

VIRGINIA COOPERATIVE EXTENSION SERVICE

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VIRGINIA-MARYLAND  
REGIONAL COLLEGE  
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## SUDDEN ACQUIRED RETINAL DEGENERATION SYNDROME (SARDS) IN THE DOG

Within the last decade, a syndrome has been increasingly recognized in dogs which is characterized by sudden and absolute vision loss. This syndrome is distinct from progressive retinal atrophy (PRA) in its acute onset and prevalence in mixed-breed dogs. These animals are usually presented to veterinarians with a history of acute blindness, without evidence of any ocular pain, of a few hours or a few days' duration.

Initial clinical examination findings consist of blindness, dilated unresponsive pupils, and a normal fundus. In contrast to acute glaucoma, which also is characterized by blindness and dilated pupils, there is no corneal edema, episcleral vascular congestion, or physical evidence of pain. A history can often be elicited from the owner of polyuria and polydipsia in the weeks or sometimes months prior to the onset of blindness.

Typically, SARDS patients develop ophthalmoscopically visible evidence of retinal atrophy (increased tapetal reflectivity, retinal vascular attenuation, optic disc pallor) several months after onset of blindness. At this point the fundus examination findings are identical to those seen with PRA, but the two syndromes are distinctly different because of the temporal relationships between onset of symptoms and development of visible fundus abnormalities. In SARDS, as in PRA, the blindness is irreversible.

The differential diagnosis for patients presented with acute onset of blindness and dilated pupils should include SARDS, acute glaucoma, optic neuritis, and PRA with owner failure to notice gradual onset. A thorough ophthalmic examination is used to rule out glaucoma and previously-undetected PRA. Since optic neuritis sometimes does not alter the appearance of the optic disc, final differentiation between optic neuritis and SARDS is possible only with electroretinography. Optic neuritis patients have normal ERGs because the condition does not affect the sensory retina itself, only the axons in the optic nerve. SARDS patients have extinguished or flat electroretinograms. If ERG capabilities are unavailable, a course of steroid treatment might be considered as a therapeutic trial with the steroids aimed at possible optic neuritis.

It is likely that SARDS is not really a "new" disease, but rather one we have recently come to recognize. In the past, these cases were probably diagnosed as retrobulbar optic neuritis which failed to respond to steroid therapy.

The cause of this puzzling syndrome has not been determined. Acute death of the photoreceptors followed by gradual loss of the remaining retinal layers is probably secondary to some acute metabolic disturbance but the nature and etiology of this disturbance remains undefined. While SARDS is the most popular name for this syndrome among ophthalmologists, it may also be called "silent retina syndrome" or "toxic metabolic retinopathy."--**Kay Schwink, DVM, Virginia-Maryland Regional College of Veterinary Medicine, Blacksburg, VA.**

### THOUGHT FOR THE MONTH

He who laughs, lasts.

### URETHROTOMY; TO SUTURE OR NOT?

Partial or complete urinary obstruction may occur in the male dog when calculi lodge in the urethra. The most common location of obstructing urethral calculi is at the base of the os penis. In many cases, the calculi can be treated by retrograde flushing (hydropropulsion) followed by cystotomy for calculi removal. When retrograde flushing is unsuccessful, urethrotomy should be performed for calculi removal. Many surgeons have recommended second intention healing of the urethra following urethrotomy at the base of the os penis. Other surgeons feel that suture closure (first intention healing) of the urethra gives the best clinical results.

The surgical approach and procedure are identical whether sutures are used or not. After clipping and preparing the ventral abdomen and prepuce for aseptic surgery, a sterile polyvinyl urinary catheter is lubricated and inserted into the penile urethra to the level of the obstruction. A skin incision is begun at the base of the os penis and extended caudally 4 to 5 cm. The subcutaneous tissue is incised and the retractor penis muscle identified, mobilized, and retracted to one side. The penis is grasped and elevated by the surgeon and the urethral lumen entered using a #15 scalpel blade. Hemorrhage from the incised corpus spongiosum urethra is prominent but minimized by the surgeons elevation of the penis. The initial urethral incision may be extended with fine scissors if necessary. All obstructing calculi are removed, saved for analysis and culture and the urethra flushed copiously with normal saline or lactated ringers.

