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Toxicology in the Emergency Department
A Review for the Advanced Practice Nurse

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Abstract
General assessment, resuscitation strategies, and risk assessment of the poisoned patient are explored in this article, including specific interventions for unresponsive patients and seizures. Sympathomimetic and anticholinergic toxidromes are described in terms of clinical presentation and treatment strategies and are compared with other common toxidromes. Controversies in gastric decontamination are also outlined, including consensus panel and national organizational recommendations. Despite available methods for toxin elimination, advances in medicine, and pharmacotherapy options, the cornerstone of toxicology remains supportive care. The purpose of this article is to equip the advanced practice nurse in the emergency setting with baseline knowledge to provide initial care of the poisoned patient. **Key words:** poisoning, toxicology, toxidromes

THE ADVANCED practice nurse (APN) in emergency care will undoubtedly be faced with the challenge of caring for poisoned patients, be it an accidental exposure or intentional ingestion. The scenarios are familiar: an unresponsive heroin addict dropped in an ambulance bay by his friends, the teenager who was involved in a fight with her parents and ingested a bottle of aspirin, or the patient in fast track seeking treatment for his tooth pain because the "8 Tylenols every hour" he has been taking for the past day are not helping. In 2009, nearly 2.5 million exposures were reported in the United States, with analgesics being the most common (Bronstein et al., 2010). The APN must possess core knowledge of basic toxicological principles to ensure prompt, often lifesaving care even when identification of the poisoning agent is unclear. This goal of this article is to create an awareness of patients with toxicology issues and to provide a brief introduction of the initial management strategies for commonly encountered poisonings. Recent studies and literature available to guide the APN will be reviewed. To illustrate the basic principles of initial toxicologic care, a case study approach will be used.

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CASE STUDY

A 19-year-old woman is brought in by ambulance after her parents found her acting strangely in her room. Her vital signs are

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as follows: heart rate, 124; respiratory rate, 18; blood pressure, 118/82; and temperature, 99.1 °F (37.3 °C). She is mildly agitated and appears to be talking to someone who is not in the room. She is not following commands and is uncooperative. You are her primary provider. Where do you begin?

APPROACH TO THE POISONED PATIENT

The basic approach to the poisoned patient can be divided into six phases: (1) immediate assessment and stabilization, (2) laboratory assessment, (3) decontamination, (4) administration of an antidote, (5) elimination enhancement of the toxin, and (6) disposition (Shannon, 2007). The emphasis of this article is on the first three phases of care. It is paramount to remember that while many patients who present to the emergency department for treatment of intoxications do so with clear complaints of ingestions and/or exposures, a large number of patients experiencing the effects of toxic ingestions do not, or cannot, readily admit to the etiology. Therefore, the APN must maintain a high suspicion for toxicological etiologies in all patients presenting with altered mental status (AMS), neurologic changes, and psychiatric complaints. Each of these patient presentations warrants the same basic approach by the APN.

Initial Assessment

As with all initial assessments, the circulation, airway, and breathing (CAB)—should first be assessed and supported as necessary. Respiratory rates less than 12 per min were shown in one study to be the best predictor of opioid poisoning (Hoffman, Schriger, & Luo, 1991). In addition to pulse oximetry measurements of oxygenation, noninvasive capnography may be used to assess ventilatory effort and monitor for hypoventilation as it becomes more widely available. High-flow oxygen should be administered to patients with suspected poisoning or ingestion who exhibit respiratory compromise or in whom deterioration is anticipated. Rapid sequence intubation may be required in patients with questionable airway protection. Vital signs, including a rectal temperature, intravenous (IV) access, and a 12-lead electrocardiogram (ECG), should initially be obtained.

Besides ischemic changes on the ECG, the APN should pay particular attention to the QRS duration and the QTc (heart rate adjusted) measurements, as an overdose of many medications may cause prolongation of these intervals. The QRS duration should be 0.06–0.10 s, with the QTc less than 0.450 s in adult females and less than 0.430 s in adult males. In pediatric patients between the ages of 1 and 15 years, the QTc should be less than 0.440 s (Goldenberg, Moss, & Zareba, 2006). While slight prolongation of either interval may not warrant immediate intervention (but must be clinically correlated), it is very important to follow the "trend" or measurements over time. Repeat ECGs should be performed at scheduled time intervals of 1–4 hr if there is concern for QRS or QTc prolongation. In addition, three problems common to toxicologic patients may need to be addressed in the initial assessment.

