedatives, anesthetics, cardiac medications, anxiolytics, hormonal treatments, steroids, otic and ophthalmic preparations, and vaccines.

Dose Tolerance Study in Cats: PROFENDER Topical Solution was applied topically one time to young cats at 10X the recommended label use rate. Two cats salivated. Another cat exhibited tremors and lethargy. These signs were self-limiting.

Oral Safety Studies in Cats: PROFENDER Topical Solution was administered orally at the recommended topical dose to young adult cats. The cats exhibited salivation, vomiting, tremors, abnormal gait, abnormal respiration and weight loss. These signs were self-limiting.

General Safety Study in Kittens: PROFENDER Topical Solution was topically applied at 0X (vehicle control), 1X, 3X and 5X the maximum dose to 48 healthy 8-week-old kittens every two weeks for six doses. One 5X kitten experienced salivation and tremors and another 5X kitten experienced salivation on the day of dosing. A third 5X kitten experienced tremors the day after dosing. Three cats vomited within 24 hours of dosing, one each in vehicle control, 3X and 5X groups.

Safety Study in Heartworm Positive Cats: Cats artificially infected with adult heartworms harvested from dogs were treated topically with PROFENDER Topical Solution at 0X, 1X or 5X the recommended dose once a month for three treatments. Clinical signs included salivation (one 1X and three 5X cats), labored breathing (all groups) and lethargy (one 5X cat). At the study conclusion, the 1X and 5X cats had fewer live heartworms recovered than the 0X group.

STORAGE INFORMATION:
Store at or below 77°F (25°C).
Protect from freezing.

HOW SUPPLIED:

Code Number	Applications per Package
82482521	20 - 0.35 mL tubes (5 blisters of 4 tubes)
03615026	40 - 0.35 mL tubes (10 blisters of 4 tubes)
82482572	20 - 0.7 mL tubes (5 blisters of 4 tubes)
03615034	40 - 0.7 mL tubes (10 blisters of 4 tubes)
82482580	20 - 1.12 mL tubes (5 blisters of 4 tubes)
82482602	40 - 1.12 mL tubes (10 blisters of 4 tubes)
81175276	8 - 0.35 mL tubes (2 blisters of 4 tubes) 20 - 0.7 mL tubes (5 blisters of 4 tubes) 8 - 1.12 mL tubes (2 blisters of 4 tubes)

Profender is protected by the following U.S. Patents: 5 514 773 and other patents pending.

Made in Germany

NADA 141-275, Approved by FDA

82482521/03615026/82482572/03615034/82482580/82482602, R.2 March, 2015
19675

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U.S. Patent Nos. 6,797,289, 8,753,697 and 9,572,791. For use under U.S. Patent Nos. 8,568,803 and 8,808,770. Additional Patent Pending.

*Source: Among veterinary brands. Survey conducted among small animal veterinarians who recommended oral joint health supplements.

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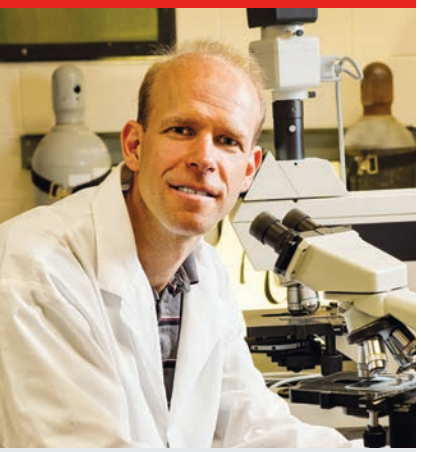

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QUARTERLY UPDATE
FALL 2018

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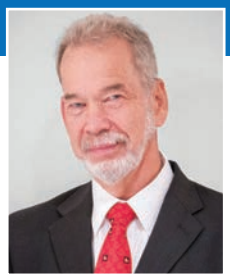
EST. 1974

LEADING THE FIGHT AGAINST HEARTWORM DISEASE

HEARTWORM GUIDELINES



TOM NELSON, DVM
ANIMAL MEDICAL CENTER
ANNISTON, ALABAMA
CHAIRMAN, AHS
RESEARCH AND
GUIDELINES COMMITTEE

The AHS Guidelines: 2018 Updates

Guidance on Prevention, Testing and Treatment

While heartworms are a tough opponent, knowledge is a powerful weapon. To arm veterinarians with the most up-to-date and comprehensive information about heartworm management, the American Heartworm Society (AHS) created guidelines on heartworm **prevention, testing** and **treatment,** as well as heartworm epidemiology and biology. Guideline revisions are published as needed, based on sound principles of heartworm management. The 2018 guidelines include the following recommendations.

HEARTWORM PREVENTION: Weigh the Risk

Heartworms have been diagnosed in all 50 states, but the risk of transmission varies considerably. Veterinarians should weigh the relative risk of heartworm infection when making specific heartworm prevention recommendations to clients. We suggest that veterinarians:

- Recommend year-round prevention with a macrocyclic lactone preventive for all pets.
- Take practical steps to limit mosquito exposure (e.g., keeping pets indoors during peak mosquito times and eliminating standing water where mosquitoes can breed).
- If mosquito proliferation and the presence of heartworm-positive animals means the risk of heartworm transmission is high, consider adding a mosquito repellent/ectoparasticide seasonally or year-round. Don't rely on repellents alone to prevent heartworm disease.

HEARTWORM TESTING: Know When to Heat the Sample

The practice of heating serum samples prior to antigen testing to unmask blocked antigen has been the subject of multiple scientific studies designed to determine optimal heartworm testing methods. For veterinarians who have questions about heat treatment, the AHS recommends:

- Rely on antigen and microfilaria testing for routine heartworm screening. These tests are highly sensitive and accurate.
- Because heat treatment is contrary to labeling for in-house antigen tests and may interfere with the accuracy of certain blood tests, don't heat-treat samples as part of routine screening.
- Consider heat treating a patient's serum sample if clinical infection in the absence of a positive antigen test result is suspected.

HEARTWORM TREATMENT: Avoid Alternatives

The goal of heartworm treatment is to kill adult worm infections with minimal complications while stopping the progression of disease. Alternative, non-arsenical-based treatment protocols have been studied to guide management of dogs that aren't candidates for melarsomine treatment; however, the treatment protocol recommended by the AHS includes pre-treatment with an ML and doxycycline, following by a month-long waiting period, then 3 doses of melarsomine on days 60, 90 and 91. Here's why:

- Waiting until day 60 to start killing the adult worms with melarsomine allows time for *Wolbachia* surface proteins and other metabolites to dissipate, as well as time for the heartworms themselves to wither.
- While non-arsenical treatment protocols have their place for a small percentage of dogs, it can take much longer to kill adult worms, during which time heartworm pathology and damage can progress. ■

To access the complete set of AHS canine and feline heartworm guidelines, visit **heartwormsociety.org**

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"A 1-year-old Pomeranian with literally every single deciduous tooth left. In between the rows was a moat of hair, calculus, and anything else you can think of." —*Alexis L*

"We recently had a dog with 9."—*Jennifer B*

"13"—*Nadja R*

"16"—*Madeleine K*

Insulinomas are relatively _____ aggressive and have a _____ prognosis in ferrets as compared with dogs.

On Twitter, you answered:

50% Less, better

50% More, worse

Correct Answer: Insulinomas are relatively *less* aggressive and have a *better* prognosis in ferrets as compared with dogs.

Who can relate?

"This could be 5 minutes apart in the same day."—*Chris T*

"Some days I think I should have just stayed in bed."—*Meryem I*

"Every day is the [other] day for me."—*Xioma S*

"It is a stethoscope kind of day!"—*Lauren B*

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[1]IDEXX Laboratories February 2018.

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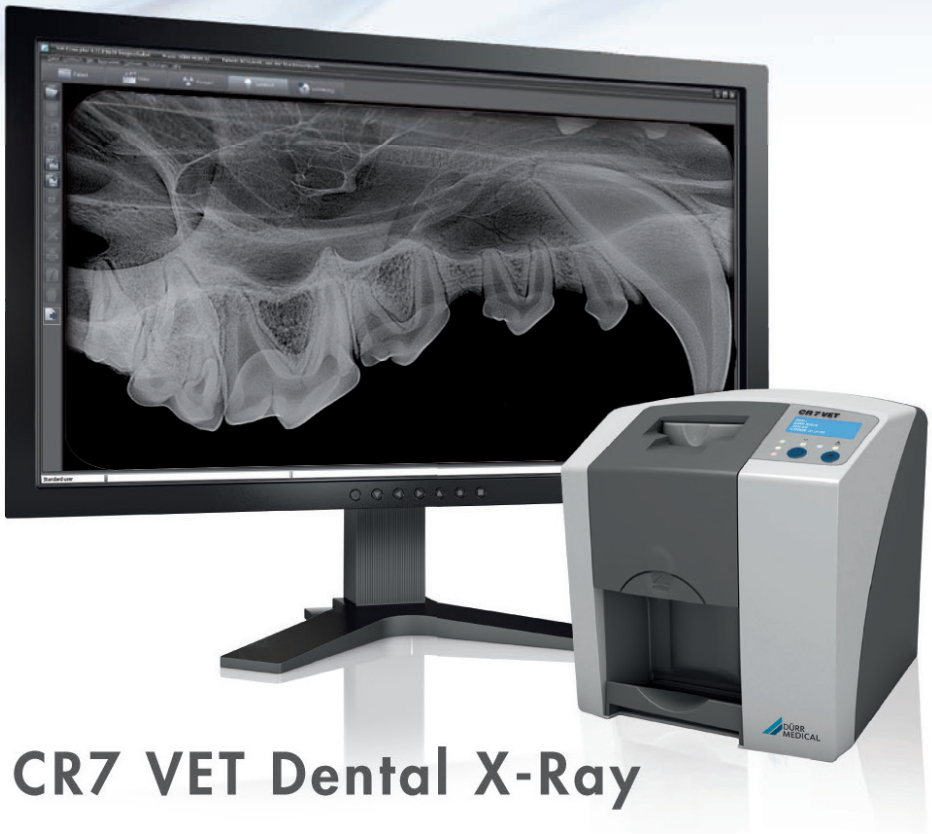

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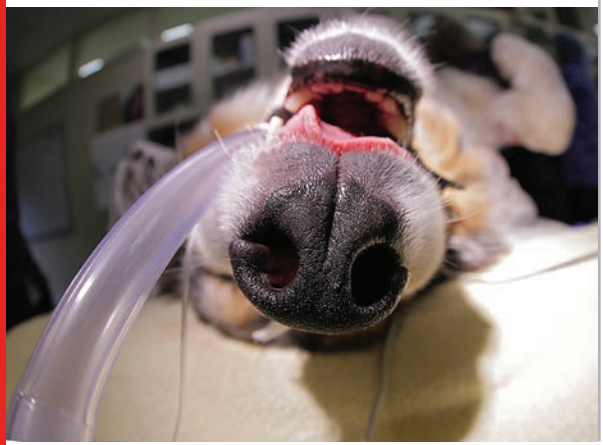
TOP 5

Top 5 Causes of Eosinophilia in Cats

Glenn A. Olah, DVM, PhD, DABVP (Feline)

PG 54

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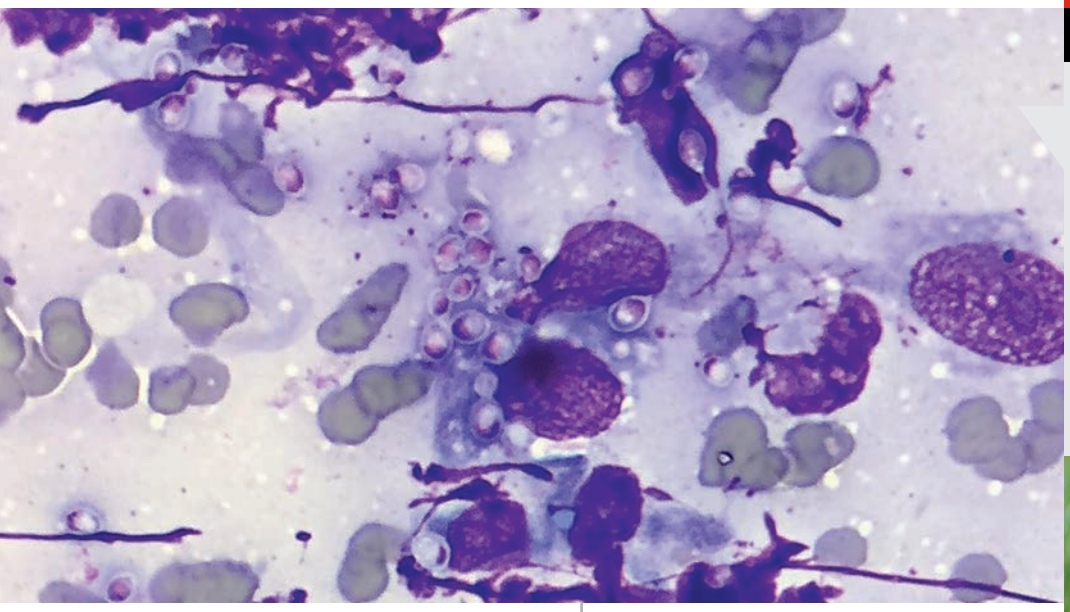

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Kyle Webb, DVM, DACVP
brief.vet/transtracheal-wash

QUIZ
Disk Disease in Dogs & Cats
Katherine I. Crook, DVM, DACVIM (Neurology)
brief.vet/quiz-disk-disease

VIDEO
Surgery Surprise
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Using QR codes from your mobile device is easy and quick!

Simply focus your phone's camera on the QR code as if taking a picture (but don't click!). A notification banner will pop up at the top of your screen; tap the banner to view the linked content.

QUIZ:

Hypertrophic Cardiomyopathy in Cats

Amara Estrada, DVM, DACVIM (Cardiology)
University of Florida

Matthew Boothe, DVM
Blue Pearl Veterinary Partners

Hypertrophic cardiomyopathy is the most common form of underlying heart disease affecting cats.

Which of the following is not a common cause of heart murmur in cats?
A. Hyperthyroidism
B. Heartworm disease
C. Physiologic causes (ie, flow murmur)
D. Underlying primary cardiac disease
E. Hypertension

CORRECT ANSWER: B

In dogs, a systolic heart murmur may be present with heartworm disease (ie, *Dirofilaria immitis*) when pulmonary hypertension is present or when worms are entangled around the tricuspid valve. The murmur is typically caused by tricuspid regurgitation. In cats, however, the lungs are the primary organ affected, as the relatively low worm burden makes development of pulmonary hypertension and a resultant murmur less likely in cats than in dogs.[1-4] Thus, a systolic heart murmur is a rare clinical examination finding.[5,6]

Hyperthyroidism, a significant cause of functional murmurs in cats, produces changes in cardiac contractility, myocardial oxygen consumption, cardiac output, blood pressure, and systemic vascular resistance.[7,8] These changes can lead to turbulent flow in the outflow tracts secondary to increased cardiac output, which can manifest as a murmur during auscultation. Hyperthyroidism can also lead to significant cardiac hypertrophy, primarily through the increased workload on the heart via an increase in the hemodynamic load.[9] Because these changes can mimic hypertrophic cardiomyopathy, it is important to check thyroid levels in cats showing echocardiographic evidence of hypertrophic cardiomyopathy (eg, left ventricular hypertrophy, eccentric hypertrophy of the left ventricle).

References can be found online.

TO ACCESS THE FULL ARTICLE & TAKE THE REST OF THIS QUIZ, VISIT cliniciansbrief.com/article/quiz-hypertrophic-cardiomyopathy-cats

Veterinary Team Brief

Career Center Launched

Veterinary Team Brief (**veterinaryteambrief.com**) has debuted its **Career Center** (**veterinaryteambrief.com/career-center**), which offers help to veterinary team members and hiring managers. *Veterinary Team Brief*'s Career Center allows veterinary professionals to:

- Search and apply for positions at practices that value credentials
- Upload anonymous résumés so potential employers can make contact, while allowing job seekers to choose where and to whom their information is released
- Receive alerts for new job openings based on personal profile, skills, interests, and preferred location(s)
- Post a job listing and find qualified candidates
- Explore career resources, including job search tips and tools

Looking for a new position? Need to hire?
Scan the QR code to see how the new *Veterinary Team Brief* Career Center can help.

Using QR codes from your mobile device is easy and quick!

Simply focus your phone's camera on the QR code as if taking a picture (but don't click!). A notification banner will pop up at the top of your screen; tap the banner to view the linked content.

OUR AUTHORS

KELLI ALMES, DVM, DACVP, is an assistant professor and the director of client services at Kansas State University, where she also earned her DVM and completed an anatomic pathology residency.

CASE IN POINT PAGE 29

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CONSULT THE EXPERTS PAGE 63

JULIE KATHLEEN BYRON, DVM, MS, DACVIM, is a professor of small animal internal medicine at The Ohio State University, where she earned her DVM and master's degree and completed a residency. Dr. Byron completed a rotating small animal internship at VCA West Los Angeles Animal Hospital. Her clinical and research interests include urinary incontinence, functional urethral obstruction, and cystoscopic interventional procedures.

THERAPEUTICS SNAPSHOT PAGE 34

AKATERINA DAVROS, DVM, is an emergency medicine intern at Fort Collins Veterinary Emergency and Rehabilitation Hospital in Fort Collins, Colorado. She earned her DVM from Kansas State University and will pursue a residency in emergency medicine and critical care. Dr. Davros is a recipient of the Merck Animal Health Veterinary Student Innovation Award for her work in improving mental health of students and staff at Kansas State University. Her clinical interests include polytrauma, multimodal pain management, and rehabilitation in the emergency setting.

CASE IN POINT PAGE 29

CHRISTINE EGGER, DVM, MVSc, CVA, CVH, DACVAA, is a professor at University of Tennessee. She earned her DVM and master's degree from University of Saskatchewan in Saskatoon, Canada. Dr. Egger completed a residency in veterinary anesthesia and is the president of the American College of Veterinary Anesthesia and Analgesia. She is certified in veterinary acupuncture and herbal medicine. Her interests include recognition and treatment of acute and chronic pain.

CONSULT THE EXPERTS PAGE 63

Continues on page 14

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IMPORTANT SAFETY INFORMATION: ENTYCE® (capromorelin oral solution) is for use in dogs only. Do not use in breeding, pregnant or lactating dogs. Use with caution in dogs with hepatic dysfunction or renal insufficiency. Adverse reactions in dogs may include diarrhea, vomiting, polydipsia, and hypersalivation. Should not be used in dogs that have a hypersensitivity to capromorelin. Please see the full Prescribing Information at entyce.aratana.com/PI.

See page 12 for product information summary.

SHANNA HILLSMAN, LVMT, is a senior technician in the intensive care unit and emergency service at University of Tennessee Veterinary Medical Center, where she teaches technical skills and mentors veterinary technician interns. She is also a blood bank technician and was a speaker at the Veterinary Partners Appreciation Conference in Knoxville, Tennessee.

DIFFERENTIAL DIAGNOSIS PAGE 53

EMILY KLOCKE, DVM, DACVS, is a clinical associate professor of small animal surgery at Kansas State University. She earned her DVM from Michigan State University and completed an internship and residency at Purdue University. Her interests are teaching senior veterinary students and training interns and surgical residents in soft tissue surgery.

CASE IN POINT PAGE 29

KATE KUKANICH, DVM, PhD, DACVIM (SAIM), is an associate professor of small animal internal medicine at Kansas State University. She earned her DVM from University of Minnesota and her PhD from University of Tennessee, where she also completed an internal medicine residency. Her clinical interests are infectious disease, respiratory disease, and public health.

CASE IN POINT PAGE 29

GLENN A. OLAH, DVM, PhD, DABVP (Feline), is the president of Winn Feline Foundation, a nonprofit organization devoted to funding feline health research and education, as well as an associate veterinarian at Albuquerque Cat Clinic in Albuquerque, New Mexico. He earned his PhD in biophysics from Rice University and his DVM from Oklahoma State University. His interests include all aspects of feline clinical medicine, particularly geriatric medicine.

DIAGNOSTIC/MANAGEMENT TREE PAGE 18
TOP 5 PAGE 54

DAVID F. SENIOR, BVSc, DACVIM (SAIM), DECVIM-CA, professor emeritus, Louisiana State University, earned his veterinary degree from University of Melbourne, where he also completed a 1-year rotating clinical internship. He also completed a residency in small animal internal medicine at University of Pennsylvania. Dr. Senior chaired the Louisiana State University clinical department for 15 years and is a founding member of the *Clinician's Brief* advisory board.

Rx SOLUTIONS PAGE 66

M. KATHERINE TOLBERT, DVM, PhD, DACVIM (SAIM), is an assistant professor at University of Tennessee. Dr. Tolbert earned her DVM from University of Georgia, where she also completed a small animal internship. She earned her PhD in comparative biomedical sciences and completed an internal medicine residency at North Carolina State University. Her clinical research is focused on the investigation of gastroprotectants and the rationale for their use in the treatment of inflammatory, metabolic, and neoplastic diseases in small animals.