If second intention healing is elected no sutures are placed in any part of the incision. Antibiotics are administered until stone or urine culture and sensitivity results are known. Long term antibiotic therapy is advised for those patients with bacteruria. The patient will urinate from the urethrotomy site and the penis until healing occurs in about 7-10 days. A major disadvantage of second intention healing is persistent hemorrhage from the incised corpus spongiosum urethra. Hemorrhage usually occurs with exercise or subsequent to urination until the incision heals. Persistent hemorrhage may require continued hospitalization of the patient.

I recommend closure of the surgical incision if the urethral mucosa is healthy and the patient's overall condition allows the added operating time (20 minutes). The urethral mucosa is closed with 4-0 or 5-0 Vicryl® or PDS.® on a tapered needle. Several sutures in the tunic albuginea will help compress the corpus spongiosum and decrease hemorrhage postoperatively. An intradermal and/or skin suture complete the closure. The absence of postoperative hemorrhage will allow early hospital discharge.

In summary, both first and second intention healing of urethrotomy incisions will usually produce a satisfactory outcome. Healing by second intention has the advantage of decreased operative time and the disadvantage of persistent hemorrhage. Precise suture closure of the incision will decrease hemorrhage and increase operative time and is my method of choice if the patients' physical status permits the increased anesthetic time.--Don R. Waldron, DVM, Diplomate ABVP, ACVS, Virginia-Maryland Regional College of Veterinary Medicine, Blacksburg, VA.

## LESIONS ASSOCIATED WITH NON STEROIDAL ANTI-INFLAMMATORY DRUGS

Non steroidal anti-inflammatory drugs are widely used in veterinary medicine today. As with many therapeutic agents, these drugs are not without their side effects. While some non steroidal anti-inflammatory drug associated lesions are incidental findings at necropsy, others may be the cause of the animal's illness or death.

The most well known lesion associated with these drugs is necrosis of the renal crest (sometimes referred to as renal papillary necrosis). This, usually incidental lesion, is most frequently seen in horses, probably because horses are most often given these agents. Many animals, including dogs, llamas, and other zoo animals, can have this lesion. It appears as a discrete focus of dull yellow green discoloration at the inner most aspect of the renal medulla. It can irregularly extend to involve more of the medulla in some cases. The affected tissue is actually necrotic renal parenchyma. In time this tissue may slough and uneventfully pass through the ureter, bladder, and urethra. In the extremely rare cases obstruction of the urinary tract may result.

In foals, gastric and duodenal ulcers are the most common non steroidal anti-inflammatory drug associated lesion. Ulcers are found in the glandular stomach, the pylorus, the proximal duodenum. In some cases, ulcers perforate and fatal peritonitis results. In other cases the mucosa is severely irritated. In foals killed at this stage because of clinical signs of gastritis, the mucosa appears as if it had been "burned" by a caustic agent. Some affected foals survive past this stage. In these foals, healing of the duodenal lesion by scarring causes duodenal stenosis. A sequella of this is gastric rupture with peritonitis.

A third non steroidal anti-inflammatory drug associated lesion is seen in the equine colon, especially the right dorsal colon. Affected horses are usually killed because of intractable diarrhea. The right dorsal colon appears granular, rough, and green, brown to red. Transverse, linear ulcers may also be present in some cases. Histologically there is evidence of colitis. Intestinal contents are watery, but non fetid.

While non steroidal anti-inflammatory drugs have a useful role in veterinary medicine, we should use them judiciously and be cognizant of the possible side effects.--Lois Roth, DVM, PhD, Diplomate ACVP, Virginia-Maryland Regional College of Veterinary Medicine, Blacksburg, VA.