Unresponsiveness

It is absolutely critical to immediately obtain a fingerstick blood glucose level on any patient with an AMS, especially those with seizures and coma. Adult patients who are hypoglycemic with AMS should receive 50 mL of IV 50% dextrose (Hazinski, Samson, & Schexnayder, 2010). A minimum 100 mg dose of IV thiamine should be given concurrently with the glucose if a diagnosis of Wernicke's encephalopathy is suspected or if there is any evidence of alcohol use (Sechi & Serra, 2007; Thomson, Cook, Touquet, & Henry, 2002). Pediatric patients found to be hypoglycemic should also receive dextrose at a dose of 0.5–1 g/kg via IV or intraosseous infusion (Hazinski et al., 2010).

If the fingerstick blood glucose is within normal limits in an unresponsive patient, IV or intraosseous naloxone should be given. The goal of naloxone in the unresponsive patient

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is adequate ventilatory effort and increased arousal to the point of protecting one's airway. In adult patients, the dose is 0.4–2 mg (Erickson, Thompson, & Lu, 2007). Weight-based dosing in pediatric patients is 0.1 mg/kg repeated every 2 min to achieve total narcotic reversal or a maximum dose of 2 mg (Hazinski et al., 2010). If there is a response to naloxone, the APN must be aware that repeat doses may be needed as the offending opiate-based agent may have a half-life that is significantly longer than naloxone. Flumazenil should not be routinely administered as the risks of withdrawal seizures most often outweigh any potential benefits (Erickson et al., 2007; Seger, 2004). Ongoing close monitoring is required in the instances of improvement following glucose or naloxone administration.

Hypothermia and Hyperthermia

A *core* temperature should be initially obtained in any patient with AMS and then monitored on an ongoing basis. While temperature variations can be the direct result of some agents (e.g., salicylates or methamphetamine causing hyperthermia), variations in temperature may also result from environmental exposures postingestion. For example, hypothermia in a heroin overdose patient with a decreased level of consciousness may result from prolonged exposure on a cold basement floor. Dependent upon the patient's temperature, passive or active rewarming or cooling measures may be required.

Seizures

Occasionally, seizure control becomes an emergent intervention in the poisoned patient. While the mechanism of seizure generation in the poisoned patient differs from other etiologies, first-line therapy remains IV benzodiazepines (diazepam or lorazepam) in standard therapeutic dosing. Barbiturates are currently considered second-line therapy. Phenytoin is unlikely to be effective and may in some circumstances be proconvulsive (Anoop & Eddleston, 2010; Kunisaki & Augenstein, 1994; Olson, Kearney, & Dyer, 1994).

Seizures resulting from an isoniazid (INH) overdose represent a caveat within seizure management. With overdoses of INH, toxicity ultimately occurs because of a depletion of pyridoxine (vitamin B_6), which is essential for the formation of γ-aminobutyric acid. The resulting acute γ-aminobutyric acid deficiency lowers the seizure threshold and can cause seizures that are not responsive to standard therapy such as benzodiazepines and barbiturates (Maw & Aitken, 2003). These seizures should instead be treated with pyridoxine in a gram for gram IV dose (Maw & Aitken, 2003; Romero & Kuczler, 1998). For example, if a patient took 3 g of INH, he or she should be given 3 g of pyridoxine. If the amount is unknown, 5 g of pyridoxine are given.

CASE STUDY

Within the patient presentation described earlier, these assessment principles are easily identified. The patient demonstrates a patent airway by talking, despite her confusion and hallucinations. Her vital signs reveal a mild tachycardia (her ECG showed a sinus tachycardia of 126 beats per min, with a QRS of 0.06 s and a QTc of 0.442 s) and mild hyperthermia (temperature, 99.4 °F). Intravenous access is obtained, labs are drawn (specific laboratory investigations are discussed later), 10 mg of diazepam is given intravenously for her agitation, and a 2-L fluid bolus of normal saline is initiated.