DIFFERENTIAL DIAGNOSIS PAGE 53

KATHERINE TUCKER-MOHL, VMD, practices at Diagnostic Imaging, P.C., in Aurora, Colorado. She worked in emergency practice before completing a residency in diagnostic imaging at Kansas State University. Her clinical interests include ultrasonography and musculoskeletal imaging.

CASE IN POINT PAGE 29

MEGAN WILSON, DVM, recently completed a small animal surgery residency at Kansas State University and will soon start working at First Coast Veterinary Specialists in Jacksonville Beach, Florida. She earned her DVM from and completed an internship at University of Saskatchewan, where she also completed a rotating internship. Dr. Wilson also finished a small animal surgery internship at Affiliated Veterinary Specialists in Maitland, Florida.

CASE IN POINT PAGE 29

TIPS & TECHNIQUES
at New York Vet 2018

New York Vet features sessions and speakers carefully selected by practicing veterinarians, for practicing veterinarians—with guaranteed practicality.

We Asked …

What is the key takeaway that attendees will learn from your session?

The Speakers Responded …

Attendees should feel comfortable approaching any mast cell tumor case—and have a meaningful conversation with owners regarding diagnostics, prognosticators, and basic treatment options.

What's New in Mast Cell Tumors for GPs?
Andy Abbo, DVM, MS, DACVIM (Oncology)

Attendees will learn easily applied changes in perianesthetic management that can positively impact outcome in renal disease patients.

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Khursheed Mama, DVM, DACVAA

What might walk into your clinic as a dermatology emergency, and how will you handle it? We'll discuss your options.

What?! You Have a Derm Emergency?!
Karin Beale, DVM, DACVD

You'll walk away knowing the top causes of back pain in dogs and cats and how to clinically recognize a neuromuscular disease.

Polyneuropathies in Dogs: Working up the Weakness and **How to Work Up Back Pain Without Brain Pain**
Simon Platt, BVMS, MRCVS, DACVIM (Neurology), DECVN

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A prostaglandin E_2 (PGE_2) EP4 receptor antagonist; a non-cyclooxygenase inhibiting, non-steroidal anti-inflammatory drug

Caution: Federal (USA) law restricts this drug to use by or on the order of a licensed veterinarian.

Before using this product, please consult the product insert, a summary of which follows:

Indication: GALLIPRANT (grapiprant tablets) is indicated for the control of pain and inflammation associated with osteoarthritis in dogs.

Dosage and Administration: Always provide "Information for Dog Owners" Sheet with prescription. Use the lowest effective dose for the shortest duration consistent with individual response.

The dose of GALLIPRANT (grapiprant tablets) is 0.9 mg/lb (2 mg/kg) once daily.

GALLIPRANT tablets are scored and dosage should be calculated in half tablet increments. Dogs less than 8 lbs (3.6 kgs) cannot be accurately dosed. **See product insert for complete dosing and administration information.**

Contraindications: GALLIPRANT should not be used in dogs that have a hypersensitivity to grapiprant.

Warnings: Not for use in humans. Keep this and all medications out of reach of children and pets. Consult a physician in case of accidental ingestion by humans. **For use in dogs only.** Store GALLIPRANT out of reach of dogs and other pets in a secured location in order to prevent accidental ingestion or overdose.

Precautions: The safe use of GALLIPRANT has not been evaluated in dogs younger than 9 months of age and less than 8 lbs (3.6 kg), dogs used for breeding, or in pregnant or lactating dogs. Adverse reactions in dogs receiving GALLIPRANT may include vomiting, diarrhea, decreased appetite, mucoid, watery or bloody stools, and decreases in serum albumin and total protein.

If GALLIPRANT is used long term, appropriate monitoring is recommended.

Concurrent use with other anti-inflammatory drugs has not been studied. Concomitant use of GALLIPRANT with other anti-inflammatory drugs, such as COX-inhibiting NSAIDs or corticosteroids, should be avoided. If additional pain medication is needed after a daily dose of GALLIPRANT, a non-NSAID/non-corticosteroid class of analgesic may be necessary.

The concomitant use of protein-bound drugs with GALLIPRANT has not been studied. Commonly used protein-bound drugs include cardiac, anticonvulsant and behavioral medications.

Drug compatibility should be monitored in patients requiring adjunctive therapy. Consider appropriate washout times when switching from one anti-inflammatory to another or when switching from corticosteroids or COX-inhibiting NSAIDs to GALLIPRANT use.

The use of GALLIPRANT in dogs with cardiac disease has not been studied.

It is not known whether dogs with a history of hypersensitivity to sulfonamide drugs will exhibit hypersensitivity to GALLIPRANT. GALLIPRANT is a methylbenzenesulfonamide.

Adverse Reactions: In a controlled field study, 285 dogs were evaluated for safety when given either GALLIPRANT or a vehicle control (tablet minus galliprant) at a dose of 2 mg/kg (0.9 mg/lb) once daily for 28 days. GALLIPRANT-treated dogs ranged in age from 2 yrs to 16.75 years. The following adverse reactions were observed:

Adverse reaction*	GALLIPRANT (grapiprant tablets) N = 141	Vehicle control (tablets minus grapiprant) N = 144
Vomiting	24	9
Diarrhea, soft stool	17	13
Anorexia, inappetence	9	7
Lethargy	6	2
Buccal ulcer	1	0
Immune mediated hemolytic anemia	1	0

*Dogs may have experienced more than one type or occurrence during the study.

GALLIPRANT was used safely during the field studies with other concurrent therapies, including antibiotics, parasiticides and vaccinations.

To report suspected adverse drug events and/or obtain a copy of the Safety Data Sheet (SDS) or for technical assistance, call 1-888-545-5973.

For additional information about adverse drug experience reporting for animal drugs, contact FDA at 1-888-FDA-VETS or online at http://www.fda.gov/AnimalVeterinary/SafetyHealth

Information for Dog Owners: Owners should be advised of the potential for adverse reactions and be informed of the clinical signs associated with drug intolerance. Adverse reactions may include vomiting, diarrhea, decreased appetite, and decreasing albumin and total protein. Appetite and stools should be monitored and owners should be advised to consult with their veterinarian if appetite decreases or stools become abnormal.

Effectiveness: Two hundred and eighty five (285) client-owned dogs were enrolled in the study and evaluated for field safety. GALLIPRANT-treated dogs ranging in age from 2 to 16.75 years and weighing between 4.1 and 59.6 kgs (9-131 lbs) with radiographic and clinical signs of osteoarthritis were enrolled in a placebo-controlled, masked field study. Dogs had a 7-day washout from NSAID or other current OA therapy. Two hundred and sixty two (262) of the 285 dogs were included in the effectiveness evaluation. Dogs were assessed for improvements in pain and function by the owners using the Canine Brief Pain Inventory (CBPI) scoring system.[1] A statistically significant difference in the proportion of treatment successes in the GALLIPRANT group (63/131 or 48.1%) was observed compared to the vehicle control group (41/131 or 31.3%). GALLIPRANT demonstrated statistically significant differences in owner assessed pain and function. The results of the field study demonstrate that GALLIPRANT, administered at 2 mg/kg (0.9 mg/pound) once daily for 28 days was effective for the control of pain and inflammation associated with osteoarthritis.

Storage Conditions: Store at or below 86° F (30° C)

How Supplied: 20 mg, 60 mg, 100 mg flavored tablets in 7, 30 and 90 count bottles.

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Reference: 1. http://www.vet.upenn.edu/docs/default-source/VCIC/canine-bpi_userguide.pdf?sfvrsn=0
Additional information is available at 1-888-545-5973.
GALLIPRANT is a trademark of Aratana Therapeutics, Inc.

Brief Summary: AT1-040-16

Indication

Galliprant is an NSAID indicated for the control of pain and inflammation associated with osteoarthritis (OA) in dogs.

Important Safety Information

Not for use in humans. For use in dogs only. Keep this and all medications out of reach of children and pets. Store out of reach of dogs and other pets in a secured location in order to prevent accidental ingestion or overdose. Do not use in dogs that have a hypersensitivity to grapiprant. If Galliprant is used long term, appropriate monitoring is recommended. Concomitant use of Galliprant with other anti-inflammatory drugs, such as COX-inhibiting NSAIDs or corticosteroids, should be avoided. Concurrent use with other anti-inflammatory drugs or protein-bound drugs has not been studied. The safe use of Galliprant has not been evaluated in dogs younger than 9 months of age and less than 8 lbs (3.6 kg), dogs used for breeding, pregnant or lactating dogs, or dogs with cardiac disease. The most common adverse reactions were vomiting, diarrhea, decreased appetite, and lethargy. Please see brief summary to the left for full prescribing information.

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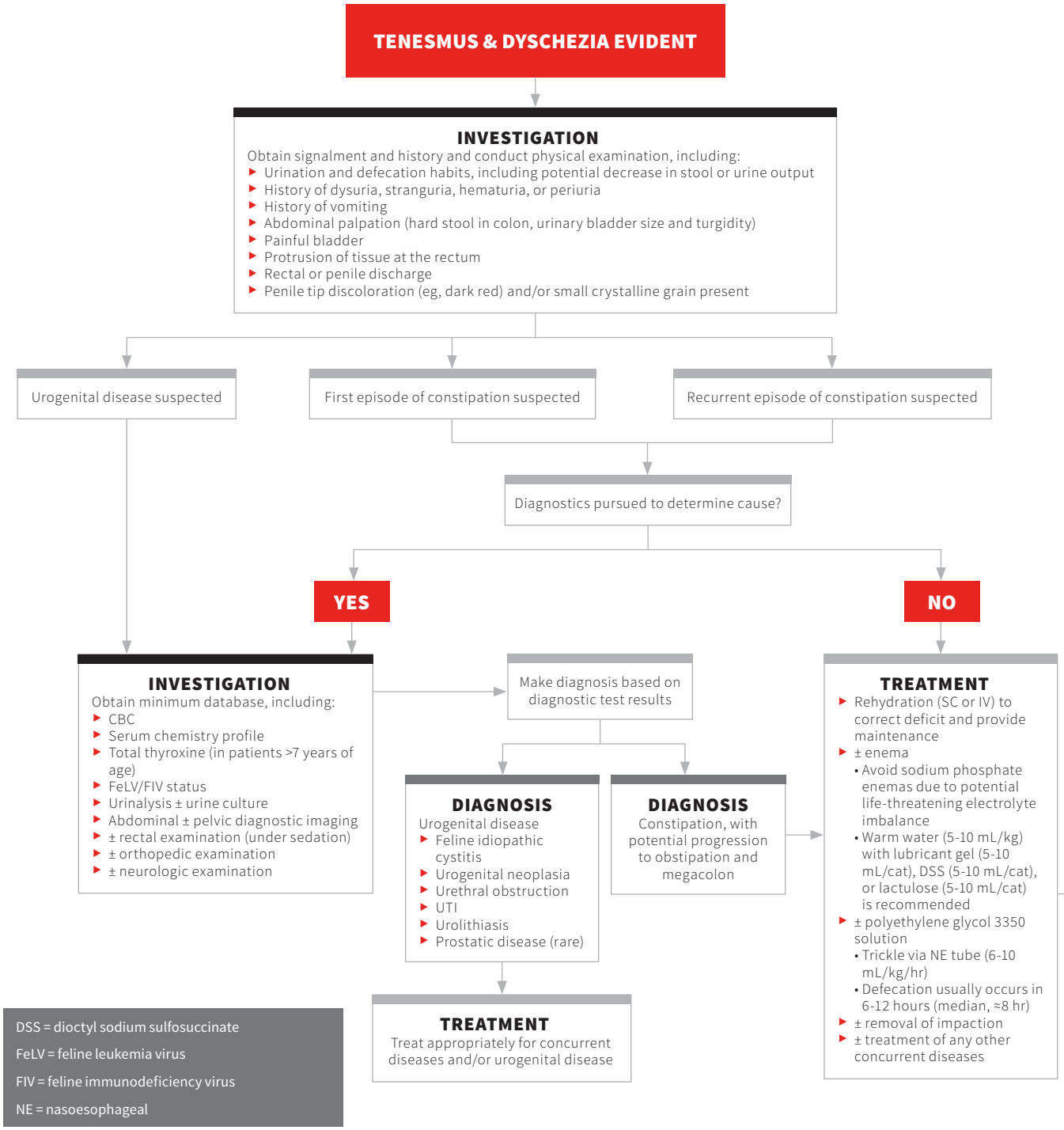
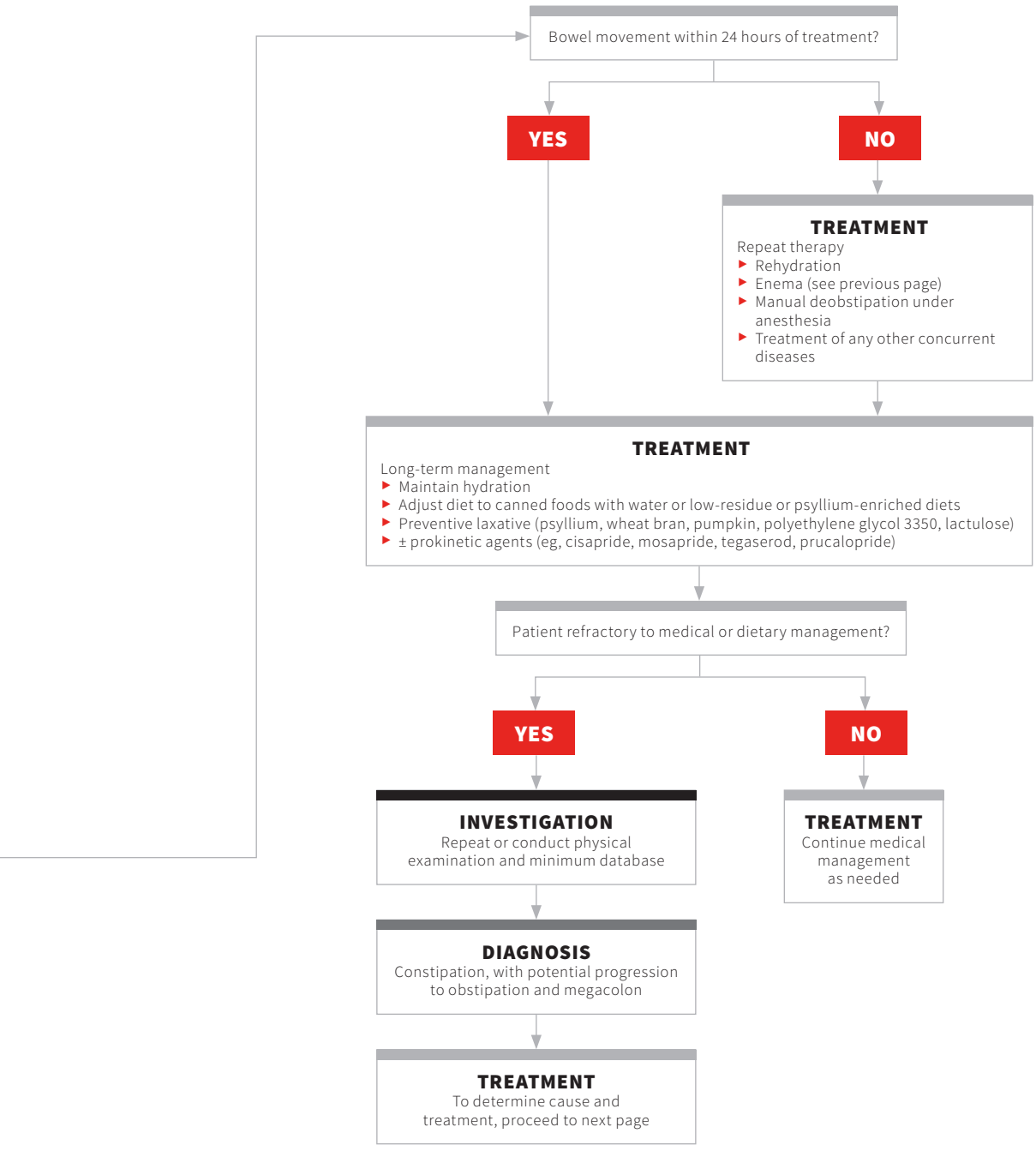
ARATANA THERAPEUTICS

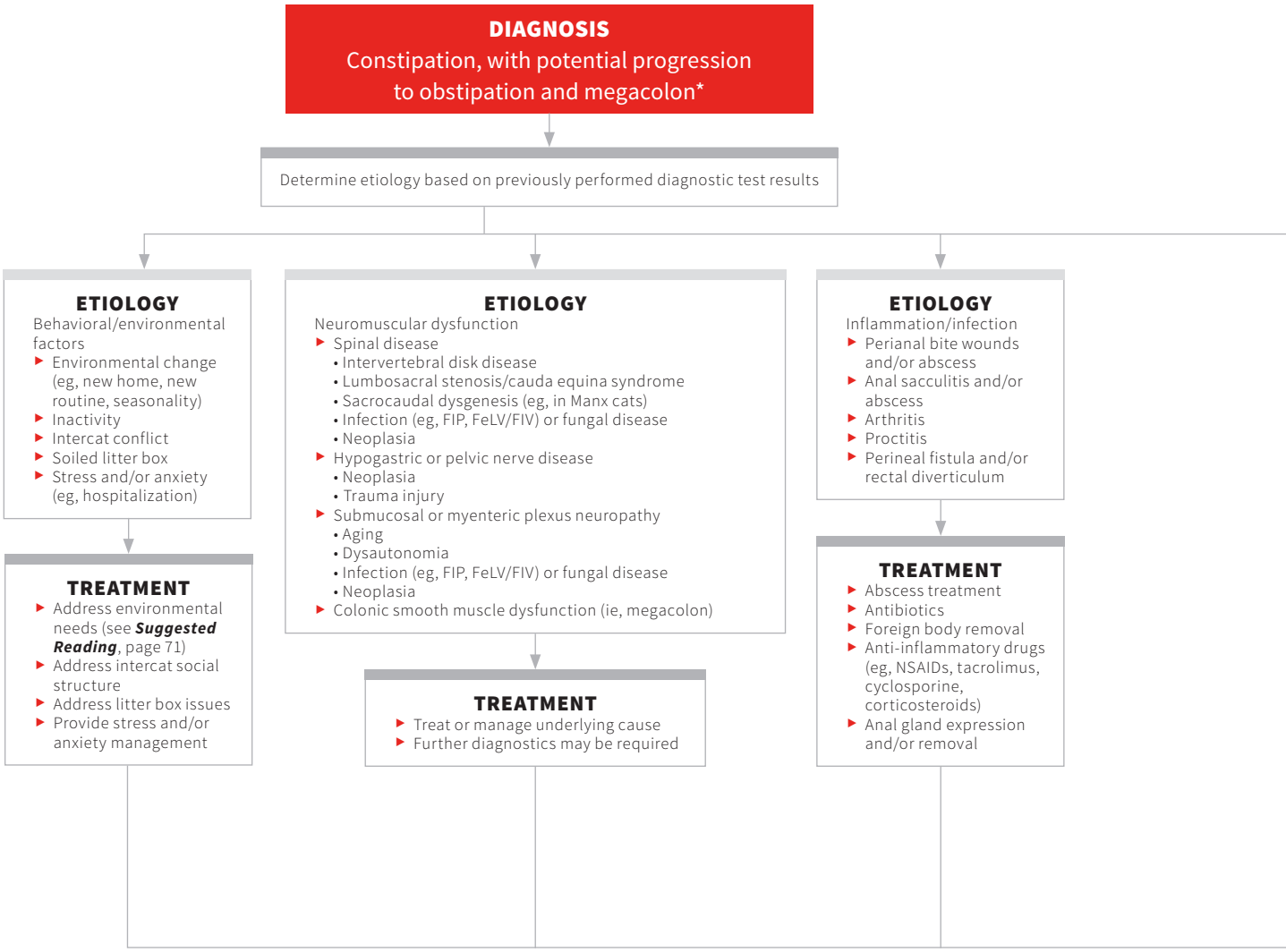
Elanco

DIAGNOSING CONSTIPATION, OBSTIPATION, & MEGACOLON IN CATS

Glenn A. Olah, DVM, PhD, DABVP (Feline)
Winn Feline Foundation
Albuquerque Cat Clinic
Albuquerque, New Mexico

