



## DRUG OVERDOSE

---

© 2000, 2007 TCHP Education Consortium.

**This educational activity expires December 31, 2017.**

All rights reserved. Copying without permission is forbidden.

## SYNOPSIS/PURPOSE STATEMENT

---

By accident or design, thousands of patients enter into the health care system each year because of drug or alcohol poisoning and overdose. The purpose of this home study is to review the initial care, assessment, management, and ongoing treatment of drug overdose.

## TARGET AUDIENCE

This home study was designed for the novice critical care or telemetry nurse; however, other health care professionals are invited to complete this packet.

## CONTENT OBJECTIVES

1. Identify the scope of alcohol and drug abuse, overdose, and withdrawal in the United States.
2. Describe the initial and ongoing assessment of the patient experiencing a drug or alcohol overdose.
3. Describe the initial management of the patient who has overdosed on drugs or alcohol.
4. Identify indications, contraindications, and methods of poison neutralization.
5. Identify the contributing factors, signs and symptoms, and interventions for overdose on legal drugs and poisons.
6. Describe the assessment and management of the person with acute or chronic alcohol poisoning.
7. Identify desired effect of drug, signs, and interventions for overdose on selected illicit drugs.

## DISCLOSURES

In accordance with ANCC requirements governing approved providers of education, the following disclosures are being made to you prior to the beginning of this educational activity:

### **Requirements for successful completion of this educational activity:**

In order to successfully complete this activity you must read the home study, complete the post-test and evaluation, and submit them for processing.

### **Conflicts of Interest**

It is the policy of the Twin Cities Health Professionals Education Consortium to provide balance, independence, and objectivity in all educational activities sponsored by TCHP. Anyone

participating in the planning, writing, reviewing, or editing of this program are expected to disclose to TCHP any real or apparent relationships of a personal, professional, or financial nature. *There are no conflicts of interest that have been disclosed to the TCHP Education Consortium.*

### **Relevant Financial Relationships and Resolution of Conflicts of Interest:**

If a conflict of interest or relevant financial relationship is found to exist, the following steps are taken to resolve the conflict:

1. Writers, content reviewers, editors and/or program planners will be instructed to carefully review the materials to eliminate any potential bias.
2. TCHP will review written materials to audit for potential bias.
3. Evaluations will be monitored for evidence of bias and steps 1 and 2 above will be taken if there is a perceived bias by the participants.

*No relevant financial relationships have been disclosed to the TCHP Education Consortium.*

### **Sponsorship or Commercial Support:**

Learners will be informed of:

- Any commercial support or sponsorship received in support of the educational activity,
- Any relationships with commercial interests noted by members of the planning committee, writers, reviewers or editors will be disclosed prior to, or at the start of, the program materials.

*This activity has received no commercial support outside of the TCHP consortium of hospitals other than tuition for the home study program by non-TCHP hospital participants.*

If participants have specific questions regarding relationships with commercial interests reported by planners, writers, reviewers or editors, please contact the TCHP office.

## CONTACT HOUR INFORMATION

### Non-Endorsement of Products:

Any products that are pictured in enduring written materials are for educational purposes only. Endorsement by WNA-CEAP, ANCC, or TCHP of these products should not be implied or inferred.

### Off-Label Use:

It is expected that writers and/or reviewers will disclose to TCHP when “off-label” uses of commercial products are discussed in enduring written materials. *Off-label use of products is not covered in this program.*

### Expiration Date for this Activity:

As required by ANCC, this continuing education activity must carry an expiration date. The last day that post tests will be accepted for this edition is **December 31, 2017**—your envelope must be postmarked on or before that day.

For completing this **Home Study and evaluation**, you are eligible to receive:

**2.0 MN Board of Nursing contact hours / 1.66 ANCC contact hours**

**Criteria for successful completion:** You must read the home study packet, complete the post-test and evaluation, and submit them to TCHP for processing.

The Twin Cities Health Professionals Education Consortium is an approved provider of continuing nursing education by the Wisconsin Nurses Association, an accredited approver by the American Nurses Credentialing Center’s Commission on Accreditation.

## PLANNING COMMITTEE/EDITORS

**Linda Checky, BSN, RN, MBA**, Assistant Program Manager for TCHP Education Consortium.

**Lynn Duane, MSN, RN**, Program Manager for TCHP Education Consortium.

## AUTHOR

**Karen Poor, MN, RN**, Former Program Manager, TCHP Education Consortium.

## CONTENT EXPERTS

**Cleo Bonham, MSN, RN**, Clinical Instructor of Critical Care, Minneapolis VA Medical center.

\***Cody Schultz, MSN, RN**, Former Clinical Educator, MICU, Regions Hospital.

\*Denotes reviewer of current edition

Please see the last page of the packet before the post-test for information on submitting your post-test and evaluation for contact hours.

## INTRODUCTION

---

We've heard the statistics regarding drug and alcohol abuse:<sup>1</sup>

- Alcohol abuse and alcoholism costs \$148 billion/year (1992)
- Drug abuse and dependence costs \$98 billion/year (1992)
- An estimated 3.6 million people used cocaine in 1997
- 600,000 people needed treatment for heroin addiction in 1997
- More than 132,000 people died as a result of drug and alcohol problems in 1992; 81% of which were related to alcohol.

But what does that mean to you in the health care setting? Each of the numbers listed in the statistics is a person -- someone that you or I will eventually end up caring for in the ER, ambulatory care, clinic, med-surg area, surgery, or ICU. We need to be aware of the initial care, detoxification, treatments, and ongoing care of the patient with a chemical abuse problem so that we can care for that person in the best way possible.

## INITIAL ASSESSMENT AND MANAGEMENT

---

Assessment of the patient who has overdosed is not a one-time deal -- constant vigilance during the acute period is absolutely vital. Initial assessment focuses on the ABC's and the patient's neurological and safety status. The following is a case example of a patient who enters the ER with an ingestion of an unknown substance.

*Peter J. was brought in by friends who rolled him out of their car in front of the Emergency Room before speeding away. The Emergency Room staff lifted him onto a gurney and brought him into a triage room.*

### AIRWAY

*Peter was unresponsive to stimuli; his tongue was found to be blocking his airway. An oral airway was inserted while preparations were made for intubation. He was positioned on his left side, in a slight Trendelenburg position.*

Why an oral airway? Patients who have airway obstruction may require an oral airway until they can be intubated. There is a high risk of vomiting and aspiration with an oral airway, so these airways should not be inserted in a patient with a gag reflex and aspiration precautions should be followed.

