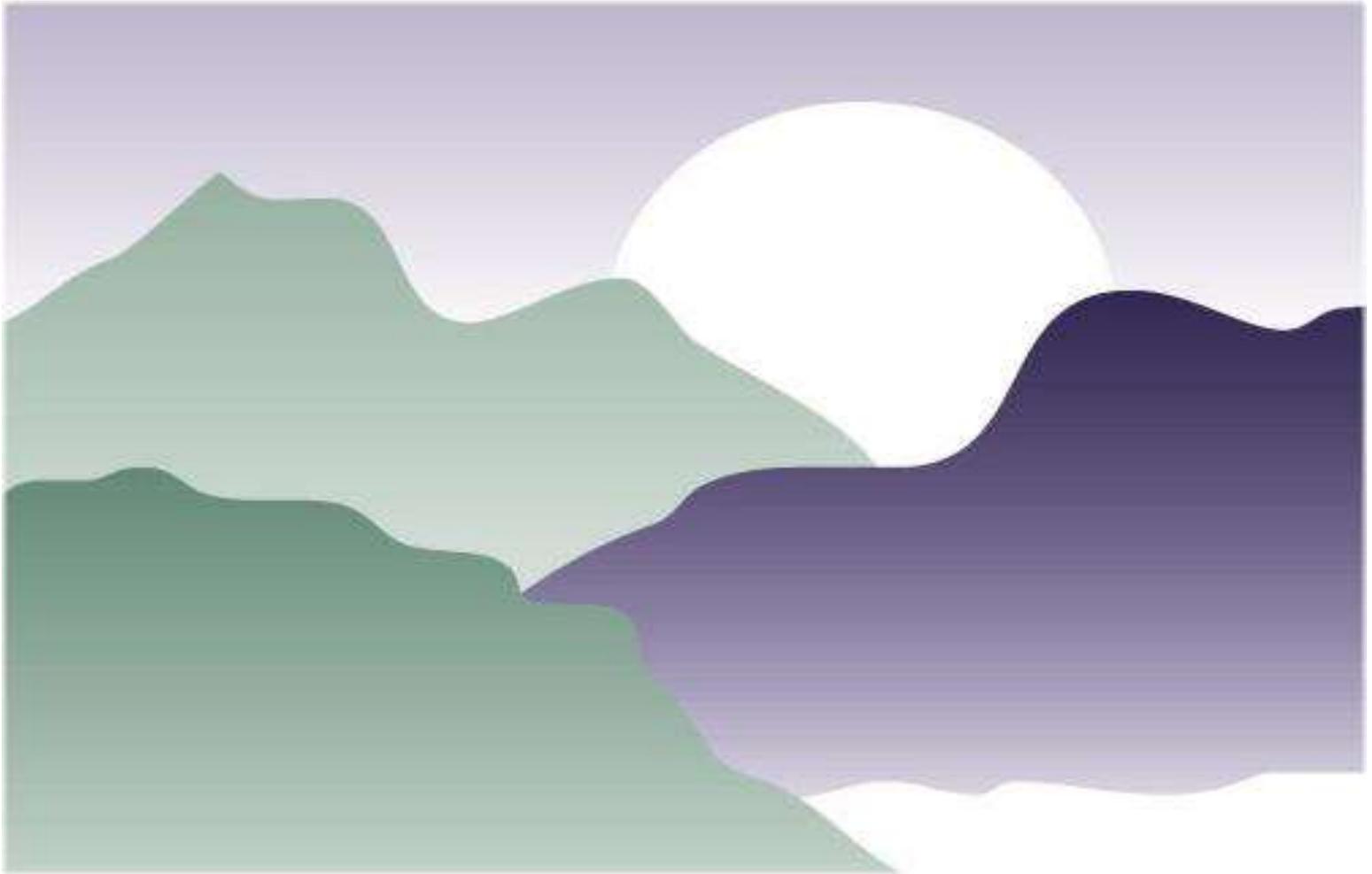


ANTICHOLINERGIC PREMEDICATION

AND THE BEAT GOES ON ...

BY NANCY BROCK, DVM



And the Beat Goes On ...

I remember receiving an urgent phone call one afternoon from a distressed veterinarian who had encountered a problem during a neuter procedure on a young healthy Beagle: the dog's heart rate had dropped precipitously and unexpectedly during surgery.

The doctor was wondering why this had happened and what to do about it as this event had caused quite a scare. He had administered intramuscular (I.M) butorphanol and acepromazine at fairly routine premedication doses followed by intravenous (I.V.) propofol induction, intubation and isoflurane maintenance.