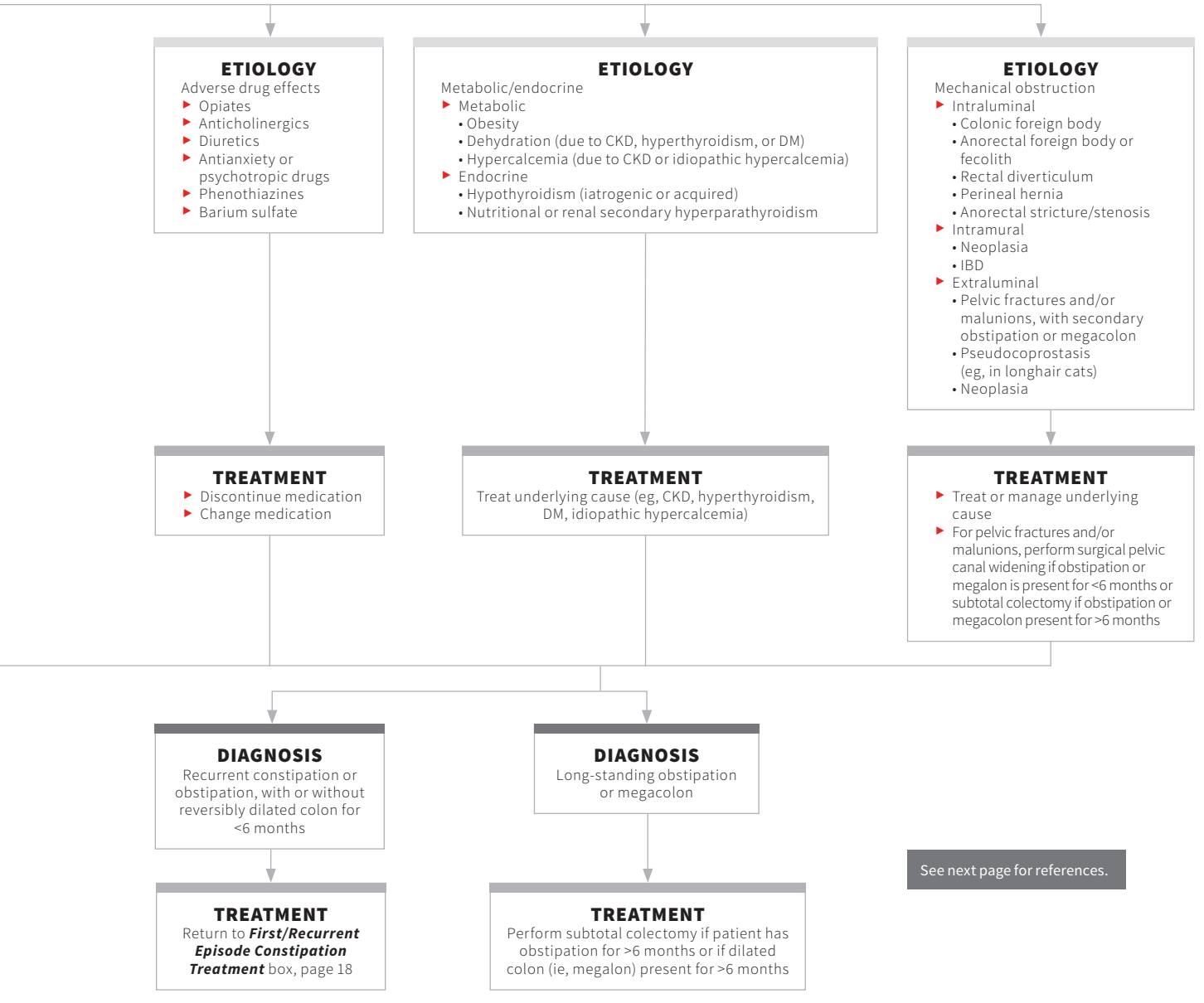



Bowel movement within 24 hours of treatment?
YES
NO
TREATMENT
Repeat therapy
Rehydration
Enema (see previous page)
Manual deobstipation under anesthesia
Treatment of any other concurrent diseases
TREATMENT
Long-term management
Maintain hydration
Adjust diet to canned foods with water or low-residue or psyllium-enriched diets
Preventive laxative (psyllium, wheat bran, pumpkin, polyethylene glycol 3350, lactulose)
± prokinetic agents (eg, cisapride, mosapride, tegaserod, prucalopride)
Patient refractory to medical or dietary management?
YES
NO
INVESTIGATION
Repeat or conduct physical examination and minimum database
TREATMENT
Continue medical management as needed
DIAGNOSIS
Constipation, with potential progression to obstipation and megacolon
TREATMENT
To determine cause and treatment, proceed to next page



*Megacolon is suggestive of neuromuscular dysfunction.

CKD = chronic kidney disease
DM = diabetes mellitus
FeLV = feline leukemia virus
FIP = feline infectious peritonitis
FIV = feline immunodeficiency virus
IBD = inflammatory bowel disease



See next page for references.

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Continues on page 71

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Mirataz™ (mirtazapine transdermal ointment) is for topical use in cats only under veterinary supervision. Do not use in cats with a known hypersensitivity to mirtazapine or any of the excipients. Do not use in cats treated with monoamine oxidase inhibitors (MAOIs). Not for human use. Keep out of reach of children. Wear gloves when handling/applying, wash hands after and avoid contact between the treated cat and people or other animals for 2 hours following application. Use with caution in cats with hepatic and kidney disease. Cat's food intake should be monitored upon discontinuation. Safety has not been evaluated in cats less than 2 kg, less than six months of age or in breeding, pregnant or lactating cats. The most common adverse reactions observed during clinical trials were application site reactions, behavioral abnormalities (vocalization and hyperactivity) and vomiting. **For additional safety information, see brief summary of prescribing information on page 22.**

Reference: 1. Mirataz™ (mirtazapine transdermal ointment) [package insert], Kindred Biosciences, Inc. (Burlingame, CA). Rev. 5/2018. **2.** Buhles W, Quimby JM, Labelle D, et al. Single and multiple dose pharmacokinetics of a novel transdermal ointment in cats. J Vet Pharmacol Ther. In press 2018.

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June 14-16, 2018
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New Understandings & New Treatments of Brachycephalic Breeds

Brachycephalic syndrome, also known as brachycephalic obstructive airway syndrome (BOAS), affects multiple body systems. This presentation focused on the respiratory components, which are characterized by sleep-disordered breathing, stenotic nares, aberrant rostral and caudal nasal turbinates, abnormal thermoregulation, elongated soft palate, laryngeal collapse and edema, macroglossia, nasopharyngeal sialoceles, and tracheal hypoplasia. To appropriately treat a patient with BOAS, it is important to remember that clinical signs of noisy breathing, exercise intolerance, inspiratory effort, and others should not be considered normal for the breed. Upper airway disease can also be responsible for a variety of esophageal, gastric, and duodenal disorders, and BOAS patients may be presented for reflux, vomiting, and gastritis. Evaluation and diagnosis of BOAS should include thin-slice CT of the head, trachea, and chest, as well as anterior and posterior rhinoscopy. Surgical correction for BOAS includes the wedge technique on the wing of the ala, shortening the elongated soft palate, and removal of everted laryngeal saccules. Newer procedures include intranasal laser-assisted turbinectomy to remove rostral and caudal aberrant turbinates, as well as more aggressive stenotic nares surgery. Because of their propensity for gastric reflux and the increased risk for aspiration during anesthesia, BOAS patients should have a prolonged fast prior to anesthetic procedures in addition to pre- and postsurgical treatment with acid suppression and promotility agents. It is recommended that the profession be aware of how selective breeding is influencing animal welfare in brachycephalic breeds.—*McKiernan BC*

Radioiodine Treatment in Cats with Hyperthyroidism

Although radioiodine treatment is considered the gold standard for feline hyperthyroidism, the method for choosing the radioiodine (I-131) dose is not clear. Clinicians either administer a 4-mCi fixed dose or choose from a range of 3 to 5 mCi. Although this resolves hyperthyroidism for most cats, onset of overt or subclinical iatrogenic hypothyroidism occurs in 70% of patients, resulting in increased progression of kidney disease and shortened life span.

This study of 450 cats sought to create a more individualized approach to treatment. For cats initially treated medically, methimazole was stopped at least a week before the study. Thyroxine, triiodothyronine, and thyroid-stimulating hormone levels were measured in each patient. Quantitative thyroid scintigraphy was used to evaluate thyroid volume and activity. Each cat received I-131 at an initial dose of 1-1.5 mCi to measure radioactivity uptake. Using these data, a final radiation dose of 200 mCi I-131 per gram of thyroid tumor tissue was administered to all cats. A second dose was administered to meet the final calculated dose when necessary. Of the study cats, 75.3% became euthyroid, whereas only 3.1% developed overt hypothyroidism, 16.9% became subclinically hypothyroid, and 4.7% remained hyperthyroid. This new algorithm yielded similar success rates as compared with traditionally used protocol doses but with greatly diminished occurrence of subclinical or overt hypothyroidism, thus reducing the rate of associated azotemia. Radiation exposure to patients and pet caretakers was also limited.—*Peterson ME*

SAVE THE DATE

2019 American College of Veterinary Internal Medicine Forum

June 6-8, 2019
Phoenix, Arizona

Key Concepts of Feline Diabetic Remission

Similar to humans with type-2 diabetes mellitus (DM), most diabetic cats have compromised β-cell function as a result of insulin resistance and pancreatic amyloidosis. Other factors that may influence diabetic development include acromegaly, chronic pancreatitis, and genetic predisposition. Metabolic and inflammatory processes and some medications may impact insulin responsiveness. Many diabetic cats likely experience a preclinical diabetic state in which blood glucose continues to rise until overt DM develops. These patients often progress to glucose toxicity whereby β-cell function is lost and insulin production ceases. If blood glucose can be controlled before β-cell apoptosis develops, insulin secretion could potentially be restored. Diabetic cats should be fed high-protein, low-carbohydrate (<12%) diets. Incretin analogues are licensed for use in humans with type-2 DM to improve insulin sensitivity and promote growth of new islet cells while supporting gradual weight loss and suppressing food intake. Similar effects have been observed in cats.

When treating cats with the goal of achieving diabetic remission, suitable patients and owners must be identified, insulin resistance and dietary issues must be addressed, and blood glucose must be controlled. Ideal patients have transient or reversible causes of insulin resistance, including obesity, exogenous steroid use, and dental disease. Remission is unlikely in cats with chronic inflammatory or endocrine disease. The presenter recommends long-acting insulins to avoid short periods of hyperglycemia. One unit of insulin glargine twice daily has been suggested as a starting point, after which owners should be instructed to adjust the insulin dose based on home glucose readings after the initial 3 days, with a goal of 65-220 mg/dL blood glucose. If all readings are below 120 mg/dL, insulin should be gradually decreased.—*Cook AK*

Remission is unlikely in cats with chronic inflammatory or endocrine disease.

Interstitial & Diffuse Parenchymal Lung Diseases

Interstitial lung diseases (ILDs), sometimes termed diffuse parenchymal lung diseases (DPLDs), are a large and heterogenous set of pulmonary disorders involving changes to the distal lung parenchyma, including airways, pulmonary parenchyma, blood vessels, and/or pleura. More than 200 distinct ILDs have been described in humans, but only approximately a dozen have been described in veterinary patients. ILDs are typically difficult to diagnose and involve close collaboration between clinicians, radiologists, and pathologists. Lung biopsy and histologic examination are often required for confirmation of diagnosis; however, these can be both cost-prohibitive and unduly risky in patients with respiratory compromise. Even when histopathology is performed, diagnosis may still be elusive, as so few diseases have been fully characterized in veterinary medicine.

Idiopathic pulmonary fibrosis (IPF) is one of the most commonly diagnosed ILDs in dogs and cats. In humans, diagnosis is based on the presence of a "usual interstitial pattern" on CT, along with specific historic, physical, and clinical laboratory findings. It is important to note that the presence of pulmonary fibrosis alone does not constitute a diagnosis of IPF, as is often mistakenly done in veterinary medicine. Veterinary patients must be fully evaluated for other potential causes of pulmonary fibrosis. If underlying inflammatory disease, inhalational injury, drugs, or infection are not fully excluded, the opportunity for specific treatment before end-stage fibrosis sets in may be missed. Because of the numerous potential causes of pulmonary fibrosis in veterinary patients, it has been proposed that the term *fibrotic lung disease* or *fibrotic ILD* replace IPF in veterinary medicine. Other less common interstitial lung diseases that have been poorly characterized in veterinary patients include idiopathic cryptogenic organizing pneumonia, hypersensitivity pneumonitis, pneumoconiosis, and others.—*Reinero C*

Clinical Application of Novel Diagnostics in Gastroenterology

GI signs caused by infectious, inflammatory, or neoplastic conditions can appear clinically similar. Thus, rapid, reliable diagnostic tests to support a diagnosis are important, and many new tests are available.

- Fluorescent in situ hybridization can detect bacteria in tissue via fluorescent microscopy and can be useful in cases of suspected bacterial infection, particularly in patients with conditions in which routine culture results might be negative or equivocal despite high suspicion for bacterial causes (eg, granulomatous and/or neutrophilic inflammation, cholangiohepatitis).
- Two new methods for differentiating lymphoma from inflammatory bowel disease are available for cases in which histopathology is not definitive. PCR for antigen receptor rearrangement (PARR) amplifies DNA, allowing differentiation between neoplastic and inflammatory lymphocytes. Immunohistochemistry detects lineage markers for B and T lymphocytes. Both PARR and immunohistochemistry used together in combination with histopathology may increase the likelihood of obtaining a correct diagnosis.
- Capsule endoscopy is a method used to investigate occult GI bleeding and potential neoplasia and is a safe alternative to routine endoscopy or exploratory surgery.
- Most tests for feline infectious peritonitis have low sensitivity and specificity and may not be able to differentiate between enteric and virulent feline coronavirus. All test results must be interpreted cautiously, and a combination of serology-based assays and reverse transcriptase PCR testing may yield better results.
- Four pancreatic enzyme assays have demonstrated good-to-excellent agreement with each other; however, any test should be evaluated in conjunction with clinical signs.
- Dysbiosis has been linked to several enteropathies. A rapid quantitative-PCR–based Dysbiosis Index is available to help differentiate dysbiosis from normobiosis; however, more studies are needed to evaluate therapeutic strategies.—*Sullivant AM*

Most tests for feline infectious peritonitis have low sensitivity and specificity and may not be able to differentiate between enteric and virulent feline coronavirus.

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1. Karadzovska, D., et al. (2017). A randomized, controlled field study to assess the efficacy and safety of lotilaner flavored chewable tablets (Credelio™) in eliminating fleas in client-owned dogs in the USA. *Parasites & Vectors*, 10:528. **2.** Murphy, M., et al. (2017). Laboratory evaluation of the speed of kill of lotilaner (Credelio™) against *Ixodes ricinus* ticks on dogs. *Parasites & Vectors*,10:541. **3.** Cavalleri, D., et al. (2017). Assessment of the speed of flea kill of lotilaner (Credelio™) throughout the month following oral administration to dogs. *Parasites & Vectors*, 10:529.

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Intrahepatic Splenosis in a Labrador Retriever

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Megan Wilson, DVM
First Coast Veterinary Specialists
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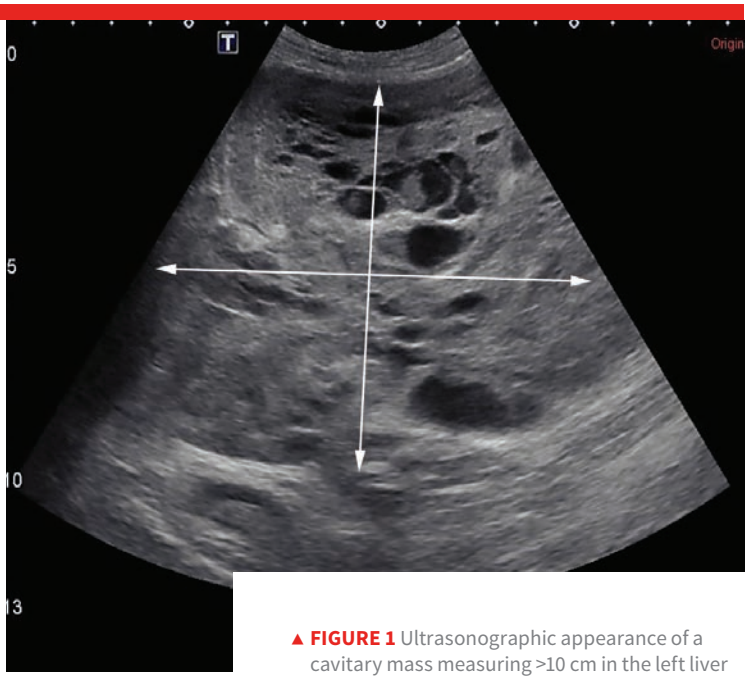


▲ **FIGURE 1** Ultrasonographic appearance of a cavitary mass measuring >10 cm in the left liver on initial presentation. *Area of mass demonstrated by arrows*

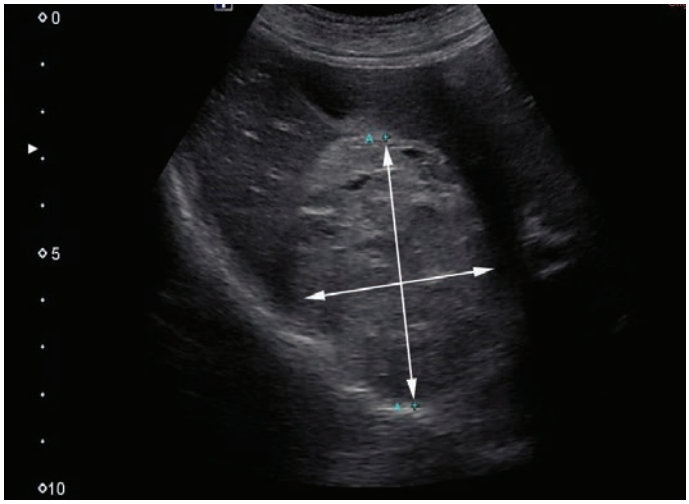
Sophie, a 9-year-old spayed Labrador retriever, was presented to the internal medicine service for abdominal distension. She had a clinical history of acute hemoabdomen, splenectomy, and benign hemangioma on splenic histopathology 2 years prior to presentation. One month prior to presentation, the owners noted that Sophie was gaining weight and that her abdomen appeared larger than usual. Abdominal radiographs obtained by the primary veterinarian revealed a large mid-abdominal mass. She was up-to-date on vaccinations and received routine flea, tick, and heartworm preventives.

Physical Examination Findings

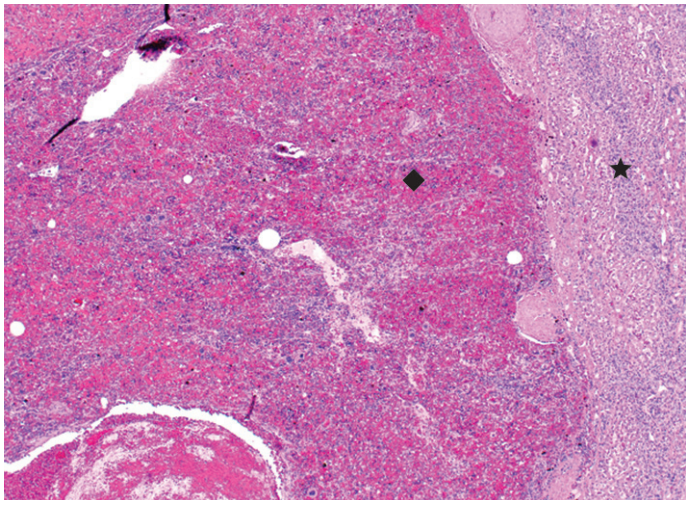
Sophie was bright, alert, and responsive, with normal vital signs. The only abnormal examination findings were abdominal distension and a large and palpable left-sided cranial abdominal mass.

Diagnosis

Mild leukocytosis (17.3 × 10^3/µL; reference range, 4.3-13.6 × 10^3/µL) and mild regenerative anemia (hematocrit, 33% [reference range, 40%-57%]; reticulocytes, 150 000/µL) were noted. The remainder of the CBC, coagulation profile, and serum chemistry profile were within normal limits. Abdominal ultrasonography



▲ **FIGURE 2** Ultrasonographic appearance of a second hepatic mass measuring 5.7 cm on initial presentation. *Area of mass demonstrated by arrows*



▲ **FIGURE 3** Histopathology of hepatic mass with compressed hepatocytes (***star***) and splenic red pulp (***diamond***). Necrosis can be seen in the lower left corner.

demonstrated a large (>10 cm in diameter) hepatic mass (***Figure 1***, previous page) originating from the left aspect of the liver that contained numerous variably sized anechoic regions and mild anechoic peritoneal effusion. Two smaller masses were visible in the hepatic parenchyma (***Figure 2***). Ultrasound-guided fine-needle aspiration of the large hepatic mass was performed, and cytology showed evidence of previous hemorrhage and mild extramedullary hematopoiesis. Ultrasound-guided biopsies were not considered safe because of the risk for hemorrhage due to the cavitated nature of the masses. The top differential diagnosis was malignant neoplasia (eg, hemangiosarcoma, histiocytic sarcoma, hepatic adenocarcinoma); benign (eg, hemangioma) or infectious (eg, fungal or parasitic) lesions were considered possible but unlikely. Thoracic radiographs were unremarkable.