Hypotension

Hypotension is not uncommon in poisoned patients, resulting from varied etiologies that include hypovolemia, decreased cardiac contractility, and/or vasodilation. Tachycardia may also be present as a compensatory mechanism for hypovolemia or due to the toxin itself. If the tachycardia is a direct result of toxin exposure, the tachycardia may be rendered nonresponsive to a fluid bolus. Regardless

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of etiology, hypotension should initially be treated with a rapid IV infusion of isotonic crystalloids. In adults, the initial bolus is 1,000–2,000 mL; children should receive 20–40 mL/kg. As with all patients, it is important to consider comorbidities (such as congestive heart failure) before a large fluid bolus. If despite fluid bolus the cardiac output remains low, vasopressors may be required.

Risk Assessment

Second only to immediate stabilization, the APN must consider the risks of the ingestion. Risk assessment involves identification of key pieces of information to predict anticipated outcomes, potential problems, and possible therapies needed based upon the patient presentation. To accomplish this assessment, the APN should gather the following information as possible: What was the ingestion/exposure? When did it occur? What was the dose (or length of exposure)? Was there any treatment initiated before presenting to the emergency department, and if so, what was the specific presentation? Is there a toxidrome present?

TOXIDROMES

Simply put, a toxidrome is a group of signs and symptoms that commonly occur in a poisoning due to a specific toxin. Of all information and examination findings available to the provider, a patient's vital signs, mental status, pupil size, skin, and mucous membranes are the most useful in determination of a specific toxidrome. Typical presentations of commonly encountered toxidromes are summarized in Table 1. In comparing the case study patient with the information in Table 1, it appears that the patient is possibly experiencing a toxidrome, most likely a sympathomimetic or anticholinergic toxidrome.

Sympathomimetic Toxidrome

Exposures to substances that produce pharmacological effects that lead to, or mimic, increased stimulation of alpha and beta-receptors present clinically as a sympathomimetic toxidrome. Simply stated, the signs and symptoms of a patient experiencing this type of toxidrome are all due to hyperstimulation. This manifests clinically as tachycardia, hypertension, tachypnea, and hyperthermia. Agitation (usually with intact, clear speech), diaphoresis, normal to dilated pupils, increased gastrointestinal (GI) motility, and seizures may also be seen. Some common agents causing sympathomimetic toxidromes are listed in Table 2.

Anticholinergic (Antimuscarinic) Toxidrome

This toxidrome occurs when an offending toxin inhibits the central and peripheral acetylcholine muscarinic receptors resulting in a blockade of these receptors. The resulting clinical presentation includes dilated pupils, tachycardia, dry axilla with flushed skin, mumbling (incoherent) speech, delirium, and urinary retention. As with any toxidrome, the severity of the individual signs/symptoms may vary. Medications and toxins commonly associated with this toxidrome are also listed in Table 2. In addition, a classic description used to recall the clinical presentation of an anticholinergic toxidrome is found in Table 3.

Clear presentations of toxidromes, as with other diseases and diagnoses, are not always apparent. Sometimes patients do not fit nicely into one toxidrome. For example, patients who are on chronic beta-blocker therapy may not experience the classical tachycardia of some toxidromes. In cases in which more than one substance was ingested, a mixed toxidrome may occur.

Distinguishing between the similar clinical presentations of anticholinergic and sympathomimetic toxidromes can also be challenging if the ingested agent is unknown. Commonly referred to as the "toxicology handshake," a simple assessment of the patient's axilla (assessing for diaphoresis) may provide a clearer distinction between the two toxidromes. As illustrated in Figure 1, the provider places his or her gloved hands in the patient's

