Anticholinergics
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KEY POINTS

- Antimuscarinic poisoning syndrome is a more appropriate description than anticholinergic overdose because only the muscarinic receptors, not the nicotinic acetylcholine receptors, are involved. In this chapter the term anticholinergic will be used for consistency and is specifically meant to indicate antimuscarinic.
- Anticholinergic agents antagonize the neurotransmitter acetylcholine at both central and peripheral muscarinic acetylcholine receptors, which leads to altered mental status, mydriasis, tachycardia, urinary retention, ileus, and dry, flushed skin.
- The diagnosis of anticholinergic syndrome is largely clinical and should include physical examination, fingerstick serum glucose measurement, and an electrocardiogram.
- Anticholinergic syndrome is a key clinical finding leading to the diagnosis of poisoning by tricyclic antidepressants (a subset of antimuscarinic agents).
- Basic treatment involves supportive care of the vital signs, activated charcoal, benzodiazepines for agitation, sodium bicarbonate for a QRS complex longer than 100 msec or wide-complex tachycardia, and phystostigmine for consequential central and peripheral anticholinergic (antimuscarinic) manifestations, if appropriate.

EPIDEMIOLOGY

Because anticholinergic toxicologic syndrome (toxicdrome) is common, recognition of its associated signs and symptoms is a necessary clinical skill. It occurs following exposure to many seemingly unrelated agents, many of which are available without prescription or used in patients with a propensity toward self-harm (Box 145.1). For example, according to data from the American Association of Poison Control Centers, 25,788 single exposures to diphenhydramine alone occurred in 2008, with 201 major outcomes and 3 deaths.1

PATHOPHYSIOLOGY AND PHARMACOLOGY

Acetylcholine is the neurotransmitter released from cholinergic nerve endings in the central (brain and spinal cord) and peripheral (autonomic and somatic) nervous systems. In the autonomic (sympathetic and parasympathetic) nervous system, acetylcholine is released from all preganglionic neurons, as well as from postganglionic parasympathetic neurons. Degradation of acetylcholine by the enzyme acetylcholinesterase occurs in the synapse between the presynaptic and postsynaptic membranes.

There are two types of postsynaptic acetylcholine receptors: nicotinic and muscarinic. Nicotinic acetylcholine receptors are ion channels that open in response to stimulation. Found throughout the central nervous system (CNS) (most abundantly in the spinal cord), they are the postsynaptic receptors in the preganglionic sympathetic and parasympathetic neurons. Additionally, these receptors are found in the somatic nervous system at postganglionic skeletal neuromuscular junctions that mediate muscle contraction, as well as in postganglionic neurons of the adrenal medulla, which are subsequently responsible for the release of epinephrine and norepinephrine.

Muscarinic acetylcholine receptors are linked to G proteins to execute their postreceptor effects. They are found primarily in the CNS (most abundantly in the brain). They are also present at effector organs innervated by postganglionic parasympathetic neurons. Stimulation of these end-organs, either pharmacologically or through enhanced neuronal output, results in miosis, lacrimation, salivation, bronchospasm, bronchorrhea, bradycardia, urination, and increased gastrointestinal motility (Table 145.1). Finally, muscarinic receptors are located in sweat glands innervated by postsynaptic sympathetic neurons and cause diaphoresis when stimulated.

Muscarinic acetylcholine receptor antagonists competitively inhibit muscarinic acetylcholine receptors. These agents cause the classic anticholinergic poisoning syndrome, which perhaps may be more appropriately designated the antimuscarinic poisoning syndrome because nicotinic acetylcholine receptors are not affected. Muscarinic receptors in different organs are not equally sensitive to antimuscarinic agents.

Tricyclic antidepressants are a unique subset of antimuscarinic agents that deserve special attention. Their antidepressant effect is achieved pharmacologically through blockade of the reuptake of norepinephrine, dopamine, and serotonin in the CNS. Additionally, tricyclic antidepressants interact with other channels and receptors and cause considerably more profound clinical toxicity in overdose than occurs with most other agents that exhibit anticholinergic effects. Adverse effects of a tricyclic antidepressant overdose include competitive inhibition at both central and peripheral muscarinic acetylcholine receptors (antimuscarinic poisoning syndrome); histamine receptor antagonism (sedation); sodium channel blockade in the myocardium (widening of the QRS complex...
Anticholinergic syndrome can be caused by many agents, including atropine, diphenhydramine, and scopolamine.