Nasal airways can be used for the patient who has a **patent** airway. Nasal airways are helpful in providing a track for suctioning and protecting the nasal passages from trauma during nasotracheal suctioning.

Placing a semi-conscious or unconscious patient in the left side, in a slight Trendelenburg position helps prevent aspiration. If the patient does vomit, the slight downward slope of the bed will allow the vomitus to drain rather than to be aspirated. Because of the anatomy of the bronchi, left side positioning is better to prevent aspiration.

## BREATHING

*Even with the insertion of the oral airway, Peter's breathing was shallow and slow at 9 breaths/minute. On auscultation, he was moving minimal air -- audible only in the upper lung fields. He was emergently intubated and placed on a ventilator.*

Intubation and mechanical ventilation is indicated for patients who:

- Have an inadequate respiratory effort
- Are unconscious
- Have an impaired or absent gag reflex
- Are in *status epilepticus*
- Have ingested a drug that is known to cause rapid deterioration

Mechanical ventilation accomplishes several things:

1. It provides oxygen to the compromised patient.
2. The endotracheal tube provides some protection from aspiration.
3. It helps alkalize the blood in certain drug overdoses

Assessment after intubation should include:

1. Check end-tidal CO<sub>2</sub> for bedside assessment of tube placement

2. Auscultation of lung fields to ensure that breath sounds are equal
3. Monitoring of oxygen saturation
4. X-ray for tube placement
5. ABG analysis

## CIRCULATION

On the cardiac monitor, Peter had a rapid heart rate of 180 beats/min in a supraventricular tachycardia with frequent PVC's. His blood pressure was 80/54. His extremities were cool and clammy. His skin color was ashen.



Many drugs, taken in an overdose, can themselves cause cardiac dysrhythmias. Drugs that will cause dysrhythmias are listed in Table 1. Dysrhythmias can also be caused by hypoxia, acidosis, and electrolyte imbalances. It's important to keep a close eye on the cardiac rhythm to see changes or abnormalities in the rate, rhythm, QRS width, PR interval, and QT interval.

**Table 1: Drugs That Cause Rhythm Disturbances**

Conduction Abnormalities/Heart Block <sup>2</sup>	
<ul style="list-style-type: none"> <li>• Alpha-1 antagonists (Cardura, Minipres)</li> <li>• Alpha-2 agonists (Aldomet, Catapres, Clonidine)</li> <li>• Arsenic</li> <li>• Beta blockers</li> <li>• Bupivacaine</li> <li>• Calcium channel blockers</li> </ul>	<ul style="list-style-type: none"> <li>• Carbamazepine</li> <li>• Cocaine</li> <li>• Cyclic antidepressants</li> <li>• Digoxin</li> <li>• Pentamidine</li> <li>• Phenothiazines (antipsychotics)</li> <li>• Propoxyphene</li> </ul>

### Bradycardia<sup>3</sup>

<ul style="list-style-type: none"> <li>• Alpha-2 agonists</li> <li>• Beta blockers</li> <li>• Digoxin</li> <li>• Edrophonium</li> <li>• Massive exposures to anticholinergics, cocaine, cyclic antidepressants, sympathomimetics</li> </ul>	<ul style="list-style-type: none"> <li>• Misoprostol</li> <li>• Neostigmine</li> <li>• Opioids</li> <li>• Organophosphates or Carbamates</li> <li>• Physostigmine</li> <li>• Sedative-hypnotics</li> </ul>
---	--

### Supraventricular or Ventricular Dysrhythmias<sup>4</sup>

<ul style="list-style-type: none"> <li>• Antidysrhythmias</li> <li>• Anticholinergics</li> </ul>	<ul style="list-style-type: none"> <li>• Ethanol</li> <li>• Flumazenil</li> </ul>
--	---

<ul style="list-style-type: none"> <li>• Antihistamines</li> <li>• Baclofen</li> <li>• Carbamazepine</li> <li>• Catecholamines</li> <li>• Cocaine</li> <li>• Cyclic antidepressant agents</li> <li>• Digoxin</li> </ul>	<ul style="list-style-type: none"> <li>• Hydrocarbons and solvents</li> <li>• Metal salts: arsenic, lithium</li> <li>• Pentamidine</li> <li>• Phenothiazines</li> <li>• Thyroid hormone preparations</li> </ul>
---	---

*Note: This list is not inclusive of all drugs that may cause these problems.*

**Hypotension** can also be a direct effect of the drug overdose by causing peripheral dilation, venous pooling, and myocardial depression (see Table 2). The patient may also be hypovolemic, have experienced fluid shifts, or arrhythmias.



**Table 2: Drugs That Cause Hypotension<sup>5</sup>**

<ul style="list-style-type: none"> <li>• Drugs causing vomiting and diarrhea</li> <li>• ACE inhibitors</li> <li>• Alpha-2 antagonists: alpha-methyldopa, clonidine, guanabenz</li> <li>• Reserpine</li> <li>• Prazosin</li> <li>• Hydralazine</li> <li>• Nitrates</li> <li>• Beta blockers</li> <li>• Calcium channel blockers</li> </ul>	<ul style="list-style-type: none"> <li>• Cyclic antidepressants</li> <li>• MAO inhibitors</li> <li>• Antiparkinson agents: bromocriptine, L-dopa, perolide mesylate</li> <li>• Thiazides</li> <li>• Loop diuretics</li> <li>• Ethanol</li> <li>• Opioids</li> <li>• Sedative-hypnotics</li> <li>• Phenothiazines (antipsychotics)</li> </ul>
---	--

*Note: This list is not inclusive of all drugs that may cause these problems.*

Treatment of hypotension is based either on fluid volume replacement or antagonism of ingested poison if available. Specific treatments are listed in Table 3.