TOP DIFFERENTIAL DIAGNOSIS:
MALIGNANT HEPATIC NEOPLASIA

Treatment & Long-Term Management

Although neoplasia was suspected, it could not be confirmed without histopathology. The owners elected for abdominal exploratory surgery to attempt either complete mass resection or debulking with biopsy for histopathology. The owners were prepared to consult with the oncology team following surgery if neoplasia was confirmed. Three liver masses and one mass adhered to the linea alba with omental adhesions were removed, leaving smaller masses and irregularities throughout the liver. The largest mass (11 cm), located in the left medial liver lobe, was firm and vascular and interspersed with pockets of unclotted blood. The left medial liver lobe containing the largest liver mass, 2 additional liver masses, and a mass adhered to the linea alba with omental adhesions were all removed, leaving smaller masses and irregularities throughout the liver. On histopathologic examination, the hepatic masses were found to be composed of red pulp, extramedullary hematopoietic tissue, rare white pulp, and smooth mus-

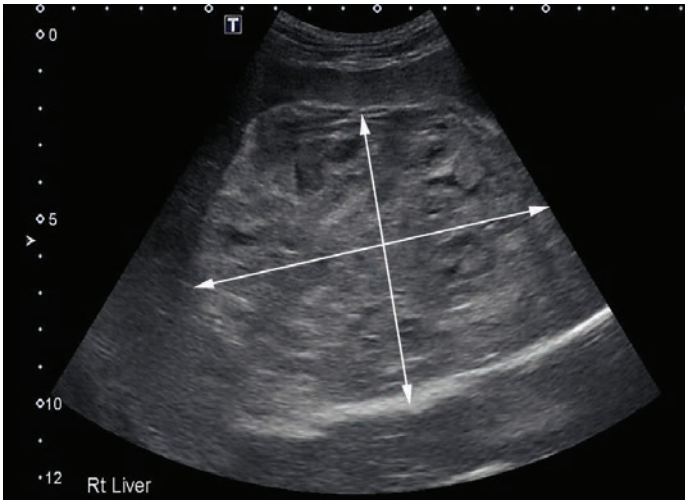
cle trabeculae (***Figure 3***). The mass associated with the linea alba was also composed of ectopic splenic tissue, with similar components, consistent with splenosis. No evidence of neoplasia was seen. Recovery was uneventful, and the patient was discharged 2 days postoperatively.

Prognosis & Outcome

Abdominal radiographs taken 3 months postoperatively were unremarkable. Sophie experienced a brief episode of vomiting 4 months postoperatively, and packed cell volume (PCV) was 34%; however, ultrasonography was not performed, and she recovered without therapy. Five months postoperatively, she was clinically normal, and a routine recheck identified a PCV of 39%. Ultrasonography at this time identified a 10.5-cm cavitated mass in the right liver near the porta hepatis (***Figure 4***) and an 8.6-cm hepatic mass with similar appearance to the presurgical ultrasound findings; hemoabdomen was not present. Although recurrence of hepatic splenosis was suspected, hemangiosarcoma or other neoplasia could not be ruled out. Cytology revealed extramedullary hematopoiesis.

Follow-up abdominal ultrasonography and blood work were performed every 2 to 3 months postoperatively. Sophie underwent a unilateral arytenoid lateralization for laryngeal paralysis (11 months after splenosis diagnosis) and a gastropexy after gastric dilatation-volvulus (1 year after splenosis diagnosis), both without complication. She was clinically stable 14 months postoperatively, with a PCV of 36%, no evidence of hemoabdomen or thoracic metastasis, and a stable appearance of the hepatic masses, with the largest mass measured at 9.85 cm. She continued long-term treatment with Yunnan Baiyao (500 mg PO q8h) for potential hemostatic effect.[1]

Splenosis is a rare complication of splenic trauma or rupture leading to implantation of splenic tissue onto vascular sites in the abdomen or intravascular seeding from surgical splenectomy. Whereas in humans splenosis typically occurs 5 to 10 years after splenectomy,[2] in dogs, splenosis has been



▲ **FIGURE 4** Ultrasonographic appearance of 10.5-cm cavitated mass in the right liver lobe near the porta hepatis on recheck examination 5 months postoperatively. The mass was suspected to be a recurrence of hepatic splenosis, and cytology was consistent with extramedullary hematopoiesis; however, biopsies for histopathology were not pursued. *Area of mass demonstrated by arrows*

INTRAHEPATIC SPLENOSIS AT A GLANCE

- If an abdominal mass is palpated, abdominal radiography and ultrasonography can help further characterize the mass, but prognosis should not be decided on imaging appearance alone.
- Thoracic radiography is recommended to look for pulmonary metastasis prior to laparotomy.
- If removing a large vascular mass, the clinician should be prepared to transfuse the patient.
- All surgically excised masses should be submitted for histopathologic examination.
- Hepatic splenosis is rare, and further prognostic information is needed. Repeating abdominal ultrasonography every 1 to 3 months is suggested to monitor for recurrence.

PCV = packed cell volume

diagnosed concurrently with a splenic lesion[3] or 2 to 5 years after trauma or splenectomy.[4-8] Prior to Sophie's diagnosis, intrahepatic splenosis had been reported in 5 dogs,[4-8] and splenosis had been reported in the pancreas,[3] mesentery,[6,7] abdominal wall,[6,7] diaphragm,[7] and jejunum[6] of dogs. Splenosis can be an incidental finding[6] but more often has been reported with inappetence, weight loss, lethargy, weakness, abdominal distension, and/or abdominal pain in dogs.[3-5,7,8] Two dogs had hemoabdomen secondary to rupture of intrahepatic splenosis lesions.[7,8] Although splenosis is benign in nature, true prognosis and recurrence rate are unknown due to the rarity of this condition. Two of the 5 patients in the literature were euthanized at or around the time of diagnosis, and the remaining 3 cases were reported to have survived at least 5, 8, and 10 months postdiagnosis.[4,6,7] Sophie's survival of at least 14 months (at the time of this article's publication) provides important prognostic information. In contrast, median survival for hemangiosarcoma after surgery and chemotherapy is 6 months.[9]

The Take-Home

Splenosis should be considered a differential diagnosis for hepatic or abdominal masses in dogs with a history of splenic trauma or splenectomy. Not all cavitated hepatic masses in older, large-breed dogs are neoplastic. Histopathology is required to assess whether neoplasia is present so that appropriate treatment options and prognosis can be determined.

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Splenosis should be considered a differential diagnosis for hepatic or abdominal masses in dogs with a history of splenic trauma or splenectomy.

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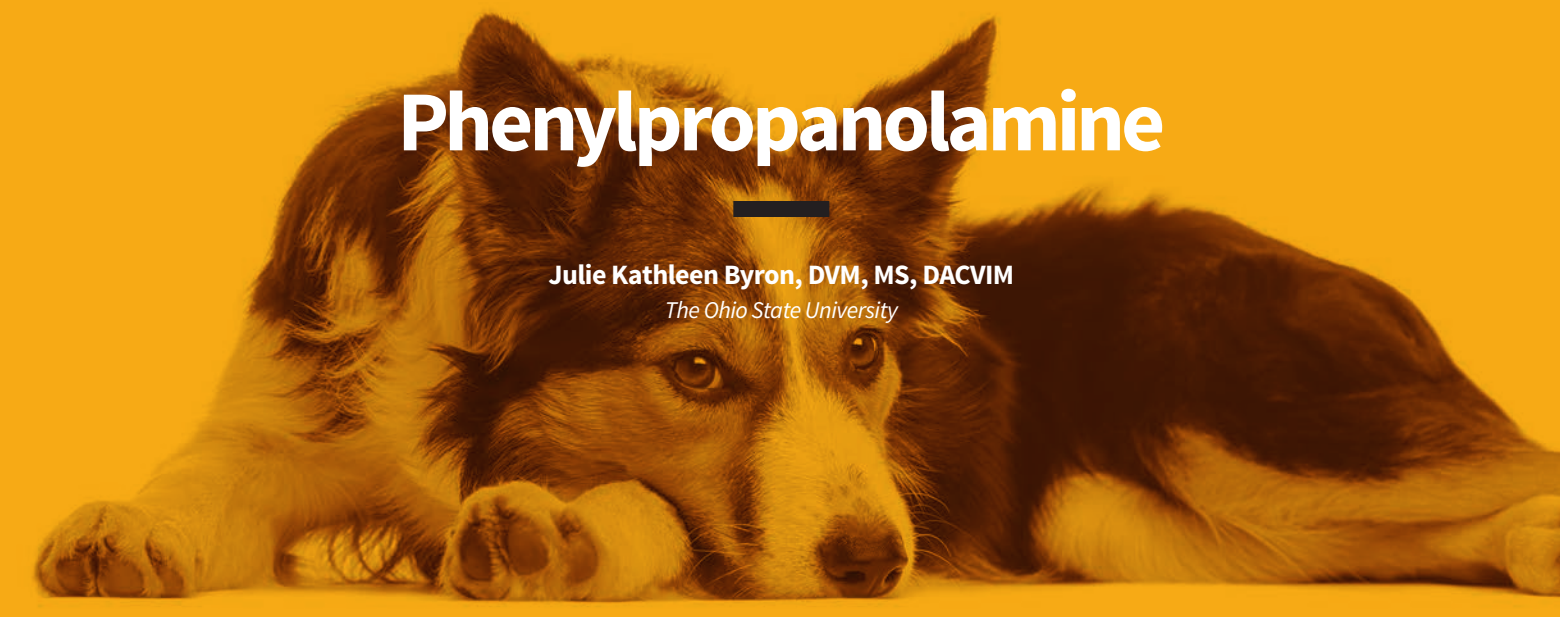

Phenylpropanolamine

Julie Kathleen Byron, DVM, MS, DACVIM
The Ohio State University

Phenylpropanolamine is an α_1-adrenergic agonist used to increase smooth muscle tone in the urethra of dogs with urethral sphincter mechanism incompetence (USMI). It is widely available in approved veterinary products and is often the first-line choice for treatment of urinary incontinence secondary to USMI.[1] Phenylpropanolamine is well tolerated, but specific adverse effects make it less appropriate in some patients, particularly those with or at risk for hypertension.

MECHANISM OF ACTION

- Phenylpropanolamine is a synthetic sympathomimetic amine that acts primarily at α receptors with some β effect, as well as in the CNS.[2]
  - Although the drug's effect on continence has been thought to come primarily from the stimulation of α receptors in urethral smooth muscle, there is evidence that phenylpropanolamine also stimulates β receptors in the bladder, leading to increased detrusor relaxation and lower bladder pressures during the storage phase of micturition (see ***Physiology of Micturition***).[3]

CLINICAL APPLICATIONS & EFFICACY

- Phenylpropanolamine is primarily used to increase urethral tone in spayed dogs with acquired urinary incontinence secondary to USMI.
  - Studies have shown resolution or improvement of incontinence in 85% to 90% of female dogs with USMI.[4]
- Phenylpropanolamine can also be used in cats, but there is little evidence of its efficacy.
  - Anecdotal evidence suggests that many cats with urinary incontinence have underlying urogenital malformations and that the incidence of USMI is likely low.
- Dosages up to 2 mg/kg PO q8-12h are considered safe in patients without comorbidities that can predispose to hypertension or in patients that are vulnerable to negative effects of increased cardiac preload (eg, mitral valve insufficiency).
  - Otherwise healthy dogs receiving higher doses are at risk for clinically significant elevations in blood pressure.[5]
- There is anecdotal evidence that phenylpropanolamine may become less effective over time in some patients and require dose escalation.

USMI = urethral sphincter mechanism incompetence

- This may be due to downregulation of adrenergic receptors, but this is unproven.
- When maximum doses are reached, an estrogen (eg, estriol, diethylstilbestrol) can be added to improve continence.

- There has been speculation that phenylpropanolamine and estrogens have a synergistic effect.
  - Only one study of this effect has been performed and did not support this theory[6]; however, further studies should be conducted to evaluate the interaction of these drugs.
- Anecdotally, patients with intolerance to higher doses of phenylpropanolamine may be maintained on lower doses with the addition of an estrogen (eg, diethylstilbestrol, estriol) at a standard dose.

MONITORING & ADVERSE EFFECTS

- The nonspecific nature of phenylpropanolamine can lead to potential for adverse effects from stimulation of the sympathetic nervous system, including hypertension, agitation, sleeplessness, and decreased appetite.[7]
  - Most adverse effects are mitigated by decreasing the dose or discontinuing use of phenylpropanolamine.
- A recent study showed that dogs receiving standard oral doses of 1 and 2 mg/kg q12h experienced increases in systolic, diastolic, and mean blood pressure, as well as compensatory decreases in heart rate.[5]
  - Of note, changes were not outside normal parameters and therefore are unlikely to be clinically significant.
- Phenylpropanolamine should be used with caution in patients with hypertension, with diseases predisposed to hypertension (eg, hyperadrenocorticism, chronic kidney disease, pheochromocytoma, hyperthyroidism), or with conditions sensitive to increased cardiac preload.
  - Blood pressure should be monitored 2 hours postadministration (at the time of maximal blood levels and smooth muscle effect) and can be evaluated after a single dose.[5,8]
  - The author recommends initial blood pressure measurement and twice-yearly rechecks in healthy patients and monitoring every 3 to 4 months in patients at risk for hypertension.

PHYSIOLOGY OF MICTURITION

Storage Phase

During the normal storage phase, stretch receptors in the bladder wall send afferent signals along the pelvic nerve, which activate a reflex arc through the hypogastric nerve to the urethra. Norepinephrine is released by postganglionic neurons to activate β-adrenergic receptors in the bladder wall, allowing for relaxation and continued filling. Norepinephrine also stimulates α_1-adrenergic receptors in the urethra and causes contraction of the circular and longitudinal smooth muscle surrounding the urethra, thus preventing urine leakage.

In addition to smooth muscle tone, the somatic-mediated contraction of the striated muscle surrounding the urethra is also important for maintenance of continence. With sudden increases in abdominal pressure, afferent signals travel up the pelvic nerve and initiate efferent signals down the pudendal nerve, releasing acetylcholine and activating nicotinic cholinergic receptors, thus causing contraction of the striated muscle.

Voiding Phase

During initiation of voiding, stretch receptors send afferent signals along the pelvic nerve and cranial to the pontine micturition center. Signals from the cerebral cortex and the hypothalamus are processed to determine if the situation is appropriate for initiation of micturition; if so, signals are sent down the pelvic nerve, leading to release of acetylcholine at the postganglionic parasympathetic neurons. Acetylcholine binds to receptors and stimulates bladder smooth muscle contraction. At the same time, inhibitory signals are sent to the sympathetic reflexes, and the urethra relaxes, allowing for normal emptying.

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FROM PAGE TO PATIENT

Designed to bridge the gap between research pages and daily practice protocol, the following pearls offer tips and techniques to grow the general practitioner's clinical excellence.

Characteristics of Granulocytic Anaplasmosis in Dogs

Janet Foley, DVM, MS, PhD
University of California, Davis

In the Literature

Chirek A, Silaghi C, Pfister K, Kohn B. Granulocytic anaplasmosis in 63 dogs: clinical signs, laboratory results, therapy, and course of disease. *J Small Anim Pract.* 2018;59(2):112-120.

FROM THE PAGE ...

This study examined medical records of 974 dogs from Germany with clinical signs suggestive of granulocytic anaplasmosis (GA). Dogs were included in the study if they tested positive for GA via real-time PCR with no comorbidities that could potentially confuse whether signs and laboratory test results were attributable to *Anaplasma phagocytophilum*. Sixty-three dogs met the study criteria and were included in the analysis.

Clinical signs in affected dogs included lethargy and reduced activity, fever, lameness, and pain on joint palpation. Of importance, 13% of dogs had hemorrhage consisting of petechiae, gingival bleeding, epistaxis, pulmonary or vaginal hemorrhage, fresh fecal blood, or hematoma; these signs were often associated with thrombocytopenia. Splenomegaly and hepatomegaly were often observed. CBC findings most consistently included thrombocytopenia; 44% of dogs tested had platelet-bound antibodies. Synovial fluid of dogs with suspected polyarthritis was cellular and occasionally tested positive for GA via real-time PCR. Doxycycline was highly effective in mitigating clinical signs when used, although hematologic abnormalities took several weeks to improve. Polyarthritis and immune-mediated hemolytic anemia were indicators for treatment with steroids or cyclosporine.

The study did not include a control group; thus, it cannot be determined which of the findings might also occur in dogs without GA—and at what frequency—from the same region or with the same signalment. The study authors acknowledged that chronic infection or occult infection in dogs with less notable clinical signs was likely overlooked but indicated that dogs are more likely to be infected in particular geographic regions and during seasons of peak tick activity. Creating a clinical picture of the dog with GA can help improve diagnostic efficiency and ensure that dogs at risk receive appropriate and timely care.

... TO YOUR PATIENTS

Key pearls to put into practice:

1. Obtaining patient travel history, which should include locations recently visited, is crucial, as risk differs across geographic regions and in forests or environments where ticks would quest. Of note, tick-borne GA is seasonal.
2. Screening should include physical examination and complete minimum database (CBC, serum chemistry profile, urinalysis) augmented with PCR testing of blood. PCR-negative results should be rechecked in a few days.
3. Although most dogs with GA respond well to doxycycline, long-term sequelae can include immune-mediated disease. Steroids should be considered when indicated.

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Owner Perceptions of Pet Pain

Kristy Broaddus, DVM, MS, DACVS
Virginia Veterinary Centers
Fredericksburg, Virginia

In the Literature

Simon BT, Scallan EM, von Pfeil DJ, et al. Perceptions and opinions of pet owners in the United States about surgery, pain management, and anesthesia in dogs and cats. *Vet Surg*. 2018;47(2):277-284.

FROM THE PAGE ...

A reliable indicator of pain as perceived by pet caretakers and pet health professionals remains elusive. Perception of pain in animal patients and pain scales are generally based on human models and human experience. Because animals cannot articulate specific pain levels, pain can be overlooked in a suffering animal.

This study gauged owner perception of pet pain related to surgery and medical illness using a short (<5 minutes) questionnaire. Owners (n = 948) responded to questions regarding perception of pain in their pet, effect of pain on their pet, and the need for pain medications after specific surgical or medical therapies.

That perception of pain in pets is challenging and that pain affects quality of life were both appreciated by owners in the study. Healthcare professionals, college graduates, and respondents who had previously experienced surgery on themselves or their pet reported an elevated appreciation of the need for pain control and the need to be informed about the level of pain expected from a procedure. This group also felt it important that a board-certified anesthesiologist perform anesthesia. High-school–educated respondents were less likely to appreciate the need for analgesics after surgery. In general, owners lacked an appreciation for the need to treat medical conditions such as aural infections with pain medications.

Studies such as this underscore the need to educate pet owners on pain perception. Common beliefs, such as a pet cannot be in pain because it is still active or that pain helps ensure a pet will rest, persist. It is essential for veterinarians to provide owners with information that clarifies signs of pain in pets.

... TO YOUR PATIENTS

Key pearls to put into practice:

1. Multimodal pain therapy is important in the management of pain in pets. Local anesthetic blocks, opioid therapy, gabapentin, and NSAIDs (when appropriate) can help owners treat their pet for pain from surgical and nonsurgical conditions.
2. Due to the current opioid crisis in the United States, veterinarians are required to examine pets more frequently before refilling controlled pain medications. This can be used as an opportunity for ongoing owner education on pet comfort.
3. Taking time to educate owners about the perception of pain in pets is invaluable. Describing how bilateral chronic otitis externa can be experienced as painful as cruciate ligament rupture is important. Describing signs of pain that owners may not appreciate (eg, lack of play, lameness, lethargy, grumpiness, inappetence) can be helpful.

Suggested Reading

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Fossum TW. Anesthesia and perioperative multimodal therapy. In: Fossum TW. *Small Animal Surgery*. 4th ed. St. Louis, MO: Elsevier Mosby; 2013:131-133.

Changes to Cephalosporin Susceptibility Reporting

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Friendship Hospital for Animals
Washington, DC

In the Literature

Papich MG, Lindeman C. Cephalexin susceptibility breakpoint for veterinary isolates: Clinical Laboratory Standards Institute revision. *J Vet Diagn Invest.* 2018;30(1):113-120.

FROM THE PAGE ...

Microbial culture and susceptibility testing is an integral part of the diagnosis and treatment of bacterial infections. The Clinical Laboratory Standards Institute (CLSI) evaluates the antibiotic concentration (ie, breakpoint) required to prevent growth of cultured bacteria, the drug's pharmacokinetic properties, and the likelihood of clinical success for typical doses of the antibiotic to determine whether an isolate will be susceptible (S), intermediate (I), or resistant (R) to that particular drug. Cephalothin, a first-generation cephalosporin, has historically been evaluated to predict the susceptibility to all other oral drugs within that class (eg, cephalexin, cefadroxil, cephapirin). However, research has shown that cephalothin is poor at predicting cephalexin susceptibility. This limitation, combined with cephalexin's position as the most commonly prescribed first-generation cephalosporin, highlights the need for cephalexin-specific CLSI guidelines.