The scary bradycardia had occurred without warning during manipulation of the spermatic cord of first testicle. I asked him why he had not included an anticholinergic in his premedication and he replied that he had recently attended a CE event at which the lecturing anesthesiologist had suggested that routine anticholinergic use wasn't necessary and might even cause harm.

I then asked him if he had administered some atropine I.V. to address this bradycardia and he replied that yes he had, but because he did not have an I.V. catheter in place, the injection had been a bit delayed so that by the time the atropine was given, the bradycardia had already begun to resolve. He also commented that as the patient's heart rate came up, his gradually came down. The dog went on the recover without problems.

Besides being reminded of why an I.V. catheter is such an important component of anesthesia safety (even in young healthy patients) I came away from this phone consultation a bit puzzled: Why and how had anticholinergic premedication developed such a "bad rap"?

Since that conversation, I have received numerous other inquiries on the same subject. So, this topic must be featured at CE events quite frequently with the not-too-surprising result that some confusion exists over the whole issue of whether or not to administer anticholinergic agents prior to anesthesia of dogs and cats. In this article, I would like to clear up some of the confusion.

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When dealing with the question of whether or not to administer an anticholinergic agent prior to anesthesia in your own practice consider the following questions:

1. Is there a risk of vagal stimulation during the planned procedure?
2. Does my patient have a medical condition that would benefit from anticholinergic use during anesthesia?
3. How do I and my staff feel about patients with low heart rates during anesthesia? (Be honest and get input from staff since anesthesia delivery involves team effort).
4. Could the administration of an anticholinergic be detrimental to my patient?

Let's look at each of the questions in turn. Questions 1 and 2 bring up the consideration that anticholinergics are warranted in some predictable situations:

1. Is there a risk of vagal stimulation during the planned procedure?

Expect vagal stimulation to occur whenever a procedure is performed on head or neck structures including the eyes, teeth or throat. Also expect vagal reflex bradycardia whenever traction is exerted on abdominal viscera including ovarian pedicles, spermatic cords, spleens, or loops of bowel.

I am not suggesting that vagal reflexes occur every time these structures are handled. You know that that is not the case. But for patients undergoing planned manipulations of these body parts, anticholinergic protection makes sense.

Remember, however, that if the vagal stimulation is pronounced enough, it can override the effect of the anticholinergic agent. Also, remember that atropine probably only lasts about 45 minutes after I.M. administration.

So, if you are in the habit of allowing a long period of time to go by between premedication and general anesthesia, the atropine may have already worn off. Glycopyrrolate lasts longer than atropine - about 90 minutes.

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2. Does my patient have a medical condition that would benefit from anticholinergic use during anesthesia?

There are indeed some conditions that benefit from the maintenance of a high normal or slightly above normal heart rate. Mitral valve dysfunction is an example of such a condition: When the heart rate is lower than normal, a greater portion of the cardiac output goes backwards through the leaky mitral valve rather than forward. So, using an anticholinergic to prevent bradycardia in such a circumstance may benefit the patient.

3. How do I and my staff feel about patients with low heart rates during anesthesia?

This is where clinicians and nurse anesthetists vary: their comfort zone with low heart rates and the hidden (or not so hidden) fear is that the slowly beating heart will stop completely.

This frightening scenario is unlikely unless a) there is severe hemodynamic instability which is detectable by monitoring of heart rate, blood pressure, and mucous membrane colour or b) a vagal reflex bradycardia occurs (as with our Beagle patient above) and goes undetected.

Bradycardia during anesthesia does not always need to be treated. If there is concurrent hypotension, then correcting the bradycardia may assist with improving blood pressure.

But I would carefully evaluate depth of anesthesia in my patient before simply reaching for atropine. A patient that is concurrently hypotensive and bradycardic is likely too profoundly anesthetized in my opinion.

There are other reasons why bradycardia occurs during anesthesia besides a deep plane of anesthesia:

- Absence of sympathetic stimulation from painful manipulation
- Depression of the sino-atrial node by inhalant anesthetic agents
- Administration of opioids
- Administration of alpha 2 agonists
- Hypothermia

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The only cause of bradycardia on that list that I try to correct is hypothermia which is easier to prevent than treat after it has occurred.

4. Could the administration of an anticholinergic be detrimental to my patient (the flip side of the anticholinergic issue)?

