

Ask the Clinical Instructor

A Q&A column for those new to the cath lab

Questions are answered by:
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“Recently, there was a code in our lab. The anesthesiologist that responded kept checking for pupillary reactions. The cardiologist told him, ‘Never mind, the patient received atropine.’ Why?”

— CIS Online Student, Tennessee.

That would be partly right and partly wrong. To explain why, we need to revisit pharmacokinetics.

Last month, we explored vagal responses. In the article (available online in the December 2008 issue, at <http://cathlabdigest.com/articles/Ask-Clinical-Instructor-A-QA-column-those-new-Cath-Lab-21>), the model of the “gas” and “brake” pedal were discussed. When the GAS pedal is pushed, this is the adrenaline surge of the sympathetic nervous system (SNS). When the BRAKE pedal is pushed, this is the increased tone of the parasympathetic nervous system (PNS). The SNS and the PNS are intended to be in balance, so that all body functions are kept normal.

When the PNS has increased tone, it is because there is a stimulation of the PNS to ‘take over’ the body functions. This stimulates many body systems. Notice in Table 1 that the first letters of each word spell out the mnemonic SHLUDGEE (of course, you can arrange them to any other

word you find easy to remember). As a paramedic, we learned this as a way to remember what happens to the body when there is stimulation of the PNS.

As presented in the December 2008 article, a vasovagal episode occurs when there is stimulation of the PNS. The patient will generally present with decreased heart rate and blood pressure. To counteract these effects, atropine is given.

In the PNS, the chemical mediator of the receptors is acetylcholine (ACh). Because of this, the PNS is often referred to as the ‘cholinergic’ system. When we give atropine due to PNS stimulation, the atropine binds with the receptors that react to ACh to cause the symptoms. This is why you will see atropine called a “cholinergic blocker” or an ‘anticholinergic’ medication. When atropine is given, it binds with those receptors, which will then prevent the PNS stimulation signs and symptoms.

With that occurring, we would



Source: Wes Todd CSA

hope that the SNS would take over (or certainly have more influence) and cause the heart rate to increase, blood pressure to increase and the reversal of the other signs and symptoms we see in the chart.

Side Notes Regarding Atropine:

- Atropine is also carried by our troops as an antidote for some chemical warfare agents, such as sarin, VX and soman.¹ These medications essentially enhance ACh. Massive doses of atropine may be needed to often these actions. Troops will carry autoinjectors to deliver the atropine quickly.

- Atropine can be found in the Belladonna plant. Belladonna gets its name from “bella donna,” which is Italian for “beautiful lady.” During the Italian Renaissance, women used the juices of the plant in their eyes, which would dilate the pupils (this will be handy knowledge later) and, they hoped, would improve their allure to men.²
- Atropine is a core medicine in the World Health Organization’s “Essential Drugs List,” which is a list of minimum medical needs for a basic health care system.³
- Atropine can be used for overdoses of organophosphates (pesticides for house and farms). Again, massive doses (10mg+) are often required for overdose or exposure to these chemicals.
- In the unlikely event of an atropine overdose, or exposure to belladonna or foxglove, the reversal agent is physostigmine.

Now that you have the background, let’s focus on the question at hand.

What happens to the pupils in cardiac arrest? They will likely eventually dilate. While the exact mechanism of this is not fully understood, it is a common assumption that it occurs due



Source: cdc.gov

to hypoxia in the brain. In early cardiac arrest, the pupils will still react to light, assuming that there is adequate CPR and oxygenation. In my experience, at some point the pupils will no longer respond to light. However, this does not appear to directly correlate to survival and hypoxic brain damage. I have participated in numerous resuscitations where the pupils were fixed and dilated, but the patient still survived the event. I am aware of one study in children where the conclusion was “Atropine administration in conventional dose causes slight pupillary dilation but does not abolish pupillary light reactivity.”⁴ Other than this article, literature research on this topic is inconclusive.

What happens to the pupils when atropine is given? First, let’s go back to understanding the difference between SNS and PNS stimulation. In SNS stimulation (or the ‘adrenaline surge’), your pupils will fully dilate. This is part of the “fight or flight” reaction in which our body wants us to have maximum visual accuracy by allowing as much light in as possible. The next time you jump out at someone and scare them, look at their eyes. For those of you who own a cat, you can do this experiment at home. When your cat is in a normal lit room, their pupils are generally in a slit-like fashion. If you begin to play with them with a toy, or my favorite, feet under

Table 1. Autonomic Nervous System (ANS)

	Parasympathetic Nervous System (PNS) Stimulation	Sympathetic Nervous System (SNS) Stimulation
Salivation	+ salivation	- salivation
Heart	- heart rate	+ heart rate
Lacrimation (tears)	+ tears	- tears. Dry eyes
Urination	+ urination	- urination
Defecation	Diarrhea	Constipation
GI Motility	+ peristalsis, cramps	- peristalsis
Emesis	Nausea & vomiting	Intestines relax
Eye	Pupillary contraction	Pupillary dilation



(No, my kitty wasn't all that cooperative... but I think the message is adequately portrayed, as she was playing with a feather. No harm came to the kitty during the production of these photos!)

the blanket (ouch!), their eyes will begin to dilate. They get this from the adrenaline their body is producing while in "stalking" mode.

During PNS stimulation, the pupillary muscle contracts, causing pupillary constriction. When atropine is given, it relaxes this muscle, causing dilation. This isn't much different than dilation of the pupils during an eye exam at the optometrist's office. Your pupils CAN still respond to light, even though it might be more reduced than what it would be in normal circumstances.

In cardiac arrest, when asystole is present, atropine administration is indicated. Generally, a 1mg dose every 5 minutes, up to 3mg, is administered. We would expect the pupils to dilate. However, it would be unknown whether the dilation has occurred due to the atropine, or due to the brain anoxia. To answer the question, in the initial stages of cardiac arrest, particularly after atropine has been administered, dilated pupils cannot be relied upon to provide a sign of potential brain function.

If you have any questions about this article, or you or your lab have a question about a particular topic, please send an email to tginapp@rcisreview.com.

Next month, we will try to make some sense out of the "shellfish vs. iodine" allergy issue.

Available online at <http://www.cleveland-clinicmeded.com/medicalpubs/diseasemanagement/infectiousdisease/chemical/chemical.htm>. Accessed December 8, 2008.

2. New World Encyclopedia: Atropine. Available online at: <http://www.newworldencyclopedia.org/entry/Atropine>. Accessed December 8, 2008.
3. WHO Model List of Essential Medicines (PDF). World Health Organization (March 2005). Available at <http://www.who.int/medicines/publications/essentialmedicines/en/>. Accessed December 8, 2008.
4. Goetting MG, Contreras E. Systemic atropine administration during cardiac arrest does not cause fixed and dilated pupils. *Ann Emerg Med* 1991 Jan;20(1):55-57.

References

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