## CANINE DISTEMPER VACCINE FAILURE

A number of factors may contribute to canine distemper vaccine failures by having a negative effect on a dog's immunocompetence.

1. High levels of maternally-derived antibodies is the most important cause of vaccine failure. Maternal antibody levels greater than or equal to 1:20 (virus neutralizing titer) protect against distemper and neutralize vaccine virus. Only 50% of puppies can respond to distemper vaccination by 6 weeks of age. Approximately 90% will develop active immunity at 12 weeks of age.

2. Inherited immunodeficiency syndromes account for some distemper vaccine failures.
3. Canine parvovirus is immunosuppressive. Vaccine-induced distemper has been reported in pups simultaneously vaccinated for distemper and infected with canine parvovirus. It has been suggested that live parvovirus vaccine in combination with distemper should be avoided.
4. Certain parasitic infections such as demodectic mange may be immunosuppressive.
5. Pups with elevated body temperatures do not develop a normal immune response to distemper vaccination. Do not vaccinate pups that are febrile.
6. Broad spectrum antibiotics such as tetracycline or chloramphenicol should be avoided at the time of vaccination.
7. Vitamin E and selenium deficiencies are immunosuppressive in dogs.
8. Stress induced by overcrowding, transportation, environmental changes and rapid fluctuation of ambient temperature or relative humidity may inhibit the immune response.

--Iowa State University's Veterinary Extension Communications #320, February 1987, as printed in the Pennsylvania State University's Veterinary News, July 1987 and Veterinary Professional Topics, Vol. 13, No. 2, 1988, University of Illinois.

#### CHLORPYRIFOS TOXICOSIS IN CATS

Chlorpyrifos is one of the most widely used organophosphorus (OP) insecticides used today. More commonly recognized by the trade names Dursban® or Lorsban®, it has found widespread use in termite control and in agriculture as a corn rootworm insecticide and cattle parasiticide. Due to chlorpyrifos' excellent efficacy against fleas, it has found wide acceptance and application in flea control management. For this purpose, chlorpyrifos is formulated as a dip, in a polymer-containing animal spray, and in flea collars. Except for flea collars, chlorpyrifos is not approved for use on cats. Cats nevertheless are frequently exposed to significant amounts of chlorpyrifos through the accidental or inappropriate use of these products. The product formulations most commonly associated with feline toxicosis are the dips and products for insect control in the home. During 1987 the Illinois Animal Poison Information Center (IAPIC) received 336 calls concerning chlorpyrifos. Of these calls, 152 (45%) involved cats, with 99 (65%) being assessed as toxicosis or suspected toxicosis.

#### Clinical signs

Signs associated with chlorpyrifos toxicosis are somewhat different from most other OP insecticide toxicoses. In the cat, signs are primarily related to the nervous and gastrointestinal systems, and onset may be delayed for 2-5 days. The predominant neurologic signs are tremors, ataxia, and seizures. The most commonly described gastrointestinal signs include salivation, diarrhea, and vomiting. Nonspecific signs of lethargy and anorexia occur fairly consistently and may persist for 2 to 4 weeks. Pulmonary effects are related to bronchiolar constriction and hypersecretion producing severe dyspnea. This can be life threatening, especially in stressed animals. Pupillary changes with OP toxicosis have no diagnostic value. Of the IAPIC calls, frequency of mydriasis and miosis occurred equally in chlorpyrifos poisoned cats. This illustrates how widely variable this particular clinical sign can be in OP toxicosis, and it should not be relied upon for diagnosis or monitoring the efficacy of treatment.

## Diagnosis

The diagnosis of chlorpyrifos toxicosis is the same as for any OP insecticide. A history of exposure to a sufficient amount of the compound coupled with compatible clinical signs can serve as a basis for a tentative diagnosis of chlorpyrifos toxicosis. This is further supported in the live animal by a marked reduction in blood cholinesterase (ChE) activity. Cat blood ChE is extremely sensitive to inhibition by OP insecticides. Clinically normal cats will frequently have extremely low blood ChE activity after being dipped with a OP insecticide; therefore a low blood ChE activity in a clinically affected cat only verifies exposure and not necessarily toxicosis.

