Anticholinergic: side effects

There are obviously numerous medications which have anticholinergic activity and side effects, even if that is not their primary indication or intent. An incomplete list includes tricyclic antidepressants, antihistamines, antipsychotics, antiepileptics, antispasmodics, ipratropium, and various plants (most classically Nightshade). Given the role of acetylcholine in activating the parasympathetic system, it is not surprising that the "side effects" of an anticholinergic medication manifests similarly to overactivation of the sympathetic nervous system.

The classic phraseology for anticholinergic toxicity is:

"Hot as a hare, red as a beet, dry as a bone, blind as a bat, mad as a hatter, full as a flask"

This represents, respectively: hyperthermia; flushing; dry mouth, eyes and skin; mydriasis; confusion or delirium; and urinary retention. However this is incomplete and you can also see hypertension, seizures, loss of consciousness, QT prolongation, hypotension, and rhabdomyolysis.

While poisoning and toxidromes are within the expected knowledge base of the anesthesiologists, I suspect the ABA is getting at something different with this keyword. The most commonly used anticholinergics by anesthesiologists are probably atropine, glycopyrrolate, scopolamine, and ipratropium. The side effects of the first three, which are systemically administered and absorbed, at clinically used doses include mydriasis, **cycloplegia** (caution in narrow angle glaucoma), reduced gastric and pancreatic secretions, **delayed gastric emptying, reduced lower esophageal sphincter tone (**increased aspiration risk)**, reduced sweat gland activity** (risk for hyperthermia)**, relaxation of bronchial smooth muscle,** reduced mucociliary clearance, **and thickening of bronchial secretions**.

Atropine and scopolamine will cross the blood brain barrier and placenta while glycopyrrolate will not. This can lead to CNS effects appropriately named **central anticholinergic syndrome** which can manifest widely as restlessness, delirium or somnolence. This can be treated with physostigmine which also crosses into the CNS. Scopolamine crosses the blood brain barrier more avidly than atropine and can result in sedation and even amnesia (ask your attending if they've ever done a scopolamine based anesthetic).

Glycopyrrolate does not cross the blood brain barrier thereby avoiding the CNS symptoms, but does have an increased ability to act as an **antisialagogue**. Ipratropium affects bronchial dilation without affecting mucociliary clearance.

Further Reading: Hemmings HC, Egan TD. Pharmacology and Physiology for Anesthesia. 2nd ed: 2019. "Autonomic Nervous System Pharmacology". pp. 294.