**Agents That Produce Anticholinergic (Antimuscarinic) Poisoning Syndrome**

### Plants
- *Atropa belladonna* (deadly nightshade)
- *Datura stramonium* (jimsonweed)
- *Mandragora officinarum* (mandrake)
- *Hyoscyamus niger* (henbane)

### Belladonna Alkaloids (Natural) and Related Synthetic Compounds
- Atropine
- Homatropine
- Scopolamine
- Glycopyrrolate (peripheral effects only)

### Antispasmodics
- Clidinium bromide (Librax)
- Cyclobenzaprine (Flexeril)
- Dicyclomine (Bentyl)
- Propantheline bromide (Pro-Banthine)
- Methantheline bromide (Banthine)
- Orphenadrine (Norflex)
- Flavoxate (Urispas)
- Oxybutynin (Ditropan)

### Antiparkinsonian Medications
- Benztropine mesylate (Cogentin)
- Biperiden (Akineton)
- Trihexyphenidyl (Artane)

### Topical Mydriatics (Ocular)
- Cyclopentolate (Cyclotrol)
- Homatropine (Isopto Homatropine)
- Tropicamide (Mydriacyl)

### Antihistamines
- Brompheniramine (Dimetane)
- Chlorpheniramine (Orade, Chlor-Trimeton)
- Cyclizine (Marezine)
- Dimenhydrinate (Dramamine)
- Diphenhydramine (Benadryl, Caladryl)
- Hydroxyzine (Atarax, Vistaril)
- Meclizine (Antivert)
- Doxylamine (Unisom)
- Promethazine (Phenergan)

### Antipsychotics
- Clozapine (Clozaril)
- Chlorpromazine (Thorazine)
- Prochlorperazine (Compazine)
- Thiothixene (Navane)
- Thioridazine (Mellaril)
- Trifluoperazine (Stelazine)
- Perphenazine (Trilafon)

### Others
- Amantadine (Symmetrel)
- Disopyramide (Norpace)
- Glutethimide (Doriden)
- Procainamide (Pronestyl)
- Quinidine (Quinidex)

### Table 145.1 Pathophysiology of Anticholinergic (Antimuscarinic Poisoning Syndrome) Symptoms

<table>
<thead>
<tr>
<th>ANTICHOLINERGIC EFFECT</th>
<th>SYMPTOMS</th>
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<tbody>
<tr>
<td>Central inhibition of muscarinic acetylcholine receptors</td>
<td>Confusion, disorientation, psychomotor agitation, ataxia, myoclonus, tremor, picking movements, abnormal speech, visual and auditory hallucinations, psychosis, seizures, cardiovascular collapse, coma</td>
</tr>
<tr>
<td>Inhibition of postsynaptic sympathetic muscarinic acetylcholine receptors in the sweat glands, as well as vasodilation of peripheral blood vessels</td>
<td>Dry, flushed skin</td>
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<tr>
<td>Inhibition of postsynaptic parasympathetic muscarinic acetylcholine receptors in the:</td>
<td>Dry mucous membranes</td>
</tr>
<tr>
<td>Salivary glands</td>
<td>Paralysis of the sphincter muscle of the iris and the ciliary muscle of the lens resulting in mydriasis, cycloplegia, and blurred vision</td>
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<tr>
<td>Eye</td>
<td>Tachycardia</td>
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<tr>
<td>Heart (vagus nerve)</td>
<td>Urinary retention and overflow incontinence</td>
</tr>
<tr>
<td>Bladder</td>
<td>Adynamic ileus</td>
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or wide-complex dysrhythmias, atrioventricular block, QT prolongation, and rightward shift of the terminal 40-msec QRS axis on an electrocardiogram [ECG], as well as negative inotropy leading to hypotension; α-adrenergic receptor antagonism on vascular smooth muscle (vasodilation leading to hypotension); and although the mechanism of this effect is unclear, γ-aminobutyric acid (GABA) antagonism (seizures). In the right clinical setting, anticholinergic syndrome is a key clinical finding leading to the diagnosis of tricyclic antidepressant poisoning (Table 145.2).

### PRESENTING SIGNS AND SYMPTOMS

Central inhibition of muscarinic acetylcholine receptors results in confusion, disorientation, psychomotor agitation, ataxia, myoclonus, tremor, picking movements, abnormal speech, visual and auditory hallucinations, psychosis, seizures, cardiovascular collapse, and coma.