**Table 3: Specific Treatments for Drugs Causing Hypotension<sup>6</sup>**

If the person took an overdose of:	And Start (in order of preference):
Beta-blocking drugs, give: Glucagon (1 <sup>st</sup> choice) and amrinone	<ol style="list-style-type: none"> <li>1. Epinephrine</li> <li>2. Dopamine</li> <li>3. Dobutamine</li> <li>4. Isoproterenol</li> </ol>
Calcium channel blockers, give calcium (1 <sup>st</sup> choice), glucagon, and	<ol style="list-style-type: none"> <li>1. Dopamine</li> <li>2. Dobutamine</li> <li>3. Isoproterenol</li> </ol>

amrinone	
Clonidine, give naloxone (Narcan)	Dopamine
Alpha antagonists or phenothiazines	<b>Don't give dopamine!</b> 1. Norepinephrine (Levophed) 2. Phenylephrine (Neo-synephrine)
Cyclic antidepressants, give sodium bicarbonate	<b>Don't give dopamine!</b> 1. Norepinephrine (Levophed) 2. Phenylephrine (Neo-synephrine)
Cholinergic drugs, give atropine	Dopamine
Opioids, give naloxone (Narcan)	Dopamine
Magnesium, give calcium	Dopamine

**Hypertension** is rare but can occur with several different poisonings (see Table 4). A benzodiazepine is the drug of choice for a sympathomimetic overdose (e.g. cocaine); sedative hypnotic drugs are often successful in managing the hypertension and tachycardia of many other drugs. If the hypertension is not controlled with a sedative drug, nitroprusside, nitroglycerin, or phentolamine can be used. Beta blocking and calcium channel blocking agents have been poorly studied, and may in fact worsen hypertension and tachycardia related to some drug poisonings.

**Table 4: Drugs That Cause Hypertension<sup>7</sup>**

<ul style="list-style-type: none"> <li>Phenylephrine (Neo-synephrine)</li> <li>Ergotamines</li> <li>Amphetamines</li> <li>Cocaine</li> <li>MAO inhibitors</li> <li>Cyclic and non-cyclic antidepressants</li> <li>Cyclobenzaprine (Flexeril)</li> <li>Dopamine</li> <li>Ephedrine</li> </ul>	<ul style="list-style-type: none"> <li>Oxymetazoline (Afrin)</li> <li>Tetrahyzoline (Visine)</li> <li>Pseudoephedrine</li> <li>Angiotensin</li> <li>Isoproterenol</li> <li>Albuterol (Proventil, Ventolin)</li> <li>Metaproterenol (Alupent)</li> <li>Terbutaline (Brethine)</li> <li>Nicotine</li> <li>Steroids</li> <li>Diphenhydramine (Benadryl)</li> </ul>
--	---

*Note: This list is not inclusive of all drugs that may cause these problems.*

## NEUROLOGICAL ASSESSMENT AND MANAGEMENT

*Peter remained unresponsive to all stimuli. His extremities were flaccid initially; after intubation, Peter*

*began to exhibit some decorticate posturing. His Glasgow Coma Scale Score was 4.*

Patients with decreased mental status of unknown cause should be treated with Narcan, thiamine, and be assessed for hypoglycemia using a bedside glucose monitor.



- Glucose is given in case the patient is experiencing a hypoglycemic coma.
- Naloxone (Narcan) is given to reverse the effects of certain narcotics.
- Thiamine is given just in case the patient is in an alcohol-induced coma. Administration of glucose containing IV fluids without thiamine can precipitate Wernicke's syndrome.

There are times when overdose patients have seizures -- from the drug effect, from anoxia, or as a result of a pre-existing condition. Many, many drugs can cause seizures. Diazepam (Valium) is the drug of choice for stopping the seizures, although phenytoin (Dilantin) or phenobarbital may be necessary to keep the seizures under control.

### Safety

Safety is a huge issue for the patient who has overdosed. Because the person is under the influence of drugs, he/she may have a decreased level of consciousness. The patient should be monitored closely, and side rails and restraints should be used as necessary.

Your patient may also be agitated or hallucinating. Reorient the patient as often as possible, utilizing standard safety measures. If your patient is at higher risk for injury, haloperidol (Haldol) or benzodiazepines may be administered. Propofol is a commonly used drug for intubated patients.

The third issue is the possible suicidal nature of the overdose. Patients who knowingly took an overdose with the intention of killing themselves are at risk for self-injurious behavior in the hospital. Suicidal patients should be monitored constantly. Keep the alarms on, the urinary catheter in place, and all IV equipment and supplies out of reach.

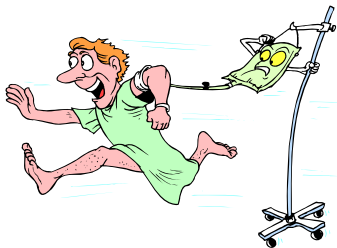
## HISTORY

*Peter has a long history of hospitalizations for mental illness and alcohol abuse. The psychiatrist treating Peter had prescribed a tricyclic antidepressant during his last hospitalization. Because Peter is comatose, he is not able to give a history.*

Getting a history from patients who overdose can be a challenge. Even if the patient is awake and alert, his/her recollection of the event or truthfulness can be in question. If the patient is able to answer questions, try to elicit the following information:

1. WHAT did he/she take?
2. WHAT route was the drug ingested by -- oral, nasal, smoking, or injection?
3. HOW much did he/she take?
4. WHEN did he/she take it?
5. Was there anything else that the person took? Many people poly-drug overdose -- they may overdose on pills, but wash it down with alcohol. Or they may have injected cocaine and taken benzodiazepines to mellow out.
6. WHY did the person overdose?

If possible, look for confirming information. Paramedics will usually look for things like pill bottles, pipes, and alcohol bottles at the immediate scene. If there is a significant other, ask that person about what the patient took, or what was available at the scene. For example, if it was an accidental overdose, ask the significant other about how many pills were in the bottle, or how much liquid there had been in the bottle.



## IDENTIFICATION

Lab results can confirm the presence of drugs and alcohol. There are several types of tests:



### Qualitative toxicology screen:

- Lists the drugs that are present in the blood.

- Seven types of commonly abused drugs are screened for: THC, cocaine, benzodiazepines, amphetamines, PCP, opiates, and barbiturates.
- Does not give amounts of drug in the blood.
- A urine sample is the specimen of choice.
- Preliminary data on a routine sample may take up to 2 hours; a stat sample can yield preliminary results in 15 minutes.
- Confirmatory methods may be needed to detect certain substances; that will increase the result time by up to 24 hours.

### Quantitative toxicology screen:

- Identifies the amount of the drug in the blood.
- The drug(s) to be screened for must be known before the test is run.
- Tegretol, Dilantin, Phenobarbital, Depakote, and tricyclic antidepressant levels can be done.