This study evaluated bacterial isolates from a 4-year time span to determine minimum inhibitory concentration (MIC) breakpoints for 4 cephalosporins used in veterinary medicine (ie, cephalexin, cephalothin, cefovecin, cefpodoxime) and simulated the likelihood of successful eradication of the infection. The study results showed discrepancies in susceptibility between cephalothin and cephalexin. Most notable were the susceptibility results for the *Staphylococcus pseudintermedius* isolates that were positive for penicillin-binding protein 2A. Only 4.3% of these isolates had MIC values ≤2 µg/mL for cephalexin as compared with 66.3% for cephalothin. These results further confirm the poor agreement of cephalothin to predict cephalexin susceptibility, which has led the CLSI to replace cephalothin with cephalexin for testing canine isolates.

... TO YOUR PATIENTS

Key pearls to put into practice:

1. Microbiology laboratories should replace cephalothin with cephalexin on culture and susceptibility reports.
2. Cephalexin at 25 mg/kg PO q12h has a 90% likelihood of clinical success in dogs when treating isolates with a MIC ≤2 µg/mL. The likelihood of success for isolates with MICs of 4 µg/mL and 8 µg/mL is only 73% and 47%, respectively. Alternative drugs should be considered at these higher MICs based on culture and susceptibility report results.
3. If a bacterial isolate is resistant to oxacillin, it should also be considered resistant to all other β-lactam drugs, including cephalexin and cephalothin. Alternative drug classes should be used to treat such infections.

Diagnosis and Nutritional Management of Cats with Chronic Kidney Disease

Valerie J. Parker, DVM, DACVIM, DACVN

Associate Professor, Department of Veterinary Clinical Sciences
The Ohio State University

Q What are the steps to early diagnosis of cats with chronic kidney disease (CKD)?

A Because CKD is common in older cats and can progress quietly for years, annual screening of cats is extremely important. While it is never too early to begin, this screening should commence by the time cats are 5 or 6 years of age so the veterinarian can establish a baseline for monitoring and evaluation over time.

The appropriate steps in CKD screening and diagnosis include a physical exam that includes assigning a body condition score (BCS) and a muscle condition score (MCS), as well as performing a kidney palpation and a fundic exam (the latter to detect early signs of kidney-related hypertension); a urinalysis to monitor urine specific gravity (USG) and the presence of proteinuria; a complete blood count (CBC); and a serum chemistry analysis.

Q How does symmetric dimethylarginine (SDMA) testing fit into the diagnostic picture?

A Veterinarians are diagnosing CKD in cats earlier than they once did, due in part to the emergence of SDMA testing as a technology for detecting CKD in early stages. My recommendation is to view SDMA as a complementary tool in CKD diagnosis within the context of the entire clinical picture vs. using it in place of traditional diagnostic strategies.

I advise veterinarians against diagnosing CKD on the basis of any one-time screening test. If the SDMA reading is the only finding suggesting the presence of CKD, veterinarians should repeat the test within the next three months to confirm repeatability. SDMA levels can fluctuate from day to day and be affected by hydration, diet, toxin ingestion or medication use. Similarly, elevated blood urea nitrogen (BUN) or creatinine can also be affected by dehydration and may not reflect the presence of kidney disease.

Q What are nutritional recommendations for cats with CKD?

A There has been a shift in philosophy regarding protein reduction in recent years, because switching a cat to a reduced protein diet too early can promote unnecessary loss of lean muscle. The newer veterinary therapeutic early stage renal diets are restricted in phosphorus but provide moderate levels of protein. Ideally, early stage cats should be fed a phosphorus restricted, moderate protein renal diet, then be switched to a diet with reduced protein in later stages.

"In making a dietary recommendation, I always consider a cat's BCS and MCS along with the stage of disease."

In making a dietary recommendation, I always consider a cat's BCS and MCS along with the stage of disease. In thin cats, my goal is to maintain (or ideally gain) weight, while in overweight or obese cats, some weight loss (certainly lack of weight gain) may be advised. In addition, a major determinant is what the cat will eat, since cats with renal disease can become very picky. Typically, I send newly diagnosed cats home with several food options to increase the odds of finding a renal diet — or diets — they will readily accept.

It is important to monitor the patient's weight, BCS and MCS as the disease progresses. If the cat isn't eating enough to maintain weight or the owner is stressed from coax-feeding the cat at every meal, the veterinarian should be comfortable discussing placement of an esophagostomy tube to use for assisted feeding.

Balancing the Need for Protein and Phosphorus in Feline CKD Diets

Raj Naik, DVM
Veterinary Communications Manager
Nestlé Purina PetCare

For years, cats diagnosed with chronic kidney disease (CKD) have been placed on reduced protein renal diets. This protocol was guided by decades-old studies and conventional clinical wisdom that dictated cats with compromised renal function be fed diets that contained reduced protein and were restricted in phosphorus.

Today that conventional wisdom is being challenged. As we learned more about managing cats with CKD, it became clear to researchers at Purina that reducing phosphorus rather than protein should be the primary goal of renal nutrition, particularly in the early stages of CKD. Why? Reducing protein is problematic for any cat, because cats are obligate carnivores.[1,2] When protein needs are not met, cats catabolize muscle and other tissues to manage the demands of protein turnover and meet metabolic needs. Inadequate intake of protein, particularly in senior cats, leads to loss of lean body mass (LBM) and increased mortality.[3]

Staging nutrition for CKD patients

The International Renal Interest Society (IRIS) staging of CKD[4] is based initially on at least two assessments of fasting blood creatinine concentration in stable patients. After that, substaging is based on proteinuria and blood pressure. Stage 1 indicates the cat is nonazotemic but has some other renal abnormality, while stage 2 indicates mild renal azotemia. Stages 3 and 4 are more serious, indicating moderate renal azotemia and increasing risk of systemic clinical signs and uremic crises, respectively.

Purina now offers two therapeutic diets formulated to meet the nutritional needs of cats with different stages of CKD. Purina® Pro Plan® Veterinary Diets NF Kidney Function® Early Care formula contains a moderate amount of protein, which may help cats maintain LBM in early stages of CKD. Purina® Pro Plan® Veterinary Diets NF Kidney Function® Advanced Care formula has a reduced amount of protein to minimize the production of filtered nitrogenous waste products (the side effects of excess urea or uremia), which may occur in the later stages of CKD. Both have restricted phosphorus to help nutritionally manage cats with CKD.

While the IRIS staging describes general parameters for the progression of the disease, there is significant individual variation in how patients progress and the clinical signs they exhibit. Most cats can be switched to the Advanced Care diet when they progress to stages 3 or 4, but individual cats in any stage of CKD may benefit from either diet. Each patient should be assessed to determine which diet is best for them at that time. Patient management is a balancing act: If a switch to a reduced protein diet is made too early, the patient may lose LBM. If the switch to a reduced protein diet is made too late, advanced-stage cats may display clinical signs of protein intolerance, such as decreased appetite and uremic ulcers.

Monitor patients, prepare clients

Keeping a close eye on body weight, body condition and muscle condition throughout the disease process is paramount, as these parameters are linked to patient survival. A nutritional study of healthy aging cats showed a significant relationship between LBM and survival.[5] Another study found that weight loss in cats with CKD is associated with shorter survival.[3]

Because CKD is progressive, it is important to educate owners about the importance of monitoring and maintaining their cats' food intake, and the connection between body condition and their cats' survival and quality of life. With this knowledge, clients can also be prepared for late-stage intervention. This may include feeding tubes, which not only enable supplemental nutrition but also allow clients to administer medications and fluids while reducing stress for both cat and owner. By preparing clients in advance, they can see such measures as positive rather than frightening.

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Prognosis Positive With Early Diagnosis and Clear Communication

James Mathews, DVM
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The bad news? Once a cat develops chronic kidney disease (CKD), there's no cure. The good news? With early diagnosis and appropriate care, cats with CKD can have many years of good-quality life ahead of them.

Put a positive foot forward

When I tell a client their cat has CKD, I have a choice: I can tell them their pet has an incurable disease or I can tell them the long-term prognosis can be very good for their cat — thanks to the fact that we've caught the disease early. In my experience, clients rarely remember everything discussed during a veterinary appointment, so I make sure to focus on the most important points they need to understand. Rather than giving clients an in-depth explanation of the mechanisms of CKD, I focus on the path forward and my strategy for slowing and monitoring disease progression.

Thankfully, early diagnosis is easier than ever before. While symmetric dimethylarginine (SDMA) testing isn't always definitive, it offers another biomarker beyond creatinine and blood urea nitrogen (BUN) that can alert us to the possibility of CKD long before symptoms are present. It's also much more common for private practices to have ultrasound machines, which make it much easier to get urine samples from feline patients. This is important because decreasing urine specific gravity over time is often the first sign of CKD. As a result, I typically can diagnose CKD at stages 1 or 2.

Nutritional management promotes quality of life

If we can catch CKD early, we have a good chance of slowing the disease's progression through nutritional and medical management, thus allowing these cats to experience minimal changes in their day-to-day lives. Nutritionally speaking, one of our primary goals for early management of cats with CKD is to prevent muscle wasting and weight loss. It's critical that cats consume sufficient amounts of protein to maintain lean muscle mass. High-quality protein is also important — a high level of digestibility helps the patient make the most of the amount it eats. As CKD progresses, protein reduction becomes more important to help prevent the development of uremia from protein catabolism.

Early diagnosis of CKD makes it possible to slow the disease progression through nutrition and medical management. This allows cats to experience minimal changes in their day-to-day lives.

As CKD progresses to stages 3 and 4, appetite loss can lead to weight loss and hasten the patient's decline. While appetite stimulants and offering a variety of foods is often helpful, I explain that feeding tubes may be needed to help maintain the cat's weight and nutritional intake. While some clients perceive feeding tubes as an end-of-life last resort, I stress that the opposite is true. Whether a naso-esophageal tube is needed temporarily to help restart the motility of the GI tract or an esophagostomy tube is put in place long-term to ensure adequate nutrient and water intake, assisted feeding can help improve and extend the patient's life.

One of the most important messages for owners is that CKD does not have to mean end of life for their cats, and that cats can have long, happy lives following diagnosis. I urge clients not to let the disease dictate how they feel about their cat, but to instead continue treating their pet with the love and attention it deserves — plus a little extra care and nurturing required for CKD management.

Key Takeaways

- A CKD diagnosis in a feline patient should be based on a combination of physical exam, urinalysis, CBC and serum chemistry analysis, the latter of which can include SDMA testing.
- Keeping a close eye on body weight, body condition and muscle condition throughout the disease process is paramount, as these parameters are linked to patient survival.
- When caught early and managed effectively, clients should know cats with CKD can still have years of good-quality life ahead of them.

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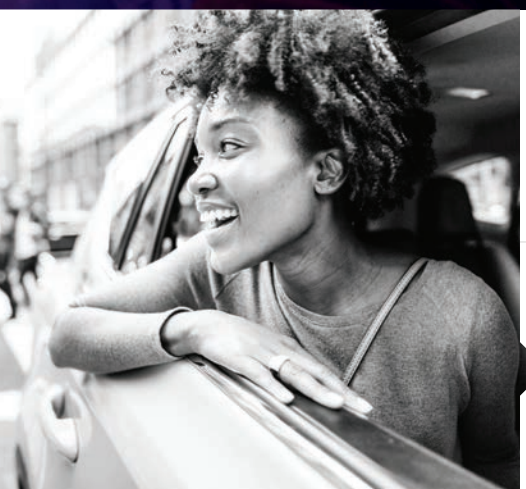

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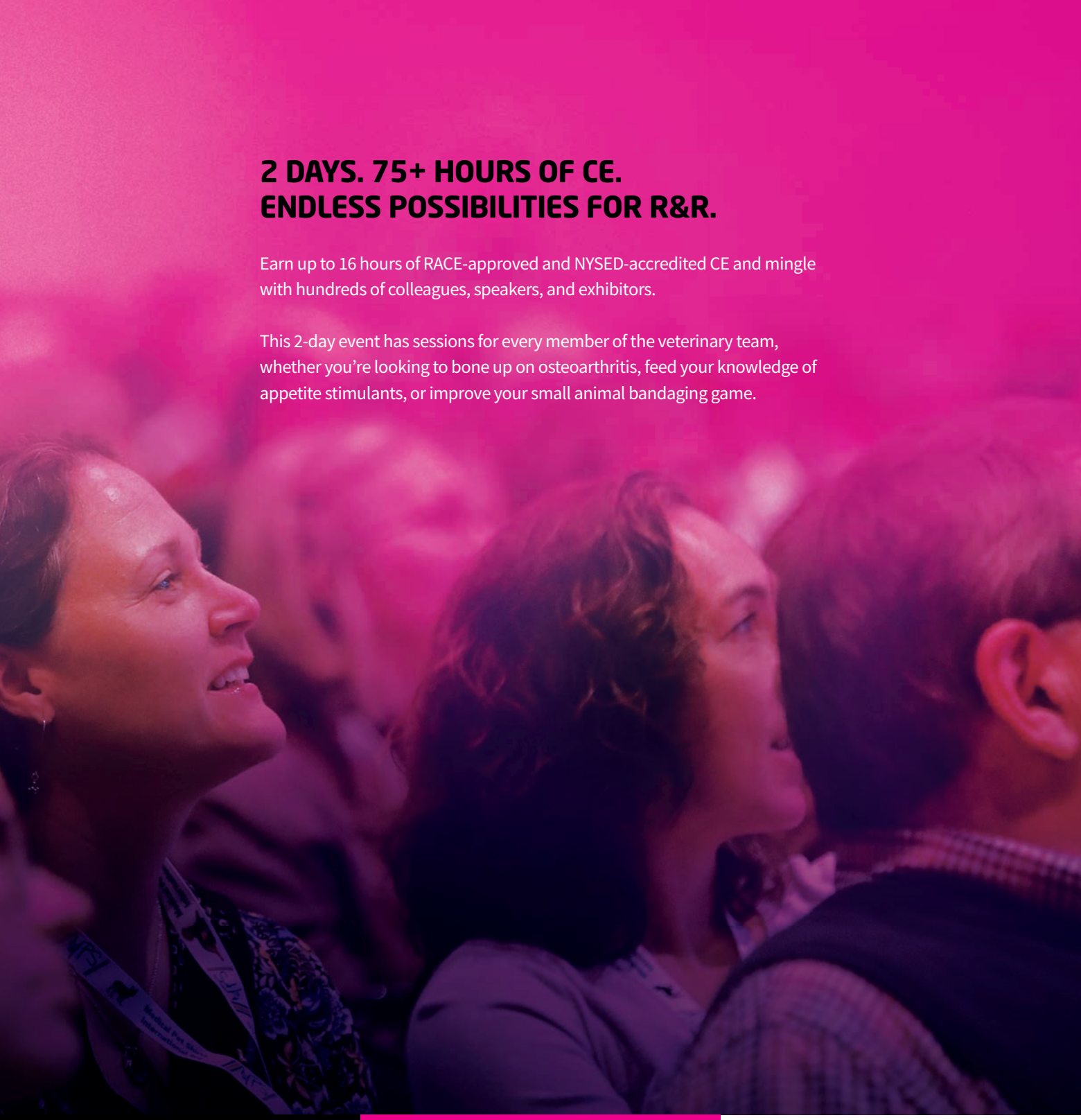

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ProZinc®

(protamine zinc recombinant human insulin)

Caution: Federal law restricts this drug to use by or on the order of a licensed veterinarian.

Description: ProZinc® insulin is a sterile aqueous protamine zinc suspension of recombinant human insulin.

Each mL contains:

recombinant human insulin	40 International Units (IU)
protamine sulfate	0.466 mg
zinc oxide	0.088 mg
glycerin	16.00 mg
dibasic sodium phosphate, heptahydrate	3.78 mg
phenol (added as preservative)	2.50 mg
hydrochloric acid	1.63 mg
water for injection (maximum)	1005 mg

pH is adjusted with hydrochloric acid and/or sodium hydroxide.

Indication: ProZinc (protamine zinc recombinant human insulin) is indicated for the reduction of hyperglycemia and hyperglycemia-associated clinical signs in cats with diabetes mellitus.

Dosage and Administration: USE OF A SYRINGE OTHER THAN A U-40 SYRINGE WILL RESULT IN INCORRECT DOSING.

FOR SUBCUTANEOUS INJECTION IN CATS ONLY.

DO NOT SHAKE OR AGITATE THE VIAL.

ProZinc insulin should be mixed by gently rolling the vial prior to withdrawing each dose from the vial. Once mixed, ProZinc suspension has a white, cloudy appearance. Clumps or visible white particles can form in insulin suspensions: do not use the product if clumps or visible white particles persist after gently rolling the vial. Using a U-40 insulin syringe, the injection should be administered subcutaneously on the back of the neck or on the side of the cat.

Always provide the Cat Owner Information Sheet with each prescription.

The initial recommended ProZinc dose is 0.1 – 0.3 IU insulin/pound of body weight (0.2 – 0.7 IU/kg) every 12 hours. The dose should be given concurrently with or right after a meal. The veterinarian should re-evaluate the cat at appropriate intervals and adjust the dose based on both clinical signs and glucose nadirs until adequate glycemic control has been attained. In the effectiveness field study, glycemic control was considered adequate if the glucose nadir from a 9-hour blood glucose curve was between 80 and 150 mg/dL and clinical signs of hyperglycemia such as polyuria, polydipsia, and weight loss were improved.

Further adjustments in the dosage may be necessary with changes in the cat's diet, body weight, or concomitant medication, or if the cat develops concurrent infection, inflammation, neoplasia, or an additional endocrine or other medical disorder.

Contraindications: ProZinc insulin is contraindicated in cats sensitive to protamine zinc recombinant human insulin or any other ingredients in the ProZinc product. ProZinc insulin is contraindicated during episodes of hypoglycemia.

Warnings: User Safety: For use in cats only. Keep out of the reach of children. Avoid contact with eyes. In case of contact, immediately flush eyes with running water for at least 15 minutes. Accidental injection may cause hypoglycemia. In case of accidental injection, seek medical attention immediately. Exposure to product may induce a local or systemic allergic reaction in sensitized individuals.

Animal Safety: Owners should be advised to observe for signs of hypoglycemia (see Cat Owner Information Sheet). Use of this product, even at established doses, has been associated with hypoglycemia. An animal with signs of hypoglycemia should be treated immediately. Glucose should be given orally or intravenously as dictated by clinical signs. Insulin should be temporarily withheld and, if indicated, the dosage adjusted.

Any change in insulin should be made cautiously and only under a veterinarian's supervision. Changes in insulin strength, manufacturer, type, species (human, animal) or method of manufacture (rDNA versus animal-source insulin) may result in the need for a change in dosage.

Appropriate diagnostic tests should be performed to rule out other endocrinopathies in diabetic cats that are difficult to regulate.

Precautions: Animals presenting with severe ketoacidosis, anorexia, lethargy, and/or vomiting should be stabilized with short-acting insulin and appropriate supportive therapy until their condition is stabilized. As with all insulin products, careful patient monitoring for hypoglycemia and hyperglycemia is essential to attain and maintain adequate glycemic control and to prevent associated complications. Overdosage can result in profound hypoglycemia and death. Progestogens, certain endocrinopathies and glucocorticoids can have an antagonistic effect on insulin activity. Progestogen and glucocorticoid use should be avoided.

Reproductive Safety: The safety and effectiveness of ProZinc insulin in breeding, pregnant, and lactating cats has not been evaluated.

Use in Kittens: The safety and effectiveness of ProZinc insulin in kittens has not been evaluated.

Adverse Reactions:

Effectiveness Field Study

In a 45-day effectiveness field study, 176 cats received ProZinc insulin. Hypoglycemia (defined as a blood glucose value of <50 mg/dL) occurred in 71 of the cats at various times throughout the study. Clinical signs of hypoglycemia were generally mild in nature (described as lethargic, sluggish, weak, trembling, uncoordinated, groggy, glassy-eyed or dazed). In 17 cases, the veterinarian provided oral glucose supplementation or food as treatment. Most cases were not associated with clinical signs and received no treatment. One cat had a serious hypoglycemic event associated with stupor, lateral recumbency, hypothermia and seizures. All cases of hypoglycemia resolved with appropriate therapy and, if needed, a dose reduction.

Three cats had injection site reactions, which were described as either small, punctate, red lesions; lesions on neck; or palpable subcutaneous thickening. All injection site reactions resolved without cessation of therapy.