Inhibition of postsynaptic sympathetic muscarinic acetylcholine receptors in the sweat glands, as well as vasodilation of peripheral blood vessels, gives rise to dry, flushed skin. Inability to sweat, particularly in the presence of altered CNS regulation, may lead to hyperthermia. Inhibition of these receptors in the salivary glands results in dry mucous membranes, whereas inhibition of these receptors in the eye (which cause pupillary constriction when activated) leads to paralysis of the sphincter muscle of the iris and the ciliary muscle of the lens with subsequent mydriasis, cycloplegia, and blurred vision. Tachycardia is caused by inhibition of postsynaptic parasympathetic muscarinic acetylcholine receptors on the vagus nerve. Dysrhythmias may be caused by antimuscarinic agents that possess additional pharmacologic effects. For example, agents that produce sodium channel blockade in the myocardium (i.e., tricyclic antidepressants, diphenhydramine, pheniramine, orphenadrine, pyrilamine) cause widening of the QRS complex or wide-complex dysrhythmias, atrioventricular block, QT prolongation, and rightward shift of the terminal 40-msec QRS axis on the ECG, as well as negative inotropy leading to hypotension. Inhibition of postsynaptic parasympathetic muscarinic acetylcholine receptors in the bladder results in urinary retention and overflow incontinence, and in the bowel it causes adynamic ileus.

Frequently, patients with anticholinergic (antimuscarinic) poisoning do not have all the previously mentioned characteristics of the classic syndrome, especially the elderly and patients with organic brain syndrome, in whom central anticholinergic (antimuscarinic) poisoning syndrome is often more pronounced than or outlasts the peripheral syndrome.

See Tables 145.1 and 145.2, which detail the pathophysiology associated with the signs and symptoms of antimuscarinic poisoning syndrome and tricyclic antidepressants.

### DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING

**DIAGNOSTIC FEATURES**

Patients who overdose with an anticholinergic (antimuscarinic) agent often have sufficient clinical findings to make the diagnosis apparent. However, the symptoms and signs in others may be less overt, and thus a substantially broader differential diagnosis is required.

Although patients with both adrenergic (sympathomimetic) and anticholinergic (antimuscarinic) poisoning may exhibit confusion, disorientation, psychomotor agitation, seizures, flushed skin, hyperthermia, mydriasis, and tachycardia, the two syndromes may be differentiated through examination of the skin and observation of the symptoms associated with the altered mental status. Patients with anticholinergic poisoning have dry skin and mucous membranes, and the alteration in mental status is characterized by mumbling speech, delirium, and tactile or visual hallucinations. In contrast, patients poisoned by sympathomimetic agents, such as cocaine toxicity, are typically, though not always, diaphoretic with agitated or violent behavior and hallucinations that are more commonly paranoid.

Multiple other toxicologic entities are associated with autonomic dysfunction (i.e., dysregulation of the heart rate, blood pressure, temperature, gastrointestinal secretion, metabolic and endocrine responses to stress). Acute withdrawal syndromes may be differentiated from anticholinergic (antimuscarinic) syndrome by a history of recent cessation of ethanol or another sedative-hypnotic agent, serotonin syndrome may be differentiated by a history of recent (minutes to hours) exposure to a serotonergic agent, and neuroleptic malignant syndrome may be differentiated by a history of exposure (within 3 to 9 days) to an agent capable of producing central dopamine blockade. Drug-induced psychosis mimicking central anticholinergic syndrome may be due to hallucinogens, phencyclidine, amphetamines, or corticosteroids.

<table>
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<tr>
<th>Table 145.2 Pathophysiology of Tricyclic Antidepressants and Associated Symptoms</th>
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<tr>
<td><strong>EFFECT</strong></td>
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<tr>
<td>Blockade of reuptake of norepinephrine, dopamine, and serotonin in the central nervous system</td>
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<tr>
<td>Competitive inhibition at both central and peripheral muscarinic acetylcholine receptors</td>
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<tr>
<td>Histamine receptor antagonism</td>
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<tr>
<td>Sodium channel blockade in the myocardium</td>
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<tr>
<td>α-Adrenergic receptor antagonism on vascular smooth muscle</td>
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<tr>
<td>γ-Aminobutyric acid antagonism</td>
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Medical diseases that produce confusion, seizures, and tachycardia, such as hypoxia, hypoglycemia, and heat stroke, or those that cause hyperthermia, hypertension, tachycardia, and mydriasis, such as thyrotoxicosis and pheochromocytoma, may also be confused with anticholinergic toxicity.