### Chemistry results:

- Acetaminophen and theophylline levels are run through a blood test in the chemistry lab -- not toxicology.
- Blood alcohol level -- tests for ethanol level; results obtained in less than 30 minutes in STAT labs.
- Methanol, ethylene glycol, and isopropyl alcohol levels must be tested for separately.



## NEUTRALIZING THE POISON

After initial stabilization, the goal is to get rid of as much of the drug as possible. There are a number of ways to accomplish this -- none of which are pleasant for the patient. All of the following measures are most effective if used in the first 30 minutes after ingestion.

## GASTRIC EMPTYING

In the old days, Syrup of Ipecac was one of the mainstays of poison control. Ipecac acts in two ways: initial vomiting is caused by irritation of the GI tract; continued vomiting results from stimulating the vomiting center in

the brain. Ipecac is no longer routinely recommended by the Poison Control Center after admission to the hospital because of its long action (> 20 minutes). Apomorphine is a short acting emetic which is given subcutaneously; its onset of action is 4-6 minutes. Apomorphine has not been studied in this application, so we don't know how well it works in the overdose population.

There are certain patients who may receive emetics -- those who have ingested substances that are too big to come out through a lavage tube. Examples include drug packets, large pills, pills that are "stuck together" in a mass, and mushroom fragments.

Patients should not receive either emetic drug if they have:

- a decreased level of consciousness
- an impaired gag reflex
- seizures
- ingested a caustic substance
- ingested a substance which is known to rapidly cause coma, seizures, or arrhythmias

## GASTRIC LAVAGE

In gastric lavage, a large bore tube, 36-40 Fr Burke or Ewald, is placed orally when the adult patient is lying on his left side (for children, the tube size should be between 30 and 32 French). Two hundred to 250 cc's of warm tap water is instilled and allowed to drain by gravity. This process is continued until the return is clear without pill fragments -- not less than 2 liters.

Placement of the tube can cause the patient to vomit -- any indication that the patient may not be able to protect his airway should be looked for very carefully. Intubation may be indicated for the patient who has an impaired gag reflex, seizures, decreased LOC, or has ingested a substance known to cause rapid deterioration. Contrary to previous practice, ingestion of a caustic substance is not a contraindication to lavage. The procedure allows for dilution and removal of the corrosive substance and will provide a clearer view should endoscopy be needed.

The complications of gastric lavage? Aspiration, laryngospasm, hypoxia and hypercapnia, oral, esophageal, or gastric injury, hypothermia (mostly in children), and fluid and electrolyte imbalances (mostly in children).

## ACTIVATED CHARCOAL



It's not the charcoal you use on your grill, but it's close. It is charcoal that has been activated by a steam or acid so that it will bind with the drugs in the gut. It also encourages the movement of drug from the bloodstream back into the gut. See Table 5 for drugs that are eliminated with multiple dose activated charcoal (MDAC). Ninety-99% of the drug is adsorbed by the charcoal within one minute of contact. The charcoal is mixed with water or sorbitol in commercial preparations and comes in a 50-100 gm bottle.

**Table 5: Toxins Eliminated by MDAC<sup>8</sup>**

Acetaminophen	Meprobamate
Amitriptyline	Methotrexate
Carbamazepine	Nadolol
Cyclosporine	Nortriptyline
Dextropropoxyphene	Phenobarbital
Diazepam	Phenylbutazone
Digoxin	Piroxicam
Disopyramide	Propoxyphene
Glutethimide	Theophylline
	Valproic acid

There is inadequate data to say if activated charcoal is effective more than one hour after ingestion, but its use is not contraindicated. The first dose of charcoal is usually mixed with a cathartic (usually sorbitol), and is given at a 0.5 - 1.0 mg/kg dose. Further doses of charcoal (multi-dose activated charcoal = MDAC) may be given; although research as to its effectiveness is mixed. Patients receive 0.5 mg/kg in later doses.

Charcoal can be taken orally by the patient; it is unpleasant and may cause vomiting. Charcoal may also be administered through an NG tube.

## WHOLE BOWEL IRRIGATION/ WHOLE GUT LAVAGE

There are certain types of overdoses that may benefit from whole bowel irrigation (WBI). Patients who have ingested multi-vitamins, iron, arsenic, lead, or sustained release calcium channel blocking drugs fall into this category. WBI is performed by administering an isotonic polyethylene glycol electrolyte lavage either orally or through an NG tube. The solution literally "flushes" the

bowel, without causing major fluid imbalances as charcoal can.

## HEMOPERFUSION AND HEMODIALYSIS

Many drugs that are absorbed into the blood stream can be removed either by hemoperfusion or hemodialysis. This is a very invasive procedure and is used only if it is felt that other methods would not be effective.

**Table 6: Drugs Removed Via Hemoperfusion and Hemodialysis<sup>9</sup>**

Acetaminophen	Methanol
Amitriptyline	Methaqualone
Bromide	Methotrexate
Carbamazepine	Nortriptyline
Digitoxin	Paraquat
Digoxin	Pentobarbital
Ethanol	Phenobarbital
Ethchlorivynl	Phenytoin
Ethylene glycol	Procainamide
Glutethimide	Salicylate
Isopropyl alcohol	Theophylline
Lithium	Trichloroethanol

Source: *Poisoning and Drug Overdose Clinical Manual* (1994)

## COMMON LEGAL DRUGS FOR OVERDOSE

There are many types of drugs on which to overdose or become poisoned. This section covers the most common drugs and alcohols in relation to their clinical presentation and specialty management.

### ACETAMINOPHEN

*Jacque A. was a 16 year old junior in high school who was admitted to the ICU following an acetaminophen overdose. She was alert, oriented, and scared. Four days ago, depressed over a breakup with her boyfriend, Jacquie took 30 acetaminophen tablets. She was nauseated over the next twenty-four hours and vomited several times. She told her mother that she had the flu.*

*Over the next two days, Jacquie felt fine, although still depressed. She still did not tell anyone that she had taken the acetaminophen.*

*At the time of admission, Jacquie had a recurrence of the nausea, and developed severe right upper quadrant pain. She told her mother that she was sick and what she had taken four days before. Her mother brought her into the ER, at which time she had petechiae and bruising, she was slightly jaundiced, and according to her mother, confused. Her liver function tests showed an elevated SGOT, SGPT, alkaline phosphatase, bilirubin, and PT. Her serum glucose was 62 mg/dL.*

Acetaminophen has unjustly earned the reputation of a "benign" drug. It isn't. It is the drug with the highest overdose rate and can cause liver failure (as Jacquie has), DIC, renal failure, cardiac dysrhythmias, and death. Acetaminophen is metabolized in the liver to form mostly harmless metabolites and the very toxic *N*-acetyl-*p*-benzoquinoneimine (NAPQI). It is the NAPQI that is very toxic to the liver.

