

Atropine response test protocol

Follow the protocol below to perform a canine/feline atropine response test (ART) if requested to do so by IDEXX Telemedicine Consultants after you have submitted an electrocardiogram (ECG) for review.

ART protocol

Inject 0.04 mg/kg atropine (or, if atropine is not available, inject 0.01 mg/kg glycopyrrolate) intravenously and then repeat the ECG after **15 minutes**.

OR

Inject 0.04 mg/kg atropine subcutaneously (or, if atropine is not available, inject 0.02 mg/kg glycopyrrolate intramuscularly or subcutaneously) and then repeat the ECG after **30 minutes**.

IMPORTANT: For optimal results, the ART should be performed on the same day as the original ECG that led to the ART recommendation and should not be performed under sedation. If the ART cannot be performed on the same day, then you should perform another baseline ECG before performing the ART.

About atropine

Atropine is a competitive muscarinic receptor antagonist. It is used routinely in human and veterinary medicine to help distinguish intrinsic nodal disease from vagally mediated bradyarrhythmias. Indications for an ART include inappropriate sinus bradycardia, frequent sinus pauses or instances of sinus arrest, second-degree atrioventricular (AV) block, and (occasionally) first-degree AV block. Most dogs or cats affected by third-degree (i.e., complete) AV block do not respond to atropine and therefore an ART may not be indicated in all cases of complete heart block. Atropine is also used to correct bradycardia during sedation, general anesthesia, or cardiopulmonary arrest.

The prevalent effect of atropine on cardiovascular function is attributable to blockade of the parasympathetic autonomic tone. This results in increased rate of discharge of the sinoatrial (SA) node and increased conduction velocity at the AV node.

A transient, almost paradoxical, occurrence of AV block and consequent ventricular bradycardia is noted relatively frequently (86% of cases in a study on healthy dogs) and usually resolves in less than 5 minutes. This occurs because atropine rapidly increases the rate of discharge of the SA node prior to inducing changes in AV nodal conduction velocity. Occurrence of AV block after administration of atropine is unlikely to compromise a patient hemodynamically.

Contraindications for the use of atropine include narrow angle glaucoma, ileus, urinary obstruction, and preexisting tachydysrhythmias. Side effects may include mydriasis/cycloplegia, slowed gastrointestinal (GI) and urinary tract motility/function, and dry secretion. Atropine, reportedly, is ineffective in puppies before 14 days of age and kittens before 11 days of age.

The recommended dose of atropine for an ART is 0.04 mg/kg. A lower dose (0.02 mg/kg) has been proven ineffective at blocking parasympathetic tone in awake dogs for the purpose of this test. A higher dose of atropine (0.06 mg/kg) is usually unnecessary.

The preferred route of administration is an intravenous injection. This is associated with more rapid and predictable onset and offset of action. Intravenous administration is also associated with faster resolution of atropine-induced AV block in patients in which this occurs.

Interpreting ART results

A convincing appropriate response to atropine, in dogs, usually implies a post-atropine heart rate (HR) \geq 135–140 BPM or at least a 50%–100% increase in HR from baseline. Consistent AV conduction should also be observed.

Response to an ART is not well studied in cats. It is however reasonable to expect onset of sinus rhythm with consistent AV conduction and a heart rate at the upper end of the normal range (normal HR range in cats: 140–240 BPM) or sinus tachycardia (i.e., HR > 240 BPM).

An appropriate response to atropine suggests a vagally-mediated bradyarrhythmia. Differential diagnoses include constitutional high vagal tone (i.e., brachycephalic breed, athletic conditioning) or pathologically elevated vagal tone secondary to concurrent extracardiac disease, more often respiratory, GI, ocular, or neurologic disorders.

An incomplete or absent response to atropine, if technical errors are ruled out, supports the diagnosis of primary or secondary disease of the SA node or AV node. Differential diagnoses of secondary nodal dysfunction may include hypothermia, hyperkalemia, severe hypothyroidism (rarely), exposure to certain toxic substances, or iatrogenic causes (beta blockers, calcium channel blockers, opioids, sedatives, etc.). Primary nodal dysfunction may reflect sick sinus syndrome (prevalent in mature female miniature Schnauzers, Dachshunds, cocker spaniels, West Highland white terriers, and pugs, although male dogs or other breeds can also be affected), persistent atrial standstill (a.k.a., atrioventricular myopathy of springer spaniels), or idiopathic AV block (often noted in cocker spaniels and pugs).

Less common mechanisms of disease include complications of myocarditis, endocarditis, or primary or secondary cardiac neoplasia.

It is important to acknowledge that some affected dogs, specifically in the context of sick sinus syndrome, may retain the ability to respond partially or even completely to atropine in the early stages of disease.

References:

Plumb DC. *Plumb's Veterinary Drug Handbook*: Desk, 9th Edition. April 2018.

Rishniw M, Kittleson MD, Jaffe RS, Kass PH. Characterization of parasympatholytic chronotropic responses following intravenous administration of atropine to clinically normal dogs. *Am J Vet Res*. August 1999;60:1000-1003.

Rishniw M; Tobias, AH; Slinker BK. Characterization of chronotropic and dysrhythmogenic effects of atropine in dogs with bradycardia. *Am J Vet Res*. March 1996;57(3):337-341.