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# Anticholinergic Drugs in Dogs

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**Abstract**
Can I use anticholinergic drugs with dexmedetomidine in dogs?

**Disciplines**
Small or Companion Animal Medicine | Veterinary Physiology | Veterinary Toxicology and Pharmacology

**Comments**
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CLINICAL TRIAL:

# Patients Wanted for Clinical Studies in Immune-Mediated Hemolytic Anemia and Thrombocytopenia

BY **Dana N LeVine, DVM, DACVIM, PhD**
*Assistant Professor, Small Animal Internal Medicine*

Dr. LeVine, board-certified small animal internist, is passionate about all things hematologic. She has two ongoing research studies that focus on immune-mediated blood disorders. Both studies are soliciting canine subjects.

*Dr. LeVine performs a physical exam on a patient enrolled in the study.*

The first study investigates reasons for thrombosis in **immune-mediated hemolytic anemia (IMHA)**. Most dogs that die of IMHA die due to fatal thrombi. Although clinicians do their best to anti-coagulate these patients, the ideal protocol remains unknown, and thrombi still occur. Recently, neutrophils have been shown to release their DNA when activated, forming webs of DNA called neutrophil extracellular traps (NETs) that capture bacteria. However, these NETs are also very procoagulant. Dr. LeVine is studying the role of NETs in IMHA in order to find a novel therapeutic target to prevent IMHA-associated thrombosis. To enroll in this study, owners must sign an informed consent agreeing to allow a 10 ml (2 teaspoon) blood sample and a voided urine sample to be collected. Dogs receive a Coomb's test, paid for by the study, if the diagnosis of IMHA is uncertain.

The second study, funded by the American Kennel Club Canine Health Foundation, focuses on **immune thrombocytopenia (ITP)**. ITP is the most common acquired bleeding disorder in dogs, causing frank and sometimes fatal hemorrhage. The underlying cause of ITP is unknown, and there are no predictors of disease severity, response, or relapse. Despite a general association between thrombocytopenia and bleeding, not all ITP patients bleed. Consequently, veterinarians treat all ITP patients with aggressive, non-specific immunosuppression. High morbidity and mortality result not only from uncontrolled bleeding, but from treatment side-effects, such as fatal opportunistic infections. Veterinarians need more specific and more individualized ITP treatment suited to disease severity and bleeding risk. Dr. LeVine's ITP study aims to develop a diagnostic ITP "profile" of immune markers that illuminates disease pathogenesis. Additionally, since a genetic component is indicated by a breed predilection in Old English sheepdogs and cocker spaniels, the profile will include genetic loci associated with ITP. This disease characterization will allow for novel treatments specifically targeting the underlying immune defect. The study hopes to identify predictors of disease severity, enabling clinicians to identify and treat those patients with significant bleeding risk.

Dr. LeVine is seeking dogs that are thrombocytopenic due to ITP and dogs that are thrombocytopenic for other reasons (DIC, neoplasia, tick-borne disease, etc.) to serve as thrombocytopenic controls. Any dog with a platelet count less than 50,000/µl may qualify.

To enroll in this study, owners must sign an informed consent agreeing to allow a 15 ml (1 tablespoon) blood sample and voided urine and fecal samples to be collected.

**Go to vetmed.iastate.edu/VetPulse to view full list of free tests for dogs enrolled in the ITP study.**

**For more information, or to refer a patient, please contact Dr. LeVine at 515-294-4900 or dnlevine@iastate.edu.**

FEATURE TOPIC:

# Anticholinergic Drugs in Dogs

BY **Bonnie L. Hay Kraus, DVM, DACVS, DACVAA**
*Assistant Professor, Anesthesia*

## Q Can I use anticholinergic drugs with dexmedetomidine in dogs?

A The use of anticholinergic drugs with α2-agonists has been an area of controversy. Some practitioners prefer to administer an anticholinergic to reduce the incidence or magnitude of bradycardia when administering the less α2-specific drug xylazine. However, the newer drug, dexmedetomidine, is a more specific α2-agonist. The primary mechanism for the bradycardia seen in dogs appears to be a baroreceptor reflex due to stimulation of peripheral α2b-receptors, rather than a central decrease in sympathetic outflow as seen with xylazine. Dexmedetomidine causes vasoconstriction, a reflex bradycardia and a decrease in cardiac output presumably due to the increase in systemic vascular resistance. Dogs typically have high normal mean arterial blood pressure (MAP) and a significant bradycardia which are dose dependent. It is tempting to treat this bradycardia with an anticholinergic drug such as atropine or glycopyrrolate.

A recent study evaluated the cardiovascular effects of dogs administered dexmedetomidine with and without atropine in dogs. Dogs received dexmedetomidine 10ug/kg IM alone or with atropine (0.02mg/kg). Heart rate, cardiac output and blood pressure were measured. Dogs receiving dexmedetomidine alone had heart rates significantly lower than baseline and exhibited sinus arrhythmia and second degree atrioventricular block. Atropine-treated dogs had heart rates that returned to baseline; however, MAPs were very high (> 170mmHg) and cardiac output was not improved. These dogs also developed ventricular premature contractions, a sign of decreased myocardial oxygen delivery.

**Recommendation:** Do not use an anticholinergic to prevent or treat bradycardia associated with dexmedetomidine in dogs if MAP is normal to high normal. Anticholinergic use may be considered in individual patients if it is associated with hypotension. Consider reversing with the α2-antagonist atipamazole if bradycardia becomes severe (<30-35bpm). Sudden arousal may occur in anesthetized patients.

**>> Submit a question at: VetPulse@iastate.edu**

*Reference: Congdon JM, Marquez M, Niyom S et al. Evaluation of the sedative and cardiovascular effects of intramuscular administration of dexmedetomidine with and without concurrent atropine administration in dogs. J Am Vet Med Assoc. 2011; 239: 81-89.*