There are four phases of acetaminophen poisoning:<sup>10</sup>

#### Phase One (1/2 hour to 24 hours after ingestion)

Anorexia, nausea/vomiting  
Diaphoresis

#### Phase Two (24 hours to 72 hours after ingestion)

Decreased physical symptomology  
RUQ tenderness/discomfort  
Labs: increased LFT's, bilirubin, PT

#### Phase Three (72 - 96 hours after ingestion)

Coagulation defects  
Jaundice  
Renal failure  
Myocardial pathology  
Hepatic encephalopathy  
Nausea/vomiting  
Anuria  
Coma to death

#### Phase Four (4 days to 2 weeks after ingestion)

Complete resolution of hepatic damage if patient survives

*Even though the overdose was four days prior, a lavage was done, but MDAC was not initiated because it was a single drug overdose. A loading dose of 11,200 mg of acetylcysteine was given (Jacquie was 80 kg) orally.*

Jacque gagged on the loading dose, but was able to keep it down.

The toxicity of acetaminophen and the blood level is closely correlated. The graph that charts out the correlation shows that a level of > 200 mg/dl four hours after ingestion or greater is toxic. The blood level of acetaminophen should drop every four hours by half. At eight hours after ingestion, the toxic level is > 100; at twelve hours, the toxic level is > 50; at 16 hours, the toxic level is > 25 mg/dl.

A drug called N-acetylcysteine (Mucomyst or NAC) can be given orally or intravenously. NAC prevents the formation of NAPQI and increases the liver's capacity to detoxify NAPQI. Oral NAC is a nasty smelling and tasting drug -- you should always mix acetylcysteine with a cola, cover the glass with a lid, and have the patient drink from a straw or "chug it." The loading dose is 140 mg/kg, followed by 17 doses of 70 mg/kg every four hours. Because acetylcysteine is noxious, and the patient is likely already nauseated, Compazine may be given to decrease the likelihood of vomiting. If the patient vomits within an hour of administration, the NAC dose is repeated. NAC is widely given IV, but is not yet approved by the FDA for IV administration. NAC must be filtered for precipitates and must be given slowly to prevent a rash, bronchospasm, hypotension, and death.

MDAC is indicated if the patient ingested drugs other than acetaminophen; e.g. codeine.

Jacque had an acetaminophen level, LFT's and coagulation studies drawn every four hours, and received vitamin K for an elevated PT level. Jacque continued the acetylcysteine treatment and had an appropriate decrease in the acetaminophen level. Her liver enzymes dropped back to normal after one week, and she was discharged to home with outpatient psychiatric care set up.

## TRICYCLIC ANTIDEPRESSANT (TCA) DRUGS

Normally prescribed for depression, this type of drug is one of the most common overdoses admitted to the ICU setting. Amitriptyline, desipramine, doxepin, imipramine, and nortriptyline are all examples of TCA drugs.

Peter, the young gentleman dropped off at the ER, was known to have been on TCA drugs for depression. Based

on his history, an overdose was suspected. Peter had the following physical examination:

*Vital signs:* HR 144; BP 78/40; RR 10; Temp 103.2°  
*EKG:* PR 0.28, QRS width 0.14, sinus tach  
*Cardiac:* Hypotensive, flushed dry skin  
*Neuro:* Comatose; non-responsive to painful stimuli. Positive Babinski reflex. Flaccid extremities, dried froth around the mouth, bruises on the back of the head and tailbone (evidence of seizure)  
*Respiratory:* Shallow, slow at rate of 10. ABGs: pH 7.27, PaO<sub>2</sub> 80, PaCO<sub>2</sub> 40, HCO<sub>3</sub>-8

These symptoms are reflective of the "typical" severe TCA overdose patient. Other symptoms that may be seen include:

- ◆ Urinary retention
- ◆ Decreased bowel sounds
- ◆ Nausea and vomiting
- ◆ Restlessness
- ◆ Myoclonic jerking
- ◆ Hallucinations
- ◆ Difficulty with vision

The life-threatening toxic level for cyclic antidepressants is somewhere around 1,000 mg/dl. Cardiac dysrhythmias, seizures, and coma commonly occur with levels > 1,000 mg/dl. According to one study, 50% of patients with a QRS complex > .16 sec (160 msec) experienced seizures.<sup>11</sup>

Peter was emergently intubated for airway protection and to assist with the correction of his metabolic acidosis. He was lavaged with tap water and given an initial dose of MDAC. A urinary catheter and NG tube were placed. Initial toxicology results showed that he had TCA and alcohol in his system.

Wide complex tachycardias, loss of consciousness, seizures, and inability to protect the airway can occur within minutes of the overdose. Almost all life-threatening symptoms will occur within six hours of the overdose, with most occurring within the first two hours.

Because of the evidence of a seizure, diazepam and phenytoin were kept at the bedside. His side rails were in the upright position with padding to prevent injury if he

was to seize again. Peter's cardiac status was of concern: he was hypotensive, tachycardic, and had prolonged PR and QRS intervals. He was given 1 liter of D5.9NS with no change in his vital signs. Peter was then started on a Levophed drip. Peter was given a 2 mEq/kg bolus of sodium bicarbonate and was placed on an infusion of bicarb to maintain a blood pH of 7.50. A temporary pacemaker was kept on standby in case he was to go into a complete heart block or asystole.

TCA drugs, as well as other cyclic antidepressants, "numb" the cardiac conduction system. The effect of the cyclic antidepressants on the myocardium can be diminished by giving sodium bicarbonate and hyperventilating the patient to achieve a serum pH of 7.50 - 7.55. The urinary pH is followed closely with a goal > 7.0, preferably 7.5-8.0. The goal is to see a QRS interval of less than 0.10 sec (100 msec) and an increase in the BP. Hypotension usually caused by peripheral vasodilation and can be treated with fluid volume. In more severe cases, hypotension may be related to myocardial depression. Dobutamine will help increase contractility to boost the BP.