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Table 1. Toxidromes

	Vital signs				Assessment findings			
Toxidrome	**BP**	**HR**	**RR**	**T°**	**Mentation**	**Pupils**	**Skin**	**Other**
Anticholinergic (antimuscarinic)	U/↑	↑	V	↑	Agitated; delirium	Dilated	Dry	Urinary retention; dry mucous membranes
Benzodiazepine/ethanol	↓	↓	↓	U/↓	Depressed; slurred speech; agitated	U–dilated	U	Ataxia; hyporeflexia
Benzodiazepine/ethanol withdrawal	↑	↑	↑	↑	Agitated; delirium	Dilated	Diaphoresis	Tremors; seizures
Cholinergic (muscarinic)	V/↑	↓	V/↑	U	Normal–depressed	V–constricted	Diaphoresis	Excessive secretions; seizures; incontinence; paralysis
Opioid	↓	↓	↓	U/↓	Depressed	Pinpoint	U	Hyporeflexia
Opioid withdrawal	↑[a]	↑	U/↑	U	Normal–anxious	Dilated	Diaphoresis; piloerection	Vomiting; rhinorrhea; diarrhea; yawning
Sympathomimetic	↑	↑	↑	↑	Agitated; clear speech	U–dilated	Diaphoresis	Tremors; hyperreflexia; seizures

Note. BP = blood pressure; ↓ = decreased; HR = heart rate; RR = respiratory rate; T° = temperature; U = unchanged; ↑ = increased; V = variable.
[a]Blood pressure may decrease with opioid withdrawal in the setting of volume depletion from prolonged vomiting and diarrhea.

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Table 2. Examples of offending agents for selected toxidromes

Sympathomimetic toxidrome	Anticholinergic toxidrome
Amphetamines	Antihistamines
Cocaine	Atropine
Methamphetamine	Diphenhydramine
Pseudoephedrine	Jimson weed
Theophylline	Scopolamine
	Tricyclic antidepressants

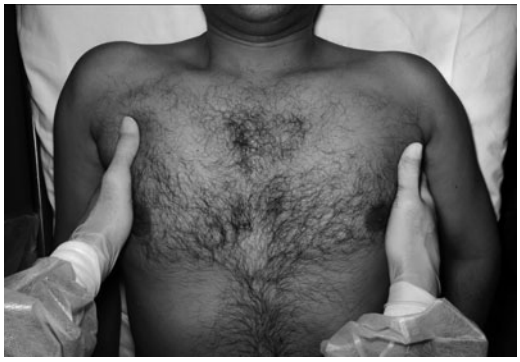

Figure 1. Toxicology handshake. Photograph courtesy of Chip Gresham, MD. Used with permission.

axilla to assess for any significant diaphoresis. Patients experiencing an anticholinergic toxidrome have dry axilla due to the inability to sweat while sympathomimetic poisoning results in significant diaphoresis, or a "wet" axilla.

Laboratory Assessment

The standard array of laboratory testing including arterial blood gases, complete blood cell count, coagulation studies, serum electrolytes and lactate, urinalysis, urine pregnancy, and drug screens should be collected and sent in addition to the blood glucose discussed earlier. Because salicylates and acetaminophen are often contained in combination drugs and/or taken intentionally as a coingestant, these levels must be included on all known or suspected overdose patients. In patients with a history of being found down, combative, rigid, or hyperthermic, a total creatinine kinase must be measured to evaluate the risk for rhabdomyolysis and ensuing acute renal failure.

Although a history of alcohol ingestion is a clear indication for measurement of the patient's blood alcohol level, the APN must not overlook more general indications for blood alcohol level measurement including any patient with AMS. Not only may the alcohol level provide evidence that the patient is indeed intoxicated from ethanol alcohol, testing may yield negative results, which indicate the need for additional differentials and testing. For example, a patient with a Glasgow Coma Scale score of 11 who smells of alcohol may initially be considered to simply be "drunk" until his or her ethanol alcohol level comes back at zero. In such an instance, other causes for his or her low Glasgow Coma Scale must be explored.

Table 3. Classic description for the anticholinergic toxidrome

Description	Sign/symptoms
Red as a beet	Flushed appearance
Dry as bone	Dry axilla and mucus membranes
	No diaphoresis
Hot as a hare	Elevated temperature
Blind as a bat	Dilated pupils/blurred vision
Mad as a hatter	Delirium/agitation

CASE STUDY

To summarize the case study so far, the patient has been stabilized from a CAB perspective. She has become less agitated after diazepam has been given but is still confused and not fully cooperative. Following a one

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liter bolus of normal saline, she remains tachycardic but is otherwise stable. Her physical examination revealed 6 mm pupils bilaterally, with dry mucus membranes and dry axilla. Laboratory studies have been sent. Her family arrived with an empty box of diphenhydramine tablets that they found in her room. They say that there was a web page open on her computer that described how to get high using the medication. The mother states that she believes her daughter obtained the pills from the family medicine cabinet and estimated that it contained about 12 pills. Diphenhydramine hydrochloride (Benadryl) is a first-generation antihistamine primarily used to treat allergic conditions. It is known to cause significant anticholinergic toxicity and is a commonly abused over-the-counter medication. This history from the family is consistent with her physical examination, indicating an anticholinergic toxidrome secondary to diphenhydramine toxicity.