Four cats developed diabetic neuropathy during the study as evidenced by plantigrade stance. Three cats entered the study with plantigrade stance, one of which resolved by Day 45. Four cats were diagnosed with diabetic ketoacidosis during the study. Two were euthanized due to poor response to treatment. Five other cats were euthanized during the study, one of which had hypoglycemia. Four cats had received ProZinc insulin for less than a week and were euthanized due to worsening concurrent medical conditions.

The following additional clinical observations or diagnoses were reported in cats during the effectiveness field study: vomiting, lethargy, diarrhea, cystitis/hematuria, upper respiratory infection, dry coat, hair loss, ocular discharge, abnormal vocalization, black stool, and rapid breathing.

Extended Use Field Study

Cats that completed the effectiveness study were enrolled into an extended use field study. In this study, 145 cats received ProZinc insulin for up to an additional 136 days. Adverse reactions were similar to those reported during the 45-day effectiveness study and are listed in order of decreasing frequency: vomiting, hypoglycemia, anorexia/poor appetite, diarrhea, lethargy, cystitis/hematuria, and weakness. Twenty cats had signs consistent with hypoglycemia described as: sluggish, lethargic, unsteady, wobbly, seizures, trembling, or dazed. Most of these were treated by the owner or veterinarian with oral glucose supplementation or food; others received intravenous glucose. One cat had a serious hypoglycemic event associated with seizures and blindness. The cat fully recovered after supportive therapy and finished the study. All cases of hypoglycemia resolved with appropriate therapy and if needed, a dose reduction.

Fourteen cats died or were euthanized during the extended use study. In two cases, continued use of insulin despite anorexia and signs of hypoglycemia contributed to the deaths. In one case, the owner decided not to continue therapy after a presumed episode of hypoglycemia. The rest were due to concurrent medical conditions or worsening of the diabetes mellitus.

To report suspected adverse reactions, or to obtain a copy of the Material Safety Data Sheet (MSDS), call 1-866-638-2226.

Information for Cat Owners: Please refer to the Cat Owner Information Sheet for more information about ProZinc insulin. ProZinc insulin, like other insulin products, is not free from adverse reactions. Owners should be advised of the potential for adverse reactions and be informed of the associated clinical signs. Potential adverse reactions include: hypoglycemia, insulin antagonism/resistance, rapid insulin metabolism, insulin-induced hyperglycemia (Somogyi Effect), and local or systemic reactions. The most common adverse reaction observed is hypoglycemia. Signs may include: weakness, depression, behavioral changes, muscle twitching, and anxiety. In severe cases of hypoglycemia, seizures and coma can occur. Hypoglycemia can be fatal if an affected cat does not receive prompt treatment. Appropriate veterinary monitoring of blood glucose, adjustment of insulin dose and regimen as needed, and stabilization of diet and activity help minimize the risk of hypoglycemic episodes. The attending veterinarian should evaluate other adverse reactions on a case-by-case basis to determine if an adjustment in therapy is appropriate, or if alternative therapy should be considered.

Effectiveness: A total of 187 client-owned cats were enrolled in a 45-day field study, with 176 receiving ProZinc insulin. One hundred and fifty-one cats were included in the effectiveness analysis. The patients included various purebred and mixed breed cats ranging in age from 3 to 19 years and in weight from 4.6 to 20.8 pounds. Of the cats included in the effectiveness analysis, 101 were castrated males, 49 were spayed females, and 1 was an intact female.

Cats were started on ProZinc insulin at a dose of 0.1-0.3 IU/lb (0.2-0.7 IU/kg) twice daily. Cats were evaluated at 7, 14, 30, and 45 days after initiation of therapy, and the dose was adjusted based on clinical signs and results of 9-hour blood glucose curves on Days 7, 14, and 30.

Effectiveness was based on successful control of diabetes, which was defined as improvement in at least one blood glucose variable (glucose curve mean, nadir, or fructosamine) and at least one clinical sign (polyuria, polydipsia, or body weight). Based on this definition, 115 of 151 cases (76.2%) were considered successful. Blood glucose curve means decreased from 415.3 mg/dL on Day 0 to 203.2 mg/dL by Day 45, and the mean blood glucose nadir decreased from 407.9 mg/dL on Day 0 to 142.4 mg/dL on Day 45. Mean fructosamine values decreased from 505.9 µmol/L on Day 0 to 380.7 µmol/L on Day 45.

Cats that completed the effectiveness study were enrolled in an extended use field study. The mean fructosamine value was 342.0 µmol/L after a total of 181 days of ProZinc therapy.

How Supplied: ProZinc insulin is supplied as a sterile injectable suspension in 10-mL multidose vials. Each mL of ProZinc product contains 40 IU recombinant human insulin.

Storage Conditions: Store in an upright position under refrigeration at 36-46°F (2-8°C). Do not freeze. Protect from light.

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Important Safety Information: For use in cats only. Animals presenting with severe ketoacidosis, anorexia, lethargy, and/or vomiting should be stabilized with short-acting insulin and appropriate supportive therapy until their condition is stabilized. As with all insulin products, careful patient monitoring for hypoglycemia and hyperglycemia is essential to attain and maintain adequate glycemic control and to prevent associated complications. Overdosage can result in profound hypoglycemia and death. Progestogen and glucocorticoid use should be avoided. PROZINC insulin is contraindicated in cats during episodes of hypoglycemia and in cats sensitive to protamine zinc recombinant human insulin or any other ingredients in the PROZINC product.

References: 1. Nelson RW, Henley K, Cole C; PZIR Clinical Study Group. Field safety and efficacy of protamine zinc recombinant human insulin for treatment of diabetes mellitus in cats. *J Vet Intern Med*. 2009;23(4):787–793. **2.** Nelson RW. Disorders of the endocrine pancreas. In: Nelson RW, Cuoto CG, eds. *Small Animal Internal Medicine*. 4th ed. St. Louis, MO: Mosby Elsevier; 2008:764–802. **3.** Rucinsky R, Cook A, Haley S, Nelson R, Zoran DL, Poundstone M; American Animal Hospital Association (AAHA). AAHA diabetes management guidelines for dogs and cats. *J Am Anim Hosp Assoc*. 2010;46(3):215–224.

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See page 44 for product information summary.

Feline Retroviruses

Glenn A. Olah, DVM, PhD, DABVP (Feline)
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In the Literature

Spada E, Perego R, Sgamma EA, Proverbio D. Survival time and effect of selected predictor variables on survival in owned pet cats seropositive for feline immunodeficiency and leukemia virus attending a referral clinic in northern Italy. *Prev Vet Med*. 2018;150:38-46.

FROM THE PAGE ...

Feline immunodeficiency virus (FIV) can cause an acquired immune deficiency syndrome, predisposing cats to other infections. However, most naturally FIV-infected cats do not develop a severe clinical syndrome, especially if appropriate husbandry and healthcare are provided.[1-3]

Feline leukemia virus (FeLV) is more pathogenic than FIV. Cats with FeLV frequently succumb to fatal diseases associated with FeLV infection (eg, bone marrow suppression leading to nonregenerative anemia, secondary infections, neoplasia), resulting in decreased life expectancy in these cats.[4]

This retrospective cohort study aimed to estimate survival times and evaluate select predictor factors on survival in cats that tested positive for FIV antibodies and/or FeLV antigen. Of the 816 cats tested, 117 (14.3%) tested positive for infection, of which 60 were FIV positive, 46 were FeLV positive, and 11 were both FIV and FeLV positive. Seroprevalence rates for FIV, FeLV, and FIV–and-FeLV–coinfected cats were thus 7.4%, 5.6%, and 1.4%, respectively.

Survival data agreed with previous studies.[2,3,5-9] Survival time for FIV-infected cats was not statistically different as compared with retrovirus-negative cats. Median survival times for FeLV-infected and FIV–and-FeLV–coinfected cats were significantly shorter (714 days and 77 days, respectively) as compared with retrovirus-negative and FIV-infected cats (3960 days and 2040 days, respectively). Median age at diagnosis for FIV-infected cats (5 years) was higher than for FeLV-infected cats (2 years), and median age of coinfected cats was 7 years. Despite shorter survival times, some cats with FeLV and cats with FIV and FeLV lived much longer than their respective median survival times (as long as 8.5 years and 4.9 years, respectively). The wide survival time distribution highlights that FeLV infection is not necessarily suggestive of an immediate death, and clinicians should assess FeLV-infected and coinfected cats case by case. Only reduced RBC count was shown to correlate negatively with median survival time in all retroviral-infected cats.

… TO YOUR PATIENTS

Key pearls to put into practice:

1. FIV and FeLV infection should not be considered indicative of pending death in infected cats.

2. FeLV infection is often more pathogenic and progresses more rapidly than does FIV infection. As represented in this study, causes of death in FeLV-infected cats may primarily be due to lymphoma and, less commonly, anemia. FeLV and FIV coinfection likely results in more severe and rapidly progressive disease.

3. Only reduced RBC counts at time of FIV and FeLV diagnosis have been shown to be a negative prognostic indictor for survival; in this study, FeLV-infected cats with reduced RBC counts at diagnosis had a death ratio 3.5 times higher than FeLV-infected cats with normal RBC counts at diagnosis. Thus, blood counts should be evaluated at diagnosis and all follow-up examinations for retrovirus-infected cats.

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Nonazole Wipes for *Malassezia* spp-Associated Dermatitis

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Although likely uncommon, there have been reports of decreased susceptibility to azoles,[1-3] creating a need for additional therapeutic options for *Malassezia* spp-associated dermatitis.

In the Literature

Sjöström Y, Mellor P, Bergvall K. A novel non-azole topical treatment reduces *Malassezia* numbers and associated dermatitis: a short term prospective, randomized, blinded and placebo-controlled trial in naturally infected dogs. *Vet Dermatol*. 2018;29(1):14-e7.

FROM THE PAGE ...

Malassezia spp-associated dermatitis (MAD) is a common skin condition in dogs that often contributes to the exacerbation of atopic dermatitis. Treatment with an azole is generally the standard of care. Although likely uncommon, there have been reports of decreased susceptibility to azoles,[1-3] creating a need for additional therapeutic options for MAD.

This study* assessed a commercially available nonazole solution. Eighteen dogs with MAD on at least 2 paws were recruited. Each dog was its own control. In a blinded fashion, the test solution was applied daily to one affected paw, and placebo was applied daily to the other. The dogs were rechecked 2 weeks after enrollment. *Malassezia* spp numbers were compared between the initial visit and the recheck. The owners also reported pruritus levels for each paw both before and after the trial.

There was significant reduction in *Malassezia* spp numbers in the test-solution group as compared with the placebo group. Owners reported improvement in pruritus of both the placebo-treated paw and the test-solution–treated paw. No significant difference in pruritus levels was observed between the 2 groups at the end of the study.

… TO YOUR PATIENTS

Key pearls to put into practice:

1 The nonazole wipes used in this study show promise as an additional tool to treat *Malassezia* spp overgrowth on canine paws; however, this product is not currently available in the United States. Further trials are also needed to compare this product with existing azole products.

2 Although yeast numbers decreased significantly in the actively treated paw, owners reported improved pruritus in both placebo and test-solution–treated paws. One potential explanation is that some of these dogs may have been atopic in addition to having MAD. It is possible that the topical treatments removed pollens and thus improved atopic pododermatitis and improved pruritus regardless of change in *Malassezia* spp numbers. This highlights the value of wiping the paws of any dog with pododermatitis.

3 *Malassezia* spp are part of the normal flora; overgrowth is typically secondary to some primary disease process (eg, allergy, endocrinopathy). Dogs with *Malassezia* spp overgrowth should also be evaluated for primary conditions. Treatment of the primary disease process may prevent secondary yeast overgrowth from developing.

*This study was partially supported by Orion Pharma Animal Health, Sollentuna.

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Research Note: **Personality & Pain Assessment in Dogs**

Pain control is a key element of animal welfare, and behavior scales are often used for quantification. Personality, defined as individual differences in behavior that are stable over time and across contexts, can affect behavior assessment scales. This study investigated whether extraversion and neuroticism (as measured by the Monash Canine Personality Questionnaire-Revised) are associated with differences in behavioral and physiologic responses to pain induced by routine neutering in dogs. Results indicated that more highly extraverted dogs had significantly higher pain scores, whereas neuroticism was not associated with physiologic or behavioral pain responses. Owners' ratings of their dog's pain tolerance were not found to be a reliable predictor of pain response.

Source

Lush J, Ijishi C. A preliminary investigation into personality and pain in dogs. *J Vet Behav*. 2018;24:62-68.

Results indicated that more highly extraverted dogs had significantly higher pain scores, whereas neuroticism was not associated with physiologic or behavioral pain responses.

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Hypoglycemia

Shanna Hillsman, LVMT
M. Katherine Tolbert, DVM, PhD, DACVIM (SAIM)
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- **Epistaxis**
- **Regurgitation**
- **Increased Total Thyroxine**
- **Decreased Total Thyroxine**

Following are differential diagnoses, listed in order of likeliness, for patients presented with hypoglycemia.

- Spurious (eg, storage artifact)
- Neonates (typically <6 weeks of age; normal finding or related to another condition [eg, porto-systemic shunt])
- Toy-breed puppy (can occur in healthy puppies when fasting or if no access to nursing)
- Exogenous insulin overdose
- Hepatic failure
- Portosystemic shunt
- Hypoadrenocorticism
- Sepsis
- Systemic inflammatory response syndrome
- Insulinoma
- Xylitol toxicity
- Leiomyoma/leiomyosarcoma
- Hepatoma
- High-intensity exercise
- Pituitary dwarfism
- Glycogen storage disease
- Renal glucosuria
- Polycythemia
- Oleander toxicity
- Amphetamine-dextroamphetamine toxicity
- *Trypanosoma congolense* infection (only in specific geographic regions)

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TOP 5

TOP 5 CAUSES OF EOSINOPHILIA IN CATS

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Eosinophils are polymorphonuclear, granulocytic leukocytes involved in the initiation and propagation of inflammatory responses that act as modulators of innate and adaptive immunity.[1] Normal eosinophil blood count typically measures between 0 and 1500/µL and may vary based on the instrumentation and methodology used (eg, reference laboratory vs in-house).[2,3] Eosinophilia is a state in which blood eosinophil levels are above the normal reference interval.

Eosinophilia in cats is most often associated with parasitic infestation or hypersensitivity reactions (ie, allergies).[4,5] Tissue sites most commonly affected by hypersensitivity conditions include the skin, lungs, and/or GI tract.[6] Transient epinephrine-induced excitement may lead to mild eosinophilia in conjunction with lymphocytosis and mild neutrophilia. Prolonged survival and increased numbers of eosinophils may also be seen in patients with hypereosinophilic syndrome, some neoplastic conditions, and/or paraneoplastic syndromes.[7-9]

Eosinophils—with or without accompanying peripheral eosinophilia—have 3 primary features, including:

- Role as a vital host defense against helminthic parasitic infections
- Frequent presence in allergic disorders (eg, asthma, atopy, food allergies)
- Nonspecific, destructive, and cytotoxic effects against both the pathogen and the host

TOP 5 CAUSES OF EOSINOPHILIA IN CATS

1. Internal & External Parasitism
2. Feline Allergic Dermatitis
3. Feline Asthma
4. Eosinophilic Gastrointestinal Disease
5. Hypereosinophilic Syndrome, Neoplasia, & Paraneoplastic Syndrome

Eosinophilia in cats is most often associated with parasitic infestation or hypersensitivity reactions (ie, allergies).[4,5]

Following are the author's 5 most common causes of eosinophilia in cats.

1 Internal & External Parasitism

Eosinophilia associated with parasitic infestations in cats includes those caused by GI helminths (eg, *Toxocara cati*, *Toxascaris leonina*, *Strongyloides stercoralis*, *Ancylostoma* spp), filarial helminths (eg, *Dirofilaria immitis*), respiratory helminths (eg, *Aelurostrongylus abstrusus*, *Eucoleus aerophilus*, *Paragonimus kellicotti*), ectoparasites (eg, fleas, ticks, mites, mosquitoes), and, rarely, protozoa (eg, *Giardia duodenalis*, *Coccidia* spp, *Isospora* spp, *Toxoplasma gondii*, *Tritrichomonas foetus*).[10,11]

Internal and external parasites may cause clinical signs ranging from subclinical infections to serious disease. Clinical signs commonly depend on the body system that is parasitized (eg, diarrhea, vomiting, failure to thrive, or weight loss with GI parasites; anemia, lackluster coat, or skin lesions with flea infestation). Clinically significant parasitism can affect any cat but depends on age (with increased prevalence and incidence in kittens), general health and immune function, geographic location, and lifestyle (with increased risk to outdoor cats, feral cats, and cats that hunt).

Eosinophil function contributes to the host's defense against helminth infection.[12] The large size of helminths precludes phagocytosis. Eosinophils adhere to a variety of tissue-invading helminth larvae coated with immunoglobulins, then degranulate and secrete soluble factors, which kill larvae.[12-16] Migrating parasites that have been in prolonged contact with host tissue are more likely to induce peripheral eosinophilia, as are migrating stages of ascarids, hookworms, lungworms, and heartworms.[17-22] Rather than persisting long-term, peripheral eosinophilia may be present for only a few weeks after helminth endoparasite infection or for a few months after heartworm infection.[17,18,22,23] A low intestinal parasite burden may not even induce peripheral eosinophilia.[23]

One report showed no difference in mean eosinophil counts in 62 cats with endoparasitism as compared with 122 cats with a negative fecal examination for endoparasite ova.[4] Although sensitivity of fecal flotation methods can vary approximately 13% to 100% depending on the exact method and combination of methods used (eg, zinc sulfate centrifugation, saturated sodium chloride, spontaneous sedimentation, formol-ether technique) and on presence of particular endoparasites,[24] absence of peripheral eosinophilia does not rule out parasitic infection. However, presence of peripheral eosinophilia in combination with other diagnostic information (eg, fecal ova or helminth larvae noted in fecal analysis, elevated serum liver parameters with liver flukes, respiratory signs with migrating larva or lungworms) is supportive of presence of clinically significant parasitic infection.

2 Feline Allergic Dermatitis

Allergic inflammation is an inappropriate immune response that arises from polarization of T cells toward a Th2 immune-mediated response. Greater expression of Th2 cytokines, along with downregulation of Th1 cytokines, is seen in allergen-challenged patients. Eosinophils are increased in number in both the serum and in skin lesions and contribute to the pathogenesis of feline allergic dermatitis. Pathogenesis involves multifaceted immune dysregulation and skin barrier dysfunction stemming from an increasingly complex interplay of genetic and environmental factors.[25-27] Impaired skin integrity increases patient susceptibility to allergens and pathogens, which may lead to activation of innate and adaptive immune responses.[27]

Hypersensitivity may manifest as reactions to allergens derived from food ingredients, inhalants (eg, atopy), contact material, and/or fleas, lice, mites, ticks, or insect bites (eg, hymenoptera). Regardless of the inciting cause, cats with allergic dermatitis are usually pruritic, and primary skin lesions (eg, erythema, maculae, papules) are frequently present. One or more of the following cutaneous reaction patterns may be observed[28,29]:

- Head, neck, and/or pinnal excoriations
- Self-induced alopecia
- Miliary dermatitis
- Eosinophilic lesions, including eosinophilic plaques, eosinophilic granulomas, and indolent ulcers

Histologic or cytologic examination of pruritus-induced skin lesions and surrounding areas commonly reveals increased numbers of eosinophils and mast cells.[30-32] The degree of tissue eosinophilia can correlate with the severity of skin lesions.[30-32]

Age at onset of allergic dermatitis signs is broad. Atopic cats have been reported to experience onset between 3 months and 12 years of age, with a mean age of approximately 2 to 3 years, and cats with food allergies have been reported to experience onset between 3 months and 11 years of age, with a mean age of 4 to 5 years.[33-35] Various studies have reported that approximately 38% to 46% of allergic cats developed signs before 2 to 3 years of age.[34,36] No particular breed or sex predilection has been firmly established for atopy,[34] although some studies have noted an increase of atopy in purebreed and female cats.[32] Higher risk for food allergies has been noted in Siamese, Siamese crossbreeds, and Birmans.[34] Positive treatment responses to atopic dermatitis therapy have been reported with cyclosporine (100%), systemic glucocorticoids (55%), and allergen-specific immunotherapy (57%), and a partial-to-good response has been reported with antihistamines (67%).[35] Avoidance of allergens is recommended.