He was also placed on a cooling blanket to control his hyperthermia. Ultimately, Peter required hemodialysis to clear his blood of TCA because of a widening QRS interval and continued hypotension and seizures.

Peter had a cardiac arrest four hours after admission to the ICU. He did not survive.

## SSRI'S - SELECTIVE SEROTONIN REUPTAKE INHIBITORS

It used to be that TCA drugs were the most common drugs on which to overdose. SSRIs, however, surpassed the TCA drugs in 1997 and accounted for 55,000 overdoses.<sup>12</sup> The first SSRI was Prozac (fluoxetine); others include Paxil (paroxetine), Luvox (fluvoxamine), Zoloft (sertraline), and Nitalpram (citalopram).

### What do SSRI's do?

Selective serotonin reuptake inhibitors were designed specifically to block the reuptake of serotonin, one of the neurotransmitters. This action allows serotonin to circulate longer, alleviating the symptoms of depression.

### What are the signs of an SSRI overdose?

A pure SSRI overdose is rarely life-threatening; still there are manifestations that should be watched for. The patient may have nausea, vomiting, dizziness, blurred vision, and possibly CNS depression and sinus tachycardia. Only rarely do seizures occur.

### What is the treatment for an SSRI overdose?

Because of the possibility of CNS depression, people who overdose on an SSRI are not given emetics. Gastric lavage is not indicated for a pure SSRI overdose because of the low lethality; however, it may be indicated if there is a polydrug overdose.

## BENZODIAZEPINES

Elsie Ativan is an 86 year old woman with chronic anxiety. She normally takes 1/2 of a lorazepam tablet to control her anxiety when she is going somewhere. Lately, she has been having more and more anxiety attacks. She went to see a physician and had a new drug prescribed -- alprazolam. Elsie began to take one tablet of lorazepam and one tablet of alprazolam 5-6 times per day because of her continued anxiety. She comes into the ER after family members noticed that she was having a change in the level of consciousness.

The benzodiazepines are typically prescribed for anxiety relief or sleep. Common benzodiazepines are diazepam (Valium), temazepam (Restoril), lorazepam (Ativan), chlordiazepoxide (Librium), oxazepam (Serax), chlorazepate (Tranxene), and alprazolam (Xanax).

When Elsie comes to the ER, her neurological status had deteriorated. She was somnolent and lethargic when aroused. Her respiratory rate was 12/minute. She moved all four extremities weakly, pupils were equal and reactive, and she was mentally confused. A toxicology screen was ordered, and STAT results showed the presence of lorazepam and alprazolam.

Benzodiazepines typically cause muscle relaxation and neurological depression. An overdose will cause an exaggeration of those effects. Death from a benzodiazepine-alone overdose is very rare; deaths occur from a combination use of a benzodiazepine and another drug, most commonly alcohol. Of concern, however, are

that overdoses on the faster-acting benzodiazepines (temazepam, alprazolam, and triazolam) have caused fatalities.

*Based on her physical examination and the toxicology screen, Elsie was given Flumazenil, 3 mg over 6 minutes. The physicians elected not to lavage Elsie because of the possibility of aspiration. Elsie had a generalized tonic-clonic seizure 10 minutes after being given the Flumazenil. She was intubated to protect her airway and lavaged.*

Flumazenil is a reversal agent for the benzodiazepines that should be used with great caution. Overdose patients may receive from 3-6 milligrams at a rate no faster than 0.5 mg/minute (6-12 minutes). Patients who are dependent on benzodiazepines may have a seizure before regaining alertness, which is treated symptomatically. Re-sedation can also occur, as the flumazenil wears off and the benzodiazepines remain in the system.

*Elsie regained alertness in 75 minutes. She required one more dose of flumazenil for rebound sedation. She was extubated shortly thereafter. She was transferred out of the ICU the next day and given effective patient education.*

Rapid neutralization or cessation of a benzodiazepine can cause withdrawal symptoms. Those symptoms include:

- Changes in perception
- Paraesthesias
- Headaches
- Tremors
- Weight loss

People who have been on benzodiazepines for a long period of time should be tapered slowly to avoid anxiety and withdrawal symptoms.

## ASPIRIN (SALICYLATES)

*Ralph W. was admitted to the MICU after a brief pass-through in the ER. The physicians were puzzled as to his diagnosis. His physical examination showed the following:*

*Vital signs: HR 179; BP 68/32; RR 46; Temp 104.1*

*EKG: SVT with occasional PVCs*

*Cardiac: Skin warm, moist, ashen; pulses faint and equal*

*Respiratory: Lungs clear; RR 46 and deep; SaO<sub>2</sub> 100%. ABGs--pH 6.99, PaO<sub>2</sub> 99, PaCO<sub>2</sub> 14, HCO<sub>3</sub><sup>-</sup> 2*

*Neuro: Extremely agitated; calling out "I'm gonna die, I'm gonna die." constantly. Unresponsive to commands. Moving all extremities. Does not appear to hear or track.*

*Ralph was treated symptomatically -- a cooling blanket for his hyperthermia, safety measures to prevent injury, supplemental oxygen, sodium bicarbonate for his metabolic acidosis. He was given 1 L of NS without change in his vital signs and was started on Dobutamine for vasopressor support. His toxicology screen came back with aspirin levels detected. Based on his clinical symptoms, a diagnosis of aspirin overdose was made.*

Aspirin -- a salicylate -- is a substance that has two major pathophysiologic consequences:

- ♦ Aspirin stimulates the respiratory center, causing hyperventilation, hypocapnia, and respiratory alkalosis.
- ♦ Aspirin interferes with the Krebs cycle, causing decreased ATP and increased lactate production, ultimately causing a metabolic acidosis.

Aspirin can also be a "hidden" overdose -- many drugs, such as over the counter cold medications and pain medications, contain aspirin. Aspirin levels will continue to rise for 8 to 10 hours after ingestion, with enteric-coated aspirin overdoses taking even longer.

Unlike acetaminophen, aspirin toxicity does not correlate well with blood levels. In general, however, a salicylate level of > 40 mg/dl is considered toxic. The blood salicylate level, the pH, and the patient's signs and symptoms are all considered when determining toxicity.