Finally, diseases that may be manifested similar to central anticholinergic syndrome include schizophrenia and other psychotic disorders, cerebral vasculitis, CNS infection (e.g., encephalitis), sepsis, and psychiatric disease.

**DIAGNOSTIC TESTING**

In the setting of anticholinergic (antimuscarinic) poisoning, results of the fingerstick serum glucose test and pulse oximetry analysis should be normal.

The ECG typically demonstrates sinus tachycardia. Some agents with anticholinergic (antimuscarinic) effects also have type IA antidysrhythmic effects that result in blockade of myocardial sodium channels. The blockade is seen on the ECG as prolongation of the QRS interval. Such agents include diphenhydramine, cyclobenzaprine, carbamazepine, and the tricyclic antidepressants.

With tricyclic antidepressant overdose in particular, the extent of prolongation of the QRS interval on an ECG is especially useful in predicting the severity of toxicity.

A QRS duration shorter than 100 msec predicts that no serious clinical toxicity will occur, a QRS duration longer than 100 msec is associated with a 30% incidence of seizures, and a QRS duration longer than 160 msec is associated with a 50% likelihood of the development of ventricular dysrythmia. Additionally, an R wave in lead aVR measuring 3 mm or greater or a terminal 40-msec right axis deviation between 130 and 270 degrees is a predictor of tricyclic antidepressant-induced toxicity.

**FACTS AND FORMULAS**

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S1 = \frac{SV}{BSA}\]

**RED FLAGS**

**Cautions for Physicians**

Physostigmine is contraindicated in patients after a tricyclic antidepressant overdose with a QRS interval longer than 100 msec because it may cause cardiac arrhythmias and profound hypotension.

Flumazenil should not generally be administered to patients with anticholinergic (antimuscarinic) toxicity because it may cause seizures or other complications.

Failure to become cholinergic (bradycardia, bronchorrhea, diaphoretic, drooling) after the administration of physostigmine is essentially diagnostic of anticholinergic toxicity.

Physostigmine will prolong the action of drugs metabolized by cholinesterases, such as succinylcholine. A nondepolarizing agent should be used instead.

Prolonged agitation and seizures can lead to the development of acidosis and rhabdomyolysis. Measurement of serum creatine phosphokinase and urine myoglobin aids in the recognition of patients at risk for the development of acute renal failure.

**TREATMENT**

Most patients with anticholinergic (antimuscarinic) toxicity can be treated adequately with general supportive care of the airway, breathing, and circulation, followed by frequent reassessment and close observation (Fig. 145.1). To avoid the risk for aspiration, activated charcoal (1 g/kg) is recommended only for patients who are capable of spontaneously drinking and protecting their own airway. It may also be administered cautiously via a nasogastric tube to patients who are endotracheally intubated. Because anticholinergic (antimuscarinic) agents may slow gastrointestinal transit, activated charcoal may be useful several hours after ingestion. The specific role of activated charcoal in most of these poisonings has not been studied.

The initial therapy for cardiovascular toxicity secondary to sodium channel blockade is hypertonic sodium bicarbonate in 1- to 2-mEq/kg boluses. Treatment with sodium bicarbonate is indicated for patients with a QRS complex longer than 100 to 120 msec or a wide-complex or ventricular tachycardia until the abnormality is reversed or serum pH reaches 7.55. The ECG should be repeated within 60 seconds after a bolus of sodium bicarbonate to check for narrowing of the QRS complex. If narrowing has occurred, a continuous infusion at 1.5 times the maintenance intravenous fluid rate (three ampules [132 mEq] of sodium bicarbonate in 1 L of 5% dextrose in water [D5W]) should be administered. Profound cardiovascular toxicity may require more aggressive interventions, such as the initiation of vasopressors or use of an intraaortic balloon pump.

Agitation should be addressed aggressively to prevent the development of more serious sequelae, such as hyperthermia, acidosis, and rhabdomyolysis. It is best controlled with benzodiazepines. The emergency physician should start with standard doses (i.e., diazepam, 5 to 10 mg intravenously) and repeat them until sedation (relief of agitation, myoclonus, tremor, picking movements, abnormal speech, and hallucinations) is achieved. Administration of physostigmine should also be considered for the treatment of agitation caused by an anticholinergic agent (see later).