Flea infestation, the most common cause of allergic dermatitis in cats, may be accompanied by peripheral eosinophilia in approximately 13% to 20% of allergic cats.[37] Peripheral eosinophilia may be identified in 20% to 50% of cats with food allergies. Concurrent GI signs, including frequent bowel movements, vomiting, diarrhea, and/or flatulence, may be exhibited.[33,34]

3 Feline Asthma

Feline asthma is a result of a reaction to inhaled aeroallergens provoking a type 1 hypersensitivity reaction characterized by eosinophilic airway inflammation and bronchoconstriction.[38] Although their clinical presentations may be similar, chronic bronchitis and asthma have different etiologies. Chronic bronchitis may arise from previous airway insult (eg, smoke, toxins, infections)—leading to permanent lung damage—and is dominated by neutrophilic inflammation of the lower airways accompanied by edema and hypertrophy of the respiratory mucosa and excessive mucus production. Although asthma is dominated by eosinophilic airway inflammation (≥17% eosinophils in bronchial lavage cytology) and bronchoconstriction, excessive mucus production and bronchial wall edema are often present to varying degrees.[39,40]

Hallmark characteristics of asthma include reversible airway inflammation, bronchoconstriction causing obstruction/airflow limitation, and airway hyperresponsiveness.[39]

Long-term asthma can lead to irreversible airway remodeling (including bronchiectasis, fibrosis, and/or emphysema), lung hyperinflation, and airway trapping. Cats with asthma are usually young to middle-aged, and no sex predilection has been identified, although middle-aged (ie, 2-8 years) female cats and some Oriental breeds appear to be overrepresented.[38] Asthma is estimated to affect approximately 1% of the general domestic cat population and possibly as many as 5% of the Oriental cat population.[39,41] Clinical presentation includes various combinations of coughing, expiratory wheezing, tachypnea, exercise intolerance, and respiratory distress characterized by an expiratory respiratory pattern.[41-44]

EE = eosinophilic enteritis
FGESF = feline gastrointestinal eosinophilic sclerosing fibroplasia
IBD = inflammatory bowel disease

Clinical signs of heartworm-associated respiratory disease may appear similar to asthma; therefore, heartworm serum antibody/antigen tests may be helpful in ruling out heartworm infection.[38,39,45,46] However, heartworm tests have poor sensitivity because antibody and antigen blood levels may only be transient and can be easily missed. Appropriate fecal analysis (eg, direct, float, centrifugation, Baermann technique for *Aelurostrongylus abstrusus*) should be performed in cats with lower respiratory disease signs if intestinal parasite larval lung migration is suspected and in cats that live in or have visited lungworm-endemic areas.

The most common radiographic change described in patients with asthma is a bronchial pattern with lung hyperinflation arising from bronchial wall thickening due to peribronchial infiltration. Focal atelectasis (typically affecting the right middle lung lobe) and/or diffuse interstitial patterns may also be seen.[41,42]

The most common treatment for asthma is long-term anti-inflammatory doses of corticosteroids administered orally (eg, prednisolone) or via an inhaler (eg, fluticasone). Bronchodilators (eg, terbutaline) may also be included in therapy. Other drugs such as immunomodulating agents (eg, cyclosporine) may be used, particularly in cats that do not respond well to corticosteroids[23] or that develop diabetes mellitus or heart disease.[47]

Approximately 20% of feline asthma patients have peripheral eosinophilia, which is not correlated with the degree of airway eosinophilia.[38,39,43,48] Hyperproteinemia has been reported in 33% to 50% of feline asthma cases.[38,49,50]

4 Eosinophilic Gastrointestinal Disease

Eosinophilic enteritis (EE) has been reported to be the second most prevalent variant of inflammatory bowel disease (IBD), only surpassed by the lymphocytic-plasmacytic form.[51] Although the etiology is poorly defined, it has been hypothesized that affected cats suffer from immunologic dysregulation triggered by one

or more factors, possibly including food ingredients (eg, food allergies or intolerance), dysbiosis of gut microbiota, or other factors (eg, ingestion of ectoparasites, endoparasites, excessive hair, or plant material).[23] Eosinophilic IBD may also involve the stomach and/or colon.[51] Diagnosis of EE is made via intestinal biopsy. Cats with primary lymphocytic-plasmacytic or lymphocytic IBD may also show subtle eosinophilic infiltrates on histopathology. In contrast, cats with EE have predominant tissue eosinophilia, variable mucosal architectural disturbances (eg, villus atrophy), and increased incidence of total intestinal wall thickening associated primarily with muscularis thickening as compared with cats with lymphocytic or lymphocytic-plasmacytic IBD.[23,52] Diffuse disease is most common, but multisegmental EE has also been reported.[52]

No breed or sex predilections have been reported for EE in cats[53,54]; however, although cats of any age can be affected, the condition may be more common in mature cats (ie, 7-9 years). Clinical signs are similar to those of other forms of chronic gastroenteritis and may include vomiting, small- or large-bowel diarrhea, weight loss, and/or anorexia.[53] Borborygmus, flatulence, abdominal pain, hematochezia, and mucoid stools are reported less commonly. Idiopathic EE may be solely or partially responsive to treatment with hypoallergenic diets and corticosteroids, which may be suggestive of an underlying immune disorder.[52]

Peripheral eosinophilia is not always present with EE but was observed in approximately 43% of cats in one study.[52] If peripheral eosinophilia is associated with GI eosinophilic inflammation, other causes (eg, GI parasites, food-responsive enteropathy, intestinal neoplasia [eg, mast cell tumor, lymphoma], hypoadrenocorticism) should be ruled out.[54] Parasitic infestation or dietary intolerance should be considered if moderate-to-large numbers of eosinophils are noted in intestinal biopsy samples with accompanying mild peripheral eosinophilia.[53]

Feline gastrointestinal eosinophilic sclerosing fibroplasia (FGESF) is a recently recognized nodular, nonneoplastic, densely fibroproliferative, eosinophil- and mast cell-rich inflammatory disease. It is thought to be a variant of EE.[54-56] Its pathogenesis is unknown, but bacteria have been postulated to be an initiating factor due to their presence in 56% to 85% of cases[55,57,58]; however, antibiotic treatment alone is ineffective.[55,57,58] Fungal infection has also been thought to be involved in the pathogenesis in some cases,[59] but in other cases, no bacteria or fungi have been detected.[55,56,60] Several other mechanisms (eg, penetrating wounds from a migrating foreign body, genetic eosinophil dysregulation, FHV-1, food hypersensitivity) have been proposed to be involved in the pathogenesis of FGESF.[55,56] FGESF carries a guarded prognosis if untreated; however, survival times may be good in some cats, with some possibly surviving for years if they receive appropriate treatment, which includes a combination of surgery, antibiotics, and immunomodulatory drugs.[57]

Peripheral eosinophilia occurs more often in cats with FGESF than in cats with EE.[52,55] One study reported that 58% of cats with FGESF had peripheral eosinophilia.[55] If peripheral eosinophilia and an abdominal mass are observed, FGESF should be considered a differential diagnosis,[57] although absence of peripheral eosinophilia alone does not rule out FGESF.[57]

Idiopathic eosinophilic enteritis may be solely or partially responsive to treatment with hypoallergenic diets and corticosteroids, which may be suggestive of an underlying immune disorder.[52]

5 Hypereosinophilic Syndrome, Neoplasia, & Paraneoplastic Syndrome

Hypereosinophilic syndrome is an uncommon systemic disorder in cats characterized by sustained eosinophilia resulting from the overproduction of eosinophils in the bone marrow and infiltration of eosinophils into multiple tissues and organs, often leading to organ damage and failure.[23,61,62]

Eosinophilic leukemia has been reported in cats and may be difficult to distinguish from hypereosinophilic syndrome, as each condition may represent different patterns of a similar neoplastic process.[7,8] Some subtle differences in bone marrow and hematologic assessment have been reported; for example, patients with eosinophilic leukemia have been reported to have a higher myeloid:erythroid ratio in bone marrow and a higher WBC count with increased immature eosinophils. However, absolute peripheral eosinophilia is often higher with hypereosinophilic syndrome, although both conditions are typically associated with severe eosinophilia (ie, 3500-130 000/µL).

Peripheral eosinophilia may also be associated with paraneoplastic syndrome.[9] Paraneoplastic peripheral eosinophilia has been observed with mast cell tumors, intestinal T-cell lymphoma, acute leukemia, and transitional cell carcinoma of the bladder and likely involves production of factors including IL-5, IL-3, and granulocyte-macrophage colony-stimulating factor by these tumors.[9,63-65]

Conclusion

In human medicine, it has been suggested that eosinophils have important regulatory roles in homeostasis and immunity.[66-68] With an increased understanding of basic eosinophil biology, improved targeted therapies in humans—and thus, potentially, in cats—toward eosinophilic diseases may be possible.[68]

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Guidelines for Emergency Patient Referral

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General practitioners generally have a rapport with pet owners and provide important primary care, including preventive medicine, diagnostics, and long-term disease treatment and control, to a variety of patients on a daily basis. However, some patients are presented with or develop conditions beyond the scope of general practice. In these instances, practitioners may refer patients to other hospitals for more sophisticated diagnostics (eg, MRI), for consultation with a boarded specialist, or for 24-hour observation or intensive nursing care. Referral practices depend on a strong relationship with general practitioners to provide continuity of care and pet owner communication and to promote patient health and safety when a patient is transferred from one practice to another. In human medicine, good communication between the referring physician and the receiving physician, as well as between referring physician and the patient, is essential for a safe referral process and prevents poor continuity of care and delayed diagnoses.[1-3] Likewise, in veterinary medicine, good communication between the general practitioner, pet owner, and specialist or referral hospital strengthens the patient care team.

Preparing the Pet Owner for Referral

Strong communication relies on the referring veterinarian as a point of reference for both the referral practice and the pet owner. When a general practitioner recommends referral, he or she should involve the pet owner in the decision: Is the owner willing to seek additional care from another facility? Does the owner have pet insurance, or would additional treatment be cost-prohibitive? Should the facility offer 24-hour care, specialty consultation, diagnostics, or a

combination of these? With agreement from the owner, the referring veterinarian should then contact the referral practice to determine their preferences for the referral process.

At this time, the referral practice can ask for patient signalment and history, request the patient's estimated time of arrival, assess the need for medication (eg, sedation, analgesia) to facilitate transport, and offer a general estimate of initial costs, which may change, depending on additional diagnostic testing after the patient is presented to the referral practice. The referring veterinarian can also inquire whether the referral practice has a transport vehicle for emergency referral cases. The referring veterinarian can use this information to help owners understand the diagnostic services or treatment provided by the referral practice.

▲ **FIGURE** Intravenous catheter placement with informative labeling, including catheter size, date and time placed, and the veterinary team member's initials

Preparing the Patient for Referral

Documentation

The referring practice should maintain careful records of treatments and diagnostics for continuity of care. If an intravenous catheter or a bandage is placed, the date, time, and signature should be included, along with additional relevant information (***Figure***).

A study involving the assessment of a referral letter in a human medicine practice noted that in addition to conveying information about the patient, referral letters also reflect the diagnostic skills, communication skills, and professionalism of the doctor.[3] Documentation should be succinct and detailed. Descriptors of the patient, presentation, physical examination findings, treatments, and diagnostics should be included, as should the date and time, doses, and routes of medications and fluids, when possible. In addition, physical and/or electronic copies of diagnostics (eg, imaging, blood work) should be included.

Medication

After the initial examination is performed by the referring veterinarian, sedation and analgesia are crucial to facilitate comfortable transport of the critical patient. The transportation time between practices will likely determine the dose and route of sedative/analgesic that is administered. Communication with the referral veterinarian should help facilitate an analgesic choice that will keep the patient comfortable without causing hemodynamic or neurologic instability before arrival at the referral practice. Drugs to consider include those that are reversible and those that will last the duration of transportation; when applicable, long-lasting antiepileptics (eg, levetiracetam, phenobarbital) and analgesics (eg, pure μ-opioid agonists) should be considered.

Every medication administered to a patient, particularly for transportation, requires a pre-

scription and associated prescription label. This allows the pet owner to be in possession of the medication (especially controlled substances) while transporting the patient. Depending on local state laws, many hospitals will not receive controlled substances, and prescribed medication for critical patients may not be used at the referral hospital.[4]

Transport

Transport of a critical patient can be difficult, especially when performed by the pet owner. Patients should be stabilized before transport, although the nature of the emergency may preclude complete stabilization. Large, open wounds should be carefully bandaged before transport. Stabilizing patients with possible fractures is ideal but may require instruction from the receiving surgeon, further promoting the importance of communication. Some larger referral hospitals may provide an ambulance service to facilitate critical patient transport. Some ambulance services may provide oxygen administration, patient monitoring, intravenous fluid administration, and safe patient restraint for transport in addition to patient transport.[5]

Using appropriate protective tools for transport (ie, those that do not prevent the patient from breathing, panting, or vomiting) such as Elizabethan collars or basket muzzles can help ensure pet owner and veterinary team safety.

Maintaining patient comfort in the vehicle is essential. Patients should be placed safely in the vehicle with support for their head and neck and any other wounds or fractures. Vehicle temperature is also critical, as patients in shock can be highly susceptible to extreme heat and cold. Respiratory distress can be worsened by heat, so cool air in the vehicle is helpful to promote anxiolysis.

Conclusion

Follow-up communication between the referring veterinarian and the referral practice is paramount to providing both continuity of care and treatment follow-through. Updates on diagnostics, findings, treatments, and patient outcome are all critical for both practices to be aware of the case progression and to facilitate an understanding of the case if the patient is returned to the referring veterinarian for additional care.

TOP 5 ITEMS TO COMMUNICATE WHEN REFERRING CRITICAL PATIENTS

- Patient signalment, presenting complaint, and relevant history
- Initial physical examination findings
- Treatments, such as intravenous catheter placement and fluids and medications given (including times, routes, and dosages)
- Diagnostics (eg, radiographs, FAST examination, blood work)
- Current patient status (eg, "stable," "experiencing respiratory difficulty")

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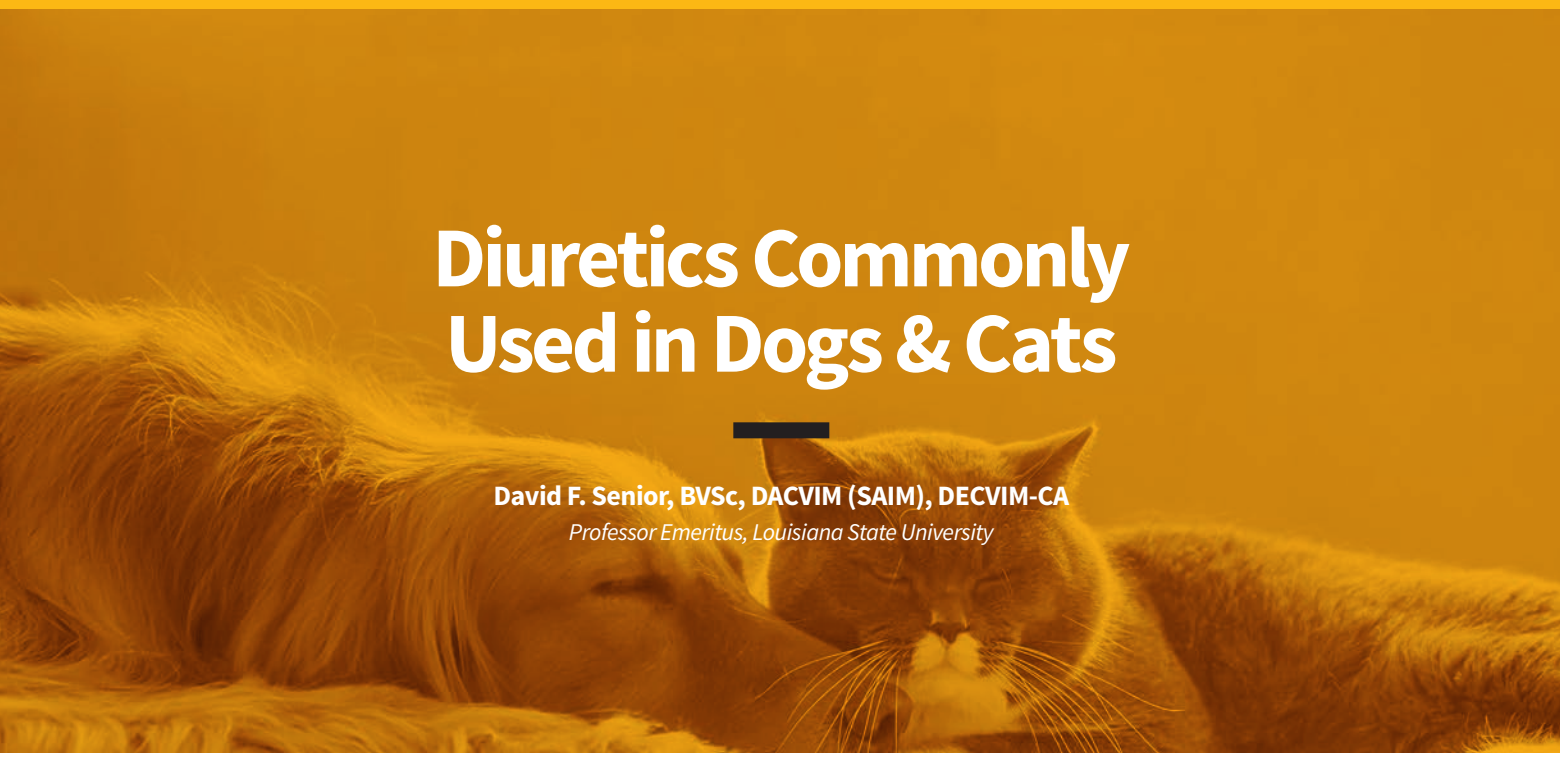

Diuretics Commonly Used in Dogs & Cats

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Diuretics increase tubular fluid flow rate and urine volume primarily by increasing the renal excretion of sodium and attendant anions. These characteristics allow diuretics to be useful in a number of clinical conditions (see ***Common Indications for Treatment with Diuretics***) in which manipulation of renal function and extracellular fluid (ECF) volume and composition are indicated.

CLASSES OF DIURETICS & CLINICAL USE

Loop Diuretics

Loop diuretics, such as furosemide, bumetanide, and torsemide, inhibit the sodium-potassium-chloride (Na-K-Cl) symporter in the luminal membrane of the thick ascending limb of the loop of Henle, which induces the countercurrent multiplier effect. Inhibition of sodium reabsorption induces osmotic diuresis in more distal sections of the tubule, enhancing urinary excretion of sodium, chloride, potassium, calcium, and water.

Furosemide

Formulations → Injectable: 50 mg/mL solution; oral solution (syrup): 10 mg/mL in 60 mL; tablets: 12.5, 50 mg

For pulmonary edema & ascites associated with congestive heart failure (CHF)[1]

Dose (dogs) → Injection: Start with 2 mg/kg q6-8h IV or IM; increase dose in increments of 1 mg/kg to effect (maximum, 5.5 mg/kg q6-8h); oral: up to 6 mg/kg PO q8h

Dose (cats) → Injection: Start with 0.5 mg/kg IV or IM q12h; increase dose in increments of 2.2 mg/kg to effect (maximum, 5 mg/kg q12-24h)

Maintenance dose → Oral: Start with 1 mg/kg PO every 2-3 days up to 2 mg/kg PO q8-12h, depending on response

Key Points

- Conservative doses should be implemented initially, then adjusted to the minimum effective dose based on a target respiration rate.[1]
  - Ideal respiration rate varies based on the individual patient and environmental conditions and is achieved when the patient exhibits a steady, relaxed respiration pattern after stabilization.
- Parenteral furosemide is usually only used for a short period in patients with acute fluid accumulation.

- Single intravenous, subcutaneous, and oral administration may be equally effective to increase urine production in dogs.[2]

▸ Also see ***General Comments***.

For fluid retention associated with glomerular disease[3]

Dose (dogs only) → 1 mg/kg IV or IM q6-12h, with incremental increases of 0.5-1 mg/kg IV or IM q6-12h

Key Points

▸ Use of diuretics for glomerular disease is reserved for patients with severe pulmonary edema or ascites that interferes with major organ function.[3]
▸ Mild peripheral edema is best left untreated in these patients.
▸ In patients with hypoalbuminemia, further ECF reduction with diuretics can cause severe hypovolemia and circulatory collapse.[3]
▸ Parenteral furosemide is usually used for a short period in patients with acute fluid accumulation.[3]
▸ Also see ***General Comments***.

For acute management of moderate-to-severe or rapidly progressing hypercalcemia[4]

Dose (dogs only) → 2-4 mg/kg IV, SC, or PO q8-12h

Key Points

▸ Furosemide is a temporary supportive treatment for short-term control of hypercalcemia until the primary cause can be identified and corrected.[4]
▸ Full hydration should be maintained in patients being treated with furosemide for hypercalcemia.[5]
▸ Furosemide is usually reserved for patients that fail to respond to solute diuresis.
▸ Also see ***General Comments***.

For prevention of cyclophosphamide-induced hemorrhagic cystitis[5]

Dose (dogs only) → 0.5-2.2 mg/kg PO or IV q24h

▸ Shown to be effective in reducing the incidence of sterile hemorrhagic cystitis in dogs currently receiving metronomic low-dose oral cyclophosphamide[6]

General Comments (Furosemide)

▸ Doses should be adjusted based on individual patient response.