### Physical and Laboratory Assessment of Salicylate Toxicity<sup>13</sup>

#### CNS

- Ringing in the ears (tinnitus)
- Vertigo
- Hallucinations

- Agitation/delirium
- Seizures

#### *Acid-Base Disturbances*

- Respiratory alkalosis (early)
- Metabolic acidosis (mid-late)

#### *Gastrointestinal/Hepatic*

- Nausea/vomiting
- Hemorrhagic gastritis
- Abnormal liver enzymes
- Altered glucose metabolism (hyper/hypoglycemia)

#### *Renal*

- Tubular damage
- Ketonuria

#### *Metabolic*

- Increased temperature
- Ketonemia

#### *Pulmonary*

- Hypernea (deep respirations)
- Tachypnea (fast respirations)
- Non-cardiogenic pulmonary edema

*After a lavage and administration of activated charcoal, Ralph is started on a bicarbonate drip with a 2 mEq/kg bolus, followed by an infusion to keep his urinary pH above 7.5. His potassium comes back at 3.0, and he is aggressively replaced with KCl. The physicians are currently discussing hemodialysis because of Ralph's rapidly rising BUN and creatinine and continued metabolic acidosis.*

### **Alkalinization**

One of the most effective treatments for severe salicylate toxicity (> 35 mg/dL) is the use of sodium bicarbonate. Sodium bicarbonate will pull salicylate (an acid) out of the tissues and into the bloodstream where it is trapped and can be excreted by the kidneys. The drip is initiated at 1-2 mg/kg of sodium bicarbonate as a bolus, then a sodium bicarbonate drip to keep the urine pH between 7.5 and 8.0.<sup>14</sup>

The effectiveness of the sodium bicarbonate drip hinges partially on the ionic balance in the body. If the patient is hypokalemic, the salicylate is much more reluctant to come out of the cells, which means that the bicarbonate can't latch onto it. Patients should have potassium replacement until the potassium is at a normal level.

### **Hemodialysis/Hemoperfusion/Peritoneal Dialysis**

Patients who are experiencing continued acid-base disturbances or are showing the signs of one (or more) organ(s) failing may be candidates for extracorporeal removal of salicylates. Of the three methods, hemodialysis is the most effective because of the ability to correct fluid imbalances and acid-base disturbances quickly. Hemoperfusion is more gentle and better able to remove the salicylates, but cannot correct the fluid, electrolyte and acid-base problems. Finally, peritoneal dialysis is only about 10-25% as effective as either of the two other methods and is generally not considered.

*After several hours, Ralph was prepared for hemodialysis, but experienced a cardiac arrest and died before HD was begun.*

### **BETA-BLOCKING AGENTS**

In 1998, over 8,000 people had toxic exposures to beta blocking agents, with 4,377 requiring treatment in health care. 206 people experienced major problems and 21 died related to this toxic exposure.<sup>15</sup>

So what are the beta-blockers? The full name for these drugs is "beta ( $\beta$ ) adrenergic blocking agents." These drugs block the sympathetic nervous system from activating the  $\beta$  receptors. The  $\beta$ -1 receptors are primarily in the heart -- activation of these receptors causes an increased heart rate and contractility and increased renin production.  $\beta$ -2 receptors are in the lungs -- activation of  $\beta$ -2 receptors causes bronchial vasodilation, increased respiratory secretions, and increased insulin production. The beta-receptors are closely linked with a protein that produces cAMP. The presence of cAMP results in glycogenolysis and vascular smooth muscle contraction, as well as positive inotropic, chronotropic, and dromotropic effects.

Propranolol (Inderal) is the most common beta-blocker and is like nadolol, oxprenolol, pindolol, sotalol and timolol in affecting both  $\beta$ -1 and  $\beta$ -2 receptors. Acebutolol, atenolol, esmolol, labetalol, metoprolol are more specific to the  $\beta$ -1 receptors.

*Betty Boom is found down in her apartment by her children, who call the paramedics. When the paramedics arrive, Betty is breathing 10 breaths/min and has a pulse of 41 and BP of 80 (palp). She is emergently intubated*

and given a 1 L bolus of NS while being transported to the hospital.

*In the ER, Betty was cool and dry, with normal heart and breath sounds. She is given 50 cc of D50, 100 mg of thiamine, 2 mg of naloxone, and 1 mg of flumazenil with no response. She is placed on a transcutaneous pacemaker after being given Atropine with no response. Her BP drops further with the pacing, which is then stopped. Her family brings in her medications and on examination, 1/2 of the bottle of propranolol, just refilled two days previously, is gone.*

Many patients with beta blocker overdoses (up to 1/3<sup>rd</sup>) are asymptomatic. If the person does have symptoms, he or she may exhibit:

- ◆ Bradycardia, including SA node abnormalities and AV block
- ◆ Impaired ability of the heart to respond to bradycardia, hypotension, decreased contractility, or vasodilation
- ◆ Delirium, coma and seizures in the hypotensive or normotensive patient
- ◆ Respiratory depression (especially with propranolol or atenolol)
- ◆ Hypoglycemia (usually in children)

The symptoms related to the overdose will start to be seen within six hours, and usually will be seen within two hours.

*Betty is intubated to protect her airway, and is given activated charcoal through an NG tube. Once the beta-blocking agent overdose is determined, Betty is given 3 mg of glucagon IV over one minute. One gram of calcium chloride is administered slow IV push. Betty's BP increases to 90/55 and her HR increases to 54.*

People with beta-blocker overdoses are not encouraged to vomit, and may not be lavaged, especially if the overdose occurred more than one hour previous to admission. Both vomiting and lavage can stimulate the vagus nerve, making the problems with bradycardia and hypotension even worse. Some practitioners recommend giving Atropine before lavage. Ventricular pacing can be tried; however, it is usually not helpful and may even drive the BP down, because the increased heart rate does not increase the cardiac output.

The treatments of choice for beta blocker overdose include:<sup>16</sup>

1. **Glucagon** 2-5 mg over 30 - 60 seconds. Glucagon increases cAMP by bypassing the beta receptor pathways.
2. **Calcium chloride** can also reverse hypotension, and is given 1 gm (10% solution) slow IV push.
3. High dose (really high dose!) **catecholamine infusions**. The idea is that by giving excessive amounts of epinephrine/norepinephrine-like drugs, the sheer volume of catecholamine will bind with the beta-receptors, causing increased effect. Isoproterenol, dopamine, dobutamine, and epinephrine have all been given. There are no hard data to support the use of one catecholamine over the other. Amrinone or Milrinone (both phosphodiesterase inhibitors) may also be attempted. Hemodialysis is not effective in removing any of the beta-blockers except atenolol.