Yes, it could. Our clinical practices have changed over the years: We use a wider selection of anesthesia drugs and drug combinations and we perform more invasive surgical procedures on an older population of patients with a higher incidence of chronic illness. So it is fitting that routine anticholinergic use be re-examined.

Let me give you some examples:

Medetomidine and dexmedetomidine are alpha 2 receptor agonist sedatives that are becoming frequent components of pre-anesthesia medication. These drugs predictably lower heart rates. They also raise blood pressure by causing vasoconstriction.

If we administer an anticholinergic agent to reverse the bradycardia that occurs after their administration, we may stress the heart muscle by speeding it up while forcing it to push blood into very vasoconstricted arteries.

We may feel better about the higher heart rate but we have not done our patient any good. If you plan to use either medetomidine or dexmedetomidine as premedication, either don't administer any anticholinergic and allow the heart rate to drop OR administer an anticholinergic at the same time as the alpha 2 agent.

I omit anticholinergic agents when using alpha 2 premedication. I accept that the heart rate will be low. The bradycardia in itself is not detrimental to the patient's circulation as long as there is no pre-existing heart disease.

One bonus side effect of omitting an anticholinergic is that I can detect when the alpha 2 agent is wearing off because I can identify the gradual increase in the heart rate as the alpha 2 agonist sedation wanes.

Another example of when an anticholinergic might complicate the life of an anesthetist rather than help is during anesthesia delivery to a patient with ventricular premature contractions or supraventricular tachycardia.

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Since anticholinergic drugs facilitate the transmission of electrical signals through the AV node of the heart, the arrhythmia could become more pronounced.

Yet another example of a patient that is not helped by anticholinergic administration is the cat with hypertrophic cardiomyopathy. The nature of this patient's heart disease is such that if the heart rate is high it cannot fill very well as tachycardia reduces the available filling time.

When do I administer anticholinergic agents as part of anesthesia premedication?

I administer glycopyrrolate:

- along with high doses (but not low doses) of opioid premedication
- if the intended procedure might generate a vagal reflex
- if there is a history of sudden bradycardia associated with previous anesthesia delivery
- as premedication whenever ketamine is to be administered for induction of anesthesia - this is to reduce the amount of salivary secretions associated with ketamine administration
- whenever I administer intramuscular alfaxalone to cats - I have witnessed many of these cats developing large amounts of upper airway secretions with slight but observable respiratory embarrassment.

I administer intramuscular atropine peri-operatively:

- if I encounter bradycardia that I perceive to be contributing to hypotension.
- if I detect vagal reflex bradycardia-this reflex is fairly easy to identify: it occurs suddenly with a sometimes dramatic reduction in the heart rate and is usually associated with some form of manipulation by the surgeon such as digging for a tooth root, pulling on an abdominal organ, traction on a spermatic cord, manipulating an eye.

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When dealing with reflex bradycardia, ask the surgeon to cease the manipulation until the anticholinergic protection is on board. This is one instance where the atropine is administered I.V (0.01 mg/kg). Usually, the heart rate will begin to rise within 30 to 60 seconds.

How do I decide between atropine or glycopyrrolate?

Well, these days I use whatever I have in my tool box since glycopyrrolate has been hard to come by as a result of the Sandoz drug shortage. The shortage will be coming to an end soon, likely in February or March.

When I have access to both anticholinergic drugs, I opt for glycopyrrolate if my main goal is reduction in secretions and/or preemptively preventing bradycardia. I reach for atropine when my goal is to elevate the heart rate.

My dose for glycopyrrolate is 0.01 mg/kg usually I.M with a maximum dose of 0.2 mg (1 ml) regardless of patient size. I have watched some of the larger dogs develop very dry mouths with their tongues uncomfortably stuck to their hard palates with higher doses.

My dose for atropine is 0.02 mg/kg IM for premedication, 0.01 mg/kg IV if attempting to correct bradycardia or vagal reflex.

If you have been administering anticholinergic premedication routinely perhaps even premixed with other premedication drugs and have not encountered adverse events, I don't think you need to change what you are doing especially if the majority of your patients are young healthy dogs and cats undergoing elective procedures.

But make sure you are not using the anticholinergic as a replacement for proper patient monitoring because eventually, even with an anticholinergic drug on board, your patients may experience vagal reflex bradycardia and the worst situation would be one in which this reflex is not detected.

Cardiac arrest although rare is not impossible in such a scenario.

If you would like to download a bradycardia troubleshooting flow chart to use during anesthesia delivery visit my website at www.nancybrockvetservices.com.