COMMON INDICATIONS FOR TREATMENT WITH DIURETICS

▸ **Control of pulmonary edema by mobilization of fluid**: Both loop and thiazide diuretics reduce ECF volume, which in turn reduces intracapillary hydrostatic pressure, a Starling force that contributes to interstitial pulmonary edema.

▸ **Acute kidney injury**: Manipulation of fluid electrolyte and acid-base disturbances in patients with oliguric acute kidney injury is easier if urine output can be increased with administration of mannitol and/or loop diuretics.

▸ **Hypercalcemia**: Loop diuretics can be administered to increase renal calcium excretion, thereby addressing extreme, life-threatening hypercalcemia.

▸ **Control of hypertension**: The reduced ECF volume and reduced plasma volume produced by loop and thiazide diuretics can render them useful as adjunct therapy to control hypertension.

▸ **Control of cerebral edema and glaucoma**: By increasing plasma osmolality, IV boluses of mannitol can increase transfer of fluid from the intracellular fluid space and transcellular fluid space to ECF space, which can rapidly reduce cerebral swelling and intraocular pressure.

▸ **Calcium oxalate urolithiasis**: Because thiazide diuretics can decrease urine calcium excretion, they are indicated in long-term prevention of calcium oxalate urolithiasis.

▸ **Nephrogenic diabetes insipidus**: Loop diuretics combined with a low-sodium diet can reduce ECF volume so a greater proportion of glomerular filtrate is reabsorbed proximally, leaving less to be excreted in the absence of antidiuretic hormone activity, also known as vasopressin.

▸ **Prevention of cyclophosphamide-induced hemorrhagic cystitis**: Concurrent administration of furosemide can reduce the likelihood of cyclophosphamide-associated sterile hemorrhagic cystitis in dogs.

CHF = congestive heart failure
ECF = extracellular fluid

- Renal function and electrolytes, especially potassium, should be monitored to prevent prerenal azotemia.
- *In cats*: Ototoxicity and hearing loss can occur at high doses.
- Furosemide can potentiate the hypotensive effects of angiotensin-converting enzyme (ACE) inhibitors.
- Severe hypokalemia and hyponatremia can develop during treatment, particularly in patients with reduced food and water intake.
- *In cats*: Extreme dehydration and hypokalemia can occur when diuretics are administered long-term; careful monitoring is required.

Bumetanide

Formulations → Oral tablets: 0.5, 1, 2 mg; injection: 0.25 mg/mL

For cardiogenic or pulmonary edema, acute oliguric kidney failure, & moderate-to-severe hypercalcemia[7-9]

Dose (anecdotal; dogs, cats) → Definitive doses have not been published.

- Because bumetanide is 25 to 50 times more potent than furosemide on a mg/mL basis, furosemide doses can be divided by 25 or 50 to arrive at a best-guess dose.[7-9]

Key Points

- There seems to be little reason to use bumetanide instead of furosemide, as their modes of action are identical.
  - Furosemide is approved for use in dogs and cats; doses are anecdotal but well established in small animal medicine.
- Extreme dehydration can occur in cats receiving long-term diuretic therapy.

Torsemide

Formulations → Oral tablets: 5, 10, 20, 100 mg; injection: 10 mg/mL

For pulmonary edema & ascites associated with CHF[10]

Dose (dogs, cats) → 0.2-0.3 mg/kg PO q8-24h

Key Points

- Approximately 10 times more potent than furosemide
- Thought to be indicated in CHF patients that prove refractory to furosemide treatment[10]
- Tablet size can make oral dosing difficult in small dogs and cats; however, compounded 5 mg/mL suspension remains stable for 90 days at room temperature.[9]
- Not as potassium-wasting as furosemide[10]
- Longer duration of action than furosemide; single-daily dosing has proven effective in dogs with degenerative mitral valve disease.[11]
- Significant dehydration can occur when cats receive long-term diuretic therapy.

ACE = angiotensin-converting enzyme
CHF = congestive heart failure

Osmotic Diuretic

Mannitol is a freely filtered, nonabsorbed 6-carbon sugar that promotes osmotic diuresis via retention of sodium and water throughout the nephron, resulting in enhanced excretion of sodium, chloride, potassium, and water.

Mannitol

Formulations → 5%, 10%, 15%, 20%, 25% IV solutions

For oliguric acute kidney injury[12]

Dose (dogs, cats) → 0.25-1 g/kg IV (slow bolus over 10-20 minutes) of 20%-25% solution

- If diuresis is induced within 30 to 60 minutes, continue administering at 60-120 mg/kg/hr IV CRI or 0.25-0.5 g/kg IV q4-6h.
- If diuresis fails to develop within 60 minutes, cautiously administer an additional 0.25-0.5 g/kg IV (slow bolus over 10-20 minutes) of 20%-25% solution.

Key Points

- Should only be administered when patients with acute kidney injury remain oliguric after complete rehydration
- Contraindicated in cases of ethylene glycol poisoning because of the preexisting hyperosmolar state
- Dose can be repeated once if initial administration fails to increase urine output.
  - Further administration of mannitol is contraindicated if diuresis fails to develop.
- *In dogs*: Short-lived impairment of platelet function may develop after mannitol administration.[13]
- Also see ***General Comments***.

For acute glaucoma[14]

Dose (dogs, cats) → 0.5-1 g/kg IV (slow bolus over 10-20 minutes) of 20% solution

Key Points

- Mannitol use is indicated in patients with acute glaucoma that is refractory to topical medication.[14]
- Should be used only after correcting for fluid, electrolyte, and/or acid-base balance and if the patient is not anuric[14]
- Withholding water for 1 to 4 hours after treatment is recommended.
- Effect begins in 20 to 30 minutes and can persist for several hours.[14]
- Also see ***General Comments***.

For increased CSF pressure/cerebral edema[14]

Dose (dogs, cats) → 0.5 g/kg IV (20% solution; slow bolus over 15-20 minutes)

- If required, repeat bolus at 0.5 g/kg IV q6-8h.

Key Points

- Should only be used after correcting for fluid, electrolyte, and/or acid-base balance and if the patient is not anuric[15]
- Contraindicated if ongoing intracranial hemorrhage is suspected
- Effect begins within 30 minutes and can persist for 6 hours.[15]
- Also see ***General Comments***.

General Comments (Mannitol)

- Fluid, electrolyte, and acid-base status must be carefully monitored.[12,14]
- Contraindications include CHF, fluid overload, and pulmonary edema.[12,14]

Thiazide Diuretics

Thiazide diuretics, such as hydrochlorothiazide and chlorothiazide, inhibit the Na-Cl cotransport system in the early distal tubule. The consequences are similar to the action of loop diuretics in terms of increased sodium, chloride, potassium, and water excretion; however, because the effect is more distal, thiazides are not as powerful as loop diuretics and calcium excretion is reduced.

Hydrochlorothiazide

Formulations → Oral tablets: 12.5, 25, 50 mg; oral capsules: 12.5 mg; many combined formulations with other drugs

For prevention of recurrent calcium oxalate urolithiasis[16,17]

Dose (dogs) → 2 mg/kg PO q12h

Dose (cats) → 1 mg/kg PO q12h

Key Points

- Used in conjunction with appropriate diets and medications to reduce calcium excretion and relative supersaturation of urine for calcium oxalate[16,17]
- Usually only added to a preventive regimen if uroliths recur, even with appropriate dietary management and potassium citrate supplementation[16]
- Patients receiving long-term hydrochlorothiazide treatment should be monitored for hypercalcemia.
  - *In cats:* Extreme dehydration can occur when cats receive long-term diuretic therapy.[17]

For pulmonary edema & ascites associated with CHF[18]

Dose (dogs) → 1-4 mg/kg PO q12h

Dose (cats) → 1-2 mg/kg PO q12h

Key Point

- Doses should be started at the lower dose range and gradually increased until desired clinical outcome is achieved.[18]

For ascites secondary to liver disease[19]

Dose (dogs, cats) → 0.5-1 mg/kg PO q12h (in 1:1 combination with spironolactone)

Key Points

- Dose based on spironolactone content of the combined drug[19]
- The combination of spironolactone with hydrochlorothiazide reduces renal potassium loss.[20]
- Doses should be started at the lower dose range and gradually increased until desired clinical outcome is achieved.[19]

For systemic hypertension[21]

Dose (dogs only) → 1 mg/kg PO q12-24h

Key Points

- Used as a second-choice/adjunct agent after ACE inhibitors and calcium-channel–blocking agents have been implemented
- Hydrochlorothiazide combined with spironolactone reduces renal potassium loss.

For nephrogenic diabetes insipidus[22]

Dose (dogs only) → 2 mg/kg PO q12h

Key Point

- Should be combined with a low-sodium diet to reduce urine volume

Chlorothiazide

Formulations → Tablets: 250, 500 mg; oral suspension: 50 mg/mL; lyophilized powder for injection: 500 mg

For diuretic & nephrogenic diabetes insipidus[22]

Dose (dogs, cats) → 20-40 mg/kg PO q12h

Key Point

- Should be combined with a low-sodium diet to reduce urine volume

General Comments (Thiazide Diuretics)

- Doses should be adjusted based on individual patient response.
- Renal function and electrolytes, especially potassium, should be monitored to prevent prerenal azotemia.
- Furosemide can potentiate the hypotensive effects of ACE inhibitors.
- Severe hypokalemia and hyponatremia can develop during treatment, particularly in patients with reduced food and water intake.
- Extreme dehydration and hypokalemia can occur when cats receive long-term diuretics; careful monitoring is required.
  - *In cats*: Ototoxicity and hearing loss can occur at high doses.

Aldosterone Antagonist

The aldosterone antagonist spironolactone inhibits the action of aldosterone on the collecting duct. Aldosterone normally stimulates Na/K-ATPase pumps on the basolateral membrane of the collecting duct cells; without this action, the tendency of potassium to passively diffuse into the tubular lumen is reduced. Thus, spironolactone can be used in combination with either loop or thiazide diuretics to reduce renal potassium excretion.

Spironolactone

Formulations → Tablets: 10, 40, 80 mg; chewable tablets: 10, 50, 100 mg; benazepril–spironolactone tablets: 2.5 mg/20 mg, 5 mg/40 mg, 10 mg/80 mg

ACE = angiotensin-converting enzyme
CHF = congestive heart failure

For adjunct treatment of CHF, ascites, & hypertension[7,19,23,24]

Dose (dogs) → 1-2 mg PO q24h

Dose (anecdotal; cats) → Not well documented but most likely same dose as used for dogs[7]

Key Points

- Reduces potassium wasting caused by loop and thiazide diuretics
- Adjunct treatment only[23,24]
- Additional benefit of adding spironolactone to a heart failure regimen for dogs and cats beyond the potassium-sparing effect is not clear.
- Ineffective as a sole agent to treat hypertension
- Used in combination with furosemide, ACE inhibitors, pimobendan, and amlodipine; spironolactone may increase survival time in dogs with CHF, although the positive effects appear unrelated to any diuretic action.[25]

For fluid accumulation in glomerular disease[7,23,24]

Dose (dogs) → 1-2 mg PO q24h

Dose (anecdotal; cats) → Not well documented but most likely same dose as used for dogs[7]

Key Points

- Reduces potassium wasting caused by loop and thiazide diuretics
- Adjunct treatment only[23,24]

For hypokalemia associated with hyperaldosteronism in cats[26]

Dose (cats only) → 1-2 mg/kg PO q12h

Key Points

- Spironolactone should be given in conjunction with oral potassium supplementation to control hypokalemia.
- Treatment usually must be continued for life.
- Concurrent treatment for hypertension with amlodipine or β blockers is usually indicated in patients with this condition.
- Severe facial ulcerative dermatitis can develop in cats treated with spironolactone.[27]

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Suggested Reading

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Performing Canine Episioplasty with CO_2 Laser

By Ray Arza, DVM

Patients with excessive perivulvar skin folds often suffer from chronic perivulvar dermatitis, recurrent urinary tract infections, vaginal fold pyoderma or vaginitis. Retention of heat, moisture from vaginal secretions and urine within the folds creates a favorable environment for bacterial proliferation and inflammation.

Constant friction of the opposing cutaneous surfaces, in combination with persistent dampness and inflammation, suppresses natural skin defense and creates potential for the development of secondary bacterial infections.

Surgical correction of the excessive perivulvar fold (episioplasty or vulvoplasty), which leaves the vulva more freely exposed to air, is an effective treatment. The incidences of UTIs, vaginitis and external irritation are typically greatly reduced after surgery. It should be noted, however, that skin fold pyoderma should be treated prior to episioplasty.

Episioplasty can be performed with a number of surgical modalities, such as a scalpel, electrosurge or laser. This article explores the benefits of episioplasty performed with a flexible hollow waveguide CO_2 laser.

Laser Settings

Outline incision: Repeat Mode M2-2 Continuous Wave (CW) or F1-2 CW with 0.4 mm focal spot size at 6 to 8 watts. This allows marking of the area to be excised before excision actually begins.

Incision/excision: 10-15 watts in the SuperPulse mode with 0.25 or 0.4 mm focal spot size. Objective is to use enough power to accomplish full thickness incision with one pass at scalpel speed.

Procedure

The patient is placed in sternal recumbency (**Figure 1**). After the patient is anesthetized, the surgeon retracts the skin fold and determines the amount of skin to be removed. The planned incision is then outlined with the laser in two arcs to remove all redundant tissue (note the demarcated crescent-shaped skin segment shown in **Figure 2**).

After marking is complete, a unidirectional arc-incision is made along the outline (**Figure 3**). The incision is done at the highest wattage with which the surgeon feels safe and comfortable. The higher power will allow for faster hand speed, which will minimize thermal collateral insult to tissue. It is important to direct laser energy perpendicular to target tissue. Adequate tension is crucial for best results. The second arc-incision is made along the peripheral demarcation of the crescent-shaped cutaneous segment. Once the incision lines are made, dissection of subcutaneous tissues begins. The excessive skin section, together with the subcutaneous tissues, is retracted and undermined with the laser until excision is finished (as shown in **Figure 4**). During resection, all char, if any, is blotted off with saline-soaked gauze.

The surgeon then assesses if an adequate amount of tissue has been excised to remove all redundant tissue. If skin fold persists, more skin is excised along the outer margin.

Wound Closure

First, the surgeon evaluates the surgical site for hemorrhage and makes sure that the wound margins are free of all char; char is wiped clean with sterile saline soaked gauze 4x4s. Subcutaneous tissues are apposed and sutured with simple interrupted or continuous pattern using 3-0 or 4-0 monofilament absorbable sutures in order to eliminate the dead space.

Finally, the cutaneous margins are approximated and closed (see **Figure 5**). I prefer using a subcuticular pattern with 3-0 or 4-0 monofilament absorbable sutures and avoid external sutures for patient comfort.

Post-op Care

Postoperative antibiotics and appropriate pain management. Elizabethan collar if needed (I find that it is usually not necessary, especially when no external sutures are used). Suture removal in 10 to 14 days.

Conclusions

Efficient hemostasis is a great advantage to the surgeon. Hemostasis allows better visualization and less manipulation of tissue. The CO_2 laser seals lymph vessels and nerve endings, significantly reducing postoperative swelling and pain and leaving patients quite comfortable after surgery.

Moreover, CO_2 laser energy reduces bacterial load at the incision site. Predictable, uncomplicated healing with good cosmetic outcome is another benefit of CO_2 laser surgery. In my experience, episioplasty performed with a flexible hollow waveguide CO_2 laser is an effective procedure to treat perivulvar dermatitis associated with excessive skin folds surrounding the vulva.

FIGURE 1. Excessive perivulvar skin fold partially covering vulva.

FIGURE 2. The planned incision is outlined with laser.

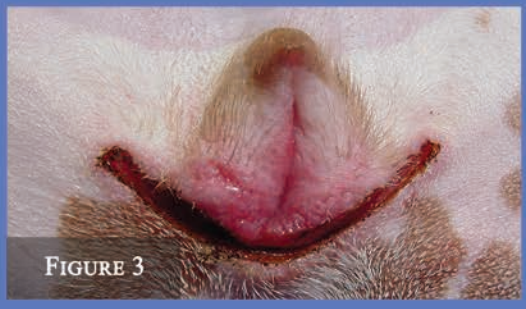

FIGURE 3

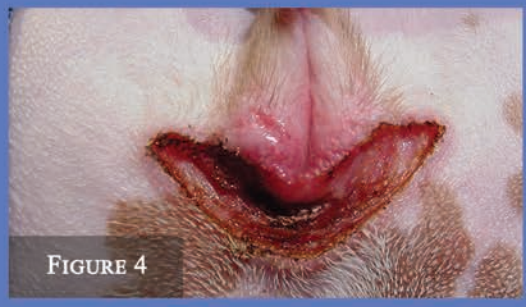

FIGURE 4

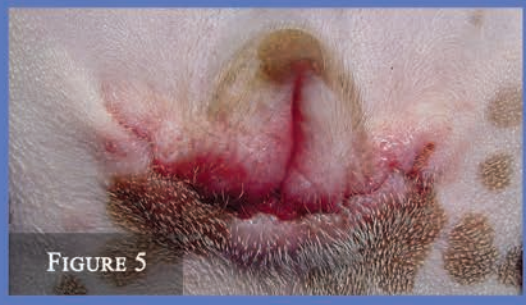

FIGURE 5

FIGURE 3. Laser incision is made along outline at high enough power to incise full thickness with one pass at scalpel speed. This greatly minimizes thermal collateral damage to tissue.

FIGURE 4. Crescent-shaped segment of tissue is excised. Note: Prior to suturing, skin edges need to be thoroughly wiped so that no char is present.

FIGURE 5. Wound closure is complete. Immediately post-op view of the surgical site.

Watch CO_2 Laser Surgery Videos at www.Aesculight.com

About Dr. Arza:

Dr. Ray Arza earned his DVM at the University of Tennessee in 1979. He was a small animal general practitioner for 23 years with a special interest in surgery and dentistry. Dr. Arza started using a surgical laser in 1998, and soon thereafter became a popular lecturer at conferences, universities, and seminars on laser technologies. In 2002, he left private practice to join industry as an educator, trainer, consultant, and lecturer.

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QUIZ CORNER

QUIZ YOURSELF

on this issue's features

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1 **DIAGNOSTIC/MANAGEMENT TREE** PAGE 18

_____ is *not* an appropriate treatment for a cat with constipation.

A. Rehydration
B. Polyethylene glycol 3350 solution
C. Long-term diet change (eg, psyllium-enriched, canned food with water)
D. Sodium phosphate enema

2 **CASE IN POINT** PAGE 29

Which of the following statements regarding splenosis in dogs is *not* true?

A. Splenosis is a rare complication of splenic rupture or trauma.
B. Splenosis has been diagnosed concurrently with a splenic lesion or 2 to 5 years after trauma or splenectomy.
C. Splenosis is a malignant neoplastic disease.
D. Affected patients may be presented with clinical signs or splenosis may be an incidental finding.

3 **THERAPEUTICS SNAPSHOT** PAGE 34

Certain adverse effects make phenylpropanolamine a less appropriate choice for patients at risk for _____.

A. Hepatic disease
B. Seizures
C. Renal disease
D. Hypertension

4 **TOP 5** PAGE 54

Eosinophilia refers to the state in which blood eosinophil levels are above the normal reference interval, which is typically _____.

A. 0 to 250/µL
B. 0 to 500/µL
C. 0 to 1000/µL
D. 0 to 1500/µL

5 **CONSULT THE EXPERTS** PAGE 63

Which of the following statements regarding emergency patient referral is *not* correct?

A. Sedation and analgesia are critical to facilitate comfortable transport of the patient.
B. The pet owner should be in possession of any dispensed medication from the referring hospital, with the exception of controlled substances.
C. Use of Elizabethan collars and basket muzzles during patient transport can help ensure owner safety.
D. The temperature of the vehicle during transport of the patient is essential for maintaining patient comfort.

6 **Rx SOLUTIONS** PAGE 66

Which class of diuretics inhibits the Na-Cl cotransport system in the early distal tubule of the nephron?

A. Loop diuretics
B. Osmotic diuretics
C. Thiazide diuretics
D. Aldosterone antagonists

1: D 2: C 3: D 4: D 5: B 6: C

POLLING PLACE

WE ASKED ...

Which of the following low-stress options for cats do you use in your practice?

YOU ANSWERED ...

A. Providing cat-exclusive examination rooms..... 6%
B. Using cat-friendly techniques (eg, nonslip table mats, pheromone diffusers, minimal restraint)..... 23%
C. Providing hiding boxes for boarding and hospitalized cats..... 6%
D. More than one of the above..... 57%
E. None of the above..... 8%

THIS MONTH'S QUESTION ...

How do you prepare for and handle emergency presentations? (Check all that apply)

A. Maintain a fully stocked crash cart
B. Provide in-house education on emergency medicine and critical care
C. Have dedicated staff for handling emergencies
D. Keep some appointment slots open to accommodate emergencies
E. Stabilize then refer all emergencies to a local emergency clinic

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