One must remember that anticholinergic toxicity may result in urinary retention; therefore, the placement of an indwelling urinary catheter is a priority. These patients will likely be receiving IV fluid boluses as well and are usually confused and unable to communicate to you that they need to urinate but cannot. Once the patient has been stabilized, the assessment completed, and laboratory studies pending, the elimination of toxins should now be considered.

GASTRIC DECONTAMINATION

Gastric decontamination includes varied methods to prevent absorption of toxic substances into the body. Not all patients suspected of an acute ingestion or poisoning will require or benefit from gastric decontamination; the risks of the substance ingested must be weighed against the risks and benefits of the decontamination method(s). While no clinical trials have shown gastric decontamination to reduce morbidity or mortality, some studies have suggested that patients may benefit if decontamination is performed within 1 hr of ingestion. Gastric decontamination may be accomplished by using one of four primary methods either alone or in combination: (1) inducing emesis using syrup of ipecac, (2) gastric lavage, (3) whole bowel irrigation (WBI), and (4) activated charcoal.

Syrup of Ipecac

Historically, syrup of ipecac was primarily used in the prehospital setting to induce vomiting in children postingestion via direct and indirect stimulation of the brain's vomiting center (Eldridge, Van Eyk, & Kornegay, 2007). Experimental studies have shown that its ability to remove ingested substances is highly variable and is significantly reduced over time. In addition, syrup of ipecac has never been shown to clinically improve the outcome of a poisoned patient and carries the increased risk of aspiration (Position Paper, 2004). For these reasons, the American Academy of Pediatrics, the American Academy of Clinical Toxicology (AACT), and the European Association of Poisons Centres and Clinical Toxicologists (EAPCCT) no longer recommend its use in either prehospital or emergency settings.

Gastric Lavage

The purpose of gastric lavage ("stomach pumping" in layman's terms) is to remove potentially toxic substances from the stomach before they are able to be systemically absorbed. This is done using a large-bore gastric tube (36–40 French for adults, 22–28 French for children) to instill and then remove water or saline and, in theory, any contents residing in the stomach. Experimental studies, which are scarce in the literature, have demonstrated that the amount of a given substance removed by gastric lavage is highly variable and decreases over time. One study noted that 1 hr postingestion, only a 12% decrease in the poison absorption was noted with the use of gastric lavage. Animal studies have demonstrated only an 8%–13% removal of stomach contents when lavage was performed 60 min postingestion (Abdallah & Tye, 1967; Arnold, Hodges, & Barta, 1959).

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Two primary facts regarding the use of gastric lavage serve as the basis for current clinical practice guidelines: (1) the results of clinical outcome studies in overdose patients are weighed heavily on the side of showing a lack of beneficial effect, and (2) serious risks of the procedure include hypoxia, dysrhythmias, laryngospasm, hypothermia, perforation of the GI tract or pharynx, fluid and electrolyte abnormalities, and aspiration pneumonitis (AACT & EAPCCT, 2004a). Compared with other methods of gastric decontamination, lavage is less effective than activated charcoal and roughly equivalent to syrup of ipecac (AACT & EAPCCT, 2004a; AACT & EAPCCT, 2004b; Chyka et al., 2005). Consequently, clinical toxicology organizations such as the AACT and the EAPCCT now recommend that gastric lavage should NOT be employed routinely, *if ever*, in the management of poisoned patients.

Whole Bowel Irrigation

Whole bowel irrigation is a method of gastric decontamination involving enteral administration of an osmotically balanced polyethylene glycol electrolyte solution. In theory, drug absorption and subsequently toxicity are reduced by physically expelling intraluminal contents and decontaminating the entire GI tract. The AACT's and the EAPCCT's position on WBI is that it should not be used routinely in the management of the poisoned patient (AACT & EAPCCT, 2004c). No controlled clinical trials have been performed and there is no conclusive evidence that WBI improves patient outcomes.