At postmortem examination, brain should be submitted for ChE activity. This is the most definitive diagnostic measure available for OP toxicosis.

## Therapy

Treatment of cats for chlorpyrifos toxicosis can be demanding and time consuming. Animals are frequently not presented for treatment until 2-5 days after exposure. Redistribution of chlorpyrifos to adipose tissue, particularly from topical exposure, may create a tissue depot that could slowly release unmetabolized insecticide, resulting in continued exposure. Treatment may need to be continued for days to weeks, even when it is initiated within a few hours after chlorpyrifos exposure.

Specific treatment recommendations are summarized as:

1. Bathe animal with a mild detergent and water if topically exposed.
2. Superactivated charcoal (Super-Char Vet®, Gulf Biosystems, Inc., Dallas, TX; 0.5-1 gm/kg) with a saline or osmotic cathartic whether the insecticide is ingested or there is topical exposure. Repeat treatment at half dosage every 12 hours for 2-3 days.
3. Atropine sulfate (0.1-0.2 mg/kg) as needed. This is especially important to alleviate respiratory distress. These doses are guidelines and the amount required may vary with the clinical response of the animal. The initial dose should be divided and 1/4 given IV with the remaining given IM or SC. Atropine should be readministered as needed; however, it should not be given in excess. Possible detrimental sequelae associated with over-atropinization include: gut stasis, tachycardia, delirium, and hyperthermia. Atropine will not relieve nicotinic signs.
4. Pralidoxime Chloride, 2-PAM® (Ayerst Laboratories, Inc., New York, NY; 20 mg/kg, IM b.i.d.) should be administered to relieve nicotinic signs such as tremors. Many OP insecticides, including chlorpyrifos, have a chemical structure that allows them to "age" once they are bound to the cholinesterase enzyme receptor. "Aging" renders enzyme reactivators such as 2-PAM ineffective. In spite of this potential complication, 2-PAM therapy is still indicated, even if clinical signs have been present for an extended period of time. Continue 2-PAM therapy until the animal is asymptomatic or if no improvement is seen after 24 to 36 hours of treatment.

5. Supportive care is extremely important in affected animals. Seizures in cats should be controlled with diazepam (Valium®), 0.75 mg/kg, IV, as needed. A barbiturate such as phenobarbital (6 mg/kg, IV, as needed) may be necessary. Cats should also be monitored for hypo- and hyperthermia and treated accordingly. Affected animals will frequently have extended anorexia and depression requiring parenteral fluid, electrolyte, and nutritional support. Maintaining hydration and tube or hand feeding enhances recovery.

--by James D. Fikes, DVM, III. An. Pois. Info. Center, as printed in NAPINet Report, May 16, 1988.

VIRGINIA-MARYLAND REGIONAL COLLEGE OF VETERINARY MEDICINE  
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<u>Date</u>	<u>Program</u>	<u>Location</u>	<u>Contact Hours</u>
*Nov. 11-12	Small Animal Urogenital Surgery Lecture/Wet Lab	Blacksburg	10
*Dec. 2-3	Critical Care Nutrition for SA Patients Lecture/Wet Lab	Blacksburg	10
Dec. 3-4	The Human/Animal Bond Workshop	Blacksburg (3) Charlottesville (4)	5 5
*Dec. 9-10	Practical Eye/Ear Surgery Lecture/Wet Lab	Blacksburg	10
*Jan. 20-21	Food Animal Computer Workshop	Blacksburg	6-9
*March 17-18	Fracture Repair: Wires & Pins Lecture/Wet Lab	Blacksburg	10
March 23	Small Animal Medicine Update	Charlottesville	4
*April 28-29	Small Animal Endoscopy Lecture/Wet Lab	Blacksburg	8

\*Limited Enrollment

Note: Program brochure are mailed approximately six weeks prior to the course date. For CE information or assistance, please contact:

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