Anticholinergic (antimuscarinic) agent–induced seizures should also be treated with standard doses of benzodiazepines (i.e., lorazepam, 2 mg intravenously) or with physostigmine. If this therapy fails, barbiturates or other GABAgic anticonvulsants should be administered. Phenytoin is rarely useful for toxin-induced seizures.

Patients with rhabdomyolysis should be administered saline intravenously at a rate sufficient to maintain brisk urine output (generally 3 to 5 mL/kg/hr after any lost intravascular volume has been replaced). If urinary pH is less than 6.0, urine alkalinization is necessary and is achieved with the use of a sodium bicarbonate infusion at 1.5 times the maintenance intravenous fluid rate (three ampules [132 mEq] of sodium bicarbonate in 1 L of D5W). Serum pH must be monitored during the sodium bicarbonate infusion, which should be stopped if the pH reaches 7.55 or higher.
**Fig. 145.1 Algorithm for the recognition and treatment of anticholinergic toxicity.**

CPK, Creatine phosphokinase; D₅W, 5% dextrose in water; ECG, electrocardiogram; IV, intravenous line; TCA, tricyclic antidepressant.

**Anticholinergic toxidrome**
- Presence of dry skin and mucous membranes, mumbling speech, delirium, tactile/visual hallucinations?
  - Yes: Anticholinergic toxidrome
  - No: Consider other diagnoses and alternative treatment pathways

**Sympathomimetic toxidrome**
- Presence of diaphoresis, aggressive behavior, and paranoid hallucinations?
  - Yes: Torsades de pointes
  - No: Monitor

**Possible TCA toxicity**
- ECG with QRS >100-120 msec
  - Yes: Possible TCA toxicity
    - 1) Sodium bicarbonate (1-2 mEq/kg bolus until ECG normalizes or pH ≥ 7.55)
    - 2) No physostigmine
  - No: Monitor

**Psychomotor agitation, seizures, hyperthermia**
- Yes: Physostigmine (1-2 mg adults, 0.02 mg/kg children over 5 minutes; may repeat in 5-10 minutes)
  - No: Benzo diazepines (titrate to sedation)
    - 3) If benzo diazepines do not stop seizures, use barbiturates
    - 4) Cool with tepid water and fans
    - 5) Serum CPK and urine myoglobin; maintain urine output (3-5 mL/kg/hr), bicarbonate as needed

**Consider activated charcoal decontamination only if airway is protected**
- Admit for observation and further treatment; watch for diaphoresis, bradycardia, or bronchorrhea after physostigmine (treat with atropine); give activated charcoal if airway protected
- Admit for observation and further treatment; give activated charcoal if airway protected
- After observation, patients who do not require sedation and who have normal vital signs, normal ECG, normal mental status, and lack of evidence for end-organ injury may be medically cleared.
ANTIDOTAL THERAPY—PHYSOSTIGMINE

Physostigmine is a tertiary amine carbamate that penetrates into the CNS. It reversibly inhibits cholinesterases in both the central and peripheral nervous systems, thereby allowing acetylcholine to accumulate within the synapse. Accumulation of acetylcholine directly antagonizes the anticholinergic effects of antimuscarinic agents.

In the setting of a clear diagnosis of anticholinergic toxicity, physostigmine should be administered. It is beneficial in the treatment of agitation and delirium and also shortens the time to recovery after agitation. This agent should not be given once a benzodiazepine has been administered because the end point of clear mental status has been lost.\(^7\)

During and immediately after the administration of physostigmine, patients must be monitored for early signs of cholinergic toxicity, such as diaphoresis and slowing of the heart rate. Atropine should be kept at the bedside and should be given in titrated doses if needed for cholinergic toxicity (development of bronchorrhea, hypoxia, bradycardia).

Physostigmine is indicated for patients with central (and perhaps peripheral) anticholinergic manifestations. It is contraindicated in patients with a QRS interval longer than 100 msec if the history suggests overdose of tricyclic antidepressant or other cardiotoxic agents. The latter contraindication is based on two case reports of patients with tricyclic antidepressant overdose in whom asystole developed after the administration of physostigmine. The cause of the asystole was theorized to be physostigmine-induced bradycardia that resulted in cardiac conduction defects and decreased cardiac output in the presence of tricyclic antidepressant-induced sodium channel blockade.\(^9\)

Other contraindications to the use of physostigmine are bronchospastic disease, peripheral vascular disease, intestinal or bladder obstruction, intraventricular conduction defects, and atrioventricular block.