Based on volunteer studies, consideration of WBI is appropriate for potentially toxic ingestions of sustained-release or enteric-coated drugs (i.e., sustained-release calcium channel blockers). Patients who have ingested substantial amounts of iron may also benefit from WBI given the high morbidity and lack of other options for GI decontamination as it is not absorbed by activated charcoal (AACT & EAPCCT, 2004c). WBI is absolutely contraindicated in patients with bowel obstruction, perforation, or ileus, and in patients with compromised or unprotected airways or who are hemodynamically unstable.

Activated Charcoal

Benefits from activated charcoal result from its ability to absorb certain ingested toxins in the GI tract, thus decreasing those toxins' possessive systemic absorption. Human volunteer studies are limited but support the consideration of activated charcoal administration in patients with ingestions of a potentially toxic amount of a poison known to be absorbed by charcoal within 1–2 hr (and possibly up to 4 hr in very few select cases) prior (Olson, 2010). Unless a patient has an intact or protected airway, the administration of charcoal is contraindicated. Based upon these data in conjunction with an exhaustive review of the literature, the AACT and the EAPCCT report that there is no evidence demonstrating improved clinical outcomes with the use of single-dose activated charcoal and recommend that it should not be administered *routinely* in the management of poisoned patients though it is justified in select overdoses (Chyka et al., 2005; Olson, 2010).

If administration of activated charcoal is to be considered, then each of the following criteria should be satisfied: the ingestion is a potentially toxic amount of a substance known to be absorbed by charcoal within the past 1 hr, the patients are willing to drink it themselves (they should never be forced), and their airway is intact with no concern of deterioration (i.e., they have not ingested a substance that is going to decrease their level of consciousness over time or a secure, artificial airway is in place). If used, dosing of activated charcoal is weight-based. In children, the dose is 0.5–1 g activated charcoal/kilogram to a maximum dose of 50 g. Typical adult and adolescent dosing ranges from 25 to 100 g (Lapus, 2007). For single-dosing regimens of activated charcoal, sorbitol is not routinely indicated. Multiple-dose activated charcoal is beyond the scope of initial emergency care and this article.

As noted above, there are many contraindications to the few therapies available for GI

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decontamination of the poisoned patient. In fact, of all the strategies discussed in this article, none of them are recommended by the AACT or the EAPCCT for routine use. Table 4 summarizes the indications, contraindications, dosing, and organizational recommendations for these potential strategies.

Disposition

The risk assessment described above, combined with the patient's clinical presentation, may be used to predict need for admission to intensive care. For ingestions involving drugs with prolonged half-lives, symptomatic periods may be longer and thus longer observations periods may be required. Patients suspected of intentional ingestions or exposures require suicide precautions and psychiatric evaluation per facility protocol, often necessitating admission to an intensive care unit (ICU). Additional criteria indicating that a patient may best be managed within an ICU are as follows: Patients with a $Paco_2$ of greater than 45 mmHg, need for emergent intubation, presence of seizures postingestion, unresponsiveness to verbal stimuli, nonsinus cardiac rhythm, second- or third-degree atrioventricular block, systolic blood pressure less than 80 mmHg, and QRS duration of 0.12 s or greater (Brett, Rothschild, Gray, & Perry, 1987).

CONCLUSION

As emergency providers, the goal is to assist our patients. If unable to fix the problem in front of us, we at least want to prevent it from getting worse. We want to "do" something. This creates frustration in caring for many "tox" patients. However as discussed, of the few options we have for these patients, there are many contraindications. In fact, of all the strategies discussed in this article, none of them are recommended by the AACT or the EAPCCT for *routine* use. Table 4 summarizes the indications, contraindications, dosing, and organizational recommendations for these potential strategies. With regard to toxicology, sometimes less is more. At a minimum, we want to "do no harm."

That being said, there are a select group of patients who may benefit from the therapies described above. Determination of who these patients are, however, remains debated in the literature. One clear fact is that each patient must be evaluated individually. This article briefly describes only broad recommendations for gastric decontamination and we encourage you to consult a medical toxicologist for the "not so straight forward patients." They are accessible through state poison centers and can be reached anytime of day or night at 1-800-222-1222. With collaborative management, the APN can ensure that the appropriate interventions are considered to optimize individual patient outcomes.

SUMMARY OF CASE STUDY

The case study presented above represents an anticholinergic toxicity due to diphenhydramine overdose. The patient's agitation was well controlled with ongoing benzodiazepines. Her ECGs (repeated every 4 hr) revealed normal QRS and QTc intervals. A urinary catheter was placed after diazepam was given and her initial urine output was close to a liter and a half. Because of her time of ingestion, AMS and the fact that she was receiving benzodiazepines regularly, the decision was made not to give activated charcoal. Given the evidence in the literature, neither gastric lavage nor WBI was considered. Her laboratory values were within normal limits, including no evidence of acetaminophen and salicylate, except for a mildly elevated creatinine kinase. This was repeated 12 hr later after IV hydration and had returned to normal limits. The local poison control center was contacted early in her evaluation and affirmed the treatment course. She was admitted to the ICU as her delirium was ongoing and requiring intensive nursing care. Eighteen hours later, her mental status was cleared, her heart rate had returned to normal, and she was medically cleared and transferred out to the floor where she was to be seen by a psychiatrist.

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Table 4. Indications and contraindications for gastric decontamination

Method of decontamination	Potential indications	Contraindications	Dosing	Organizational recommendations
Activated charcoal	Potentially toxic amount ingested within past 1 hr Substance known to be absorbed by charcoal Patient willing to drink charcoal Airway intact with no concern of deterioration	Unknown ingested substance or known corrosive ingestion Patient with altered or declining mental status Compromised or questionable airway Patient unwilling to drink the charcoal Bowel obstruction or perforation	0.5–1 g/kg of patient's total body weight, ideally within 1 hr postingestion *Typical adult dose: 25–100 g* *Up to 1 year: 10–25 g* *1–12 years: 25–50 g*	Routine use is not supported by the AACT or the EAPCCT
Gastric lavage	Potentially toxic amount ingested within past 1 hr Substance known to be highly toxic and not well absorbed by charcoal Substances with anticholinergic properties that delay gastric emptying	Ingestion of corrosive agent, low viscosity hydrocarbon Patient with altered or declining mental status Compromised or questionable airway Esophageal or gastric pathology or recent surgery Patient unable or unwilling to cooperate with the procedure	Warm normal saline or tap water lavage of 10 mL/kg (up to 300 mL until the fluid is clear)	Routine use is not supported by the AACT or the EAPCCT; minimal indications for consideration at all

(*continues*)

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Table 4. Indications and contraindications for gastric decontamination (*Continued*)

Method of decontamination	Potential indications	Contraindications	Dosing	Organizational recommendations
Syrup of ipecac	Prehospital setting use only immediately following witnessed ingestion up to 1 hr postingestion	Child younger than 6 months Patient with altered or declining mental status; absence of gag reflex Ingestion of substance with potential for airway compromise within 1 hr Ingestion of corrosive agent, low viscosity hydrocarbon, concentrated acid or bases Patient comorbidities pose risk in setting of repeated vomiting	*Typical adult dose: 30 mL by mouth with 200–300 mL of water* *6–12 months: 5–10 mL by mouth with 120–240 mL of water* *1–12 years: 15 mL by mouth with 120–240 mL of water* *Older than 12 years: 15–30 mL by mouth with 240 mL of water*	AAP against routine use of ipecac in the home; AACT and EAPCCT also against routine use
Whole bowel irrigation	Toxic ingested amount of sustained-release or enteric-coated drugs Substantial iron or drug packet ingestions Substances known to be not well adsorbed to charcoal (e.g., iron, lithium, lead)	Bowel obstruction, perforation, or ileus Significant gastrointestinal bleeding Compromised or unprotected airway Hemodynamic instability	25–40 mL/kg/hr until rectal effluent is clear or other desired effect achieved (e.g., expulsion of drug packets)	Routine use is not supported by the AACT or the EAPCCT

Note: AACT = American Academy of Clinical Toxicology; AAP = American Academy of Pediatrics; EAPCCT = European Association of Poisons Centres and Clinical Toxicologists.

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