Physostigmine is administered intravenously over a 5-minute period, 1 to 2 mg in adults and 0.02 mg/kg (maximum of 0.5 mg) in children. Its onset of action occurs within minutes.\(^3\) This initial dose can be repeated in 5 to 10 minutes if an adequate response is not achieved and muscarinic effects are not noted. Failure of the patient to become cholinergic after the administration of physostigmine is essentially diagnostic of anticholinergic toxicity. Although the total effective dose of physostigmine depends on the individual, as well as the dose and duration of action of the anticholinergic (antimuscarinic) agent, 4 mg is usually a sufficient dose for most patients.\(^10\) The half-life of physostigmine is 16 minutes, and its usual duration of action exceeds 1 hour.\(^11\)

Adverse effects may occur if physostigmine is administered rapidly, in an excessive dose, or in the absence of an anticholinergic (antimuscarinic) agent. In all these instances, an excess of acetylcholine occurs at various sites within the body and has various effects, as follows:

- At nicotinic receptors: muscle fasciculations, weakness, and paralysis
- At muscarinic receptors: bronchospasm, bronchorrhea, bradycardia, salivation, lacrimation, urination, defecation, and emesis
- At CNS sites: anxiety, dizziness, tremors, confusion, ataxia, coma, and seizures

Accordingly, overdoses of physostigmine may require atropine to control the bronchial secretions, and mechanical ventilation may be needed for neuromuscular weakness because no specific antidote is available for the excess nicotinic activity.

Additionally, it is important to note that when other cholinergic agents are used concurrently with physostigmine, the effects may be additive. Examples of such agents are pilocarpine, carbamates, organophosphates, pyridostigmine, and depolarizing neuromuscular blocking agents. Because physostigmine is an acetylcholinesterase inhibitor, it also prolongs the action of drugs metabolized by plasma cholinesterases, such as cocaine, succinylcholine, and mivacurium.

FOLLOW-UP, NEXT STEPS IN CARE, AND PATIENT EDUCATION

Patients in whom the anticholinergic toxicity resolves—who therefore do not require intervention for a period of 6 hours—and who have no evidence of complications of toxicity (e.g., aspiration pneumonia, rhabdomyolysis) may be medically

History
Any history of psychiatric illness or previous suicidal ingestions?
Time since ingestion?

Physical Examination
Does the patient look ill? Febrile? What is the patient’s mental status?
Cardiac (blood pressure, pulse, arrhythmias)?
Airway and respiratory status?
Urine output?
Repeated physical examinations while the patient is still in the emergency department

Diagnostic Studies
Electrocardiogram, blood chemistry panel with serum glucose, serum creatine phosphokinase, urine myoglobin measurements, pregnancy test?
Documentation of acetaminophen and aspirin levels if there is concern about coingestion

Medical Decision Making
Decision to begin treatment or delays in starting treatment
Consultations desired and times of calls to consulting services

Treatment
Response to treatment (especially for sodium bicarbonate, physostigmine)

Patient Instructions
Documentation of discussion with the patient regarding diagnosis, warning signs, what to do, follow-up, and when to return
With pediatric unintentional ingestions, documentation of poison prevention counseling and assessment of the home situation
PATIENT TEACHING TIPS

The regional poison control center can be contacted by telephone at 1-800-222-1222.
A dose of medication other than what was prescribed by the patient’s physician should never be taken unless it has been approved by the ordering physician.
A second medication or herbal supplement should never be added to a previously taken medication without approval from the physician.

TIPS AND TRICKS

Although patients with both adrenergic (sympathomimetic) and anticholinergic (antimuscarinic) poisoning may have similar clinical findings (altered mental status, tachycardia, mydriasis), the two syndromes may be differentiated by skin, type of alteration in mental status, and other clinical findings. Diaphoresis, agitation, paranoid hallucinations, and violent behavior are associated with sympathomimetic agents. Dry skin and mucous membranes, mumbling speech, delirium, and tactile or visual hallucinations are associated with anticholinergics.

cleared. All patients with suspected intentional overdose of any agent should be evaluated by a psychiatrist or otherwise appropriately cleared before hospital discharge according to local practice.

In patients with anticholinergic toxicity, admission to a critical care setting should be arranged for those with altered mental status, cardiac dysrhythmias or drug-related conduction abnormality, hyperthermia, or respiratory compromise requiring mechanical ventilation.

SUGGESTED READINGS


REFERENCES

References can be found on Expert Consult @ www.expertconsult.com.
REFERENCES