*Betty is started on an Isoproterenol drip and slowly exhibits an increase in BP and HR. Seventy-two hours later, Betty is awake and oriented. Her propranolol is discontinued, and a community health nurse is contacted for continuing care.*

## CALCIUM CHANNEL BLOCKING AGENTS

According to the 1998 TESS study, there were over 8,600 episodes of toxic exposure to calcium channel blocking agents. Of those, 277 suffered major outcomes, and 77 people died. The majority of the exposures were unintentional.<sup>17</sup>

Calcium channel blocking agents act by blocking the entry of calcium into myocardial and smooth muscle cells. Because calcium can't enter the mechanical or electrical cells in the heart, cardiac rate and contractility decreases. Because calcium can't enter the smooth muscle cells in the blood vessels, vasoconstriction is limited. Calcium channel blockers are used for patients with angina (to decreased cardiac oxygen demand), and in hypertension.

### What are the symptoms?

Early in the course of the overdose, patients may have dizziness, fatigue, and nausea. As more of the drug is

absorbed, or with larger doses, lethargy, syncope, and coma are seen as a result of the cardiovascular effects of the drug. Profound bradycardia (including AV blocks, junctional rhythm, and idioventricular rhythms) are seen in conjunction with hypotension. The hypotension is partly due to the bradycardia, and partly from vasodilation. Cardiogenic shock results.

### What is the treatment?

After initial decontamination with lavage, the patient is given a dose of activated charcoal. With sustained-release ingestion, whole bowel irrigation may be used.

Although Atropine is the first line of treatment for any bradycardia, it is not effective for calcium channel blocker overdose. The treatment of choice is calcium. The recommendation is that poisoned adults should receive 13-25 mEq of calcium (10-20 mL of 10% CaCl<sub>2</sub> or 30-60 cc of 10% calcium gluconate) as an IV bolus, followed by repeated doses every 15-20 minutes to a maximum of 4 doses. A continuous infusion of 0.5 mEq/kg/hour may also be utilized.

***Be sure to check the dosage on the type of calcium used -- calcium chloride has three times more calcium than calcium gluconate!***

Finally, if calcium is ineffective, epinephrine, norepinephrine, dopamine, isoproterenol, and dobutamine may be attempted. None of these drugs have been proven to be most effective. Other therapies that may be tried include glucagon administration, amrinone infusion, transvenous pacing, intra-aortic balloon pumping, and cardiopulmonary bypass. None of these therapies have been found to be especially effective.

## COMMON POISONS

### ETHANOL

*Jason S. is a college student who just celebrated his 21<sup>st</sup> birthday with friends. Part of the birthday bash included having Jason drink as many shots as he was years old. Jason reached number 20 when he stood up, staggered a few steps, and vomited as he hit the floor. The paramedics were called and Jason was rushed to the hospital. In the ER, Jason was unconscious, breathing*

*shallowly at a rate of 10/minute, and had decreased reflexes. His blood alcohol level was 620 mg/dL.*

The most common drug of abuse in the United States is alcohol. Alcohol toxicity (poisoning) accounted for over 30,000 toxic exposures, 20,544 hospitalizations, and 42 deaths, according to the 1998 TESS study. Thirty-eight percent of all traffic fatalities and 47 - 64% of adult drownings are associated with alcohol.<sup>18</sup>

### When Fun Stops and Toxicity Starts

At a blood alcohol content (BAC) level of 300 mg/dl (.30%), a person is experiencing slurred speech, sensory loss, and visual disturbances. He is confused and may be nauseated. At 400 mg/dl (0.40 %), the person is prone to hypothermia, poor muscle control, poor recall, and seizures. Death may occur at 700 mg/dl (0.70 %), where respiratory depression, decreased reflexes, and unconsciousness predominate.<sup>19</sup>

### Treating Acute Poisoning

The first treatments are aimed at the ABC's -- airway, breathing, and circulation. Patients who are not able to protect their airway should be intubated, and potentially mechanically ventilated. Acutely intoxicated people may vomit and aspirate, so aspiration precautions should be followed. The BP and HR should be monitored closely.

Before administration of any IV fluid containing dextrose (D5W, D5NS, etc...), the patient should be given 100 mg of Thiamine IV. This will help prevent Wernicke's encephalopathy and Korsakoff's syndrome.

The patient should be monitored closely to protect safety. Physical restraints or 1:1 observation may be necessary until the patient arouses to a clear level of consciousness.

All other care is supportive. The patient should not be released from the hospital until the BAC is 0.0; a hold may be necessary. Intensive education should be done with this patient to prevent further recurrences.

### Treating Chronic Toxicity

Chronic alcoholics have additional problems with malnutrition and liver failure. Treatment is the same initially as the acute poisoning, but additional steps are needed.

Because chronic alcoholics are usually malnourished, they need replacement of magnesium, multi-vitamins, folate, potassium, and phosphorus. Liver failure may cause clotting problems, so FFP and vitamin K may be indicated. If the liver failure has progressed to encephalopathy, the patient will need to be treated with cleansing enemas, lactulose, or neomycin to decrease serum ammonia levels.

*Jason is awake and oriented the next morning. He is extubated and placed on 2 L/O<sub>2</sub>/n.c. Jason is very embarrassed to find himself in the hospital and vows "never to drink again!" Post-hospitalization follow-up is planned with the college's health services program.*

## THE OTHER ALCOHOLS

*Harry Z. is a long-time alcoholic who has been admitted to your unit many times. He typically is admitted for either alcohol toxicity (poisoning) or withdrawal. This time, however, the ER calls up the report that Harry has been drinking isopropyl alcohol because he ran out of money for his drink of choice. He came into the hospital because of severe abdominal pain.*

Ethanol is the most common of the alcohols that people drink, but it's not the only alcohol! By accident or by design, people can ingest isopropyl (rubbing) alcohol, methanol (wood alcohol), or ethylene glycol (antifreeze).

### Isopropyl Alcohol

People who are poisoned by rubbing alcohol have multiple-system symptoms. GI symptoms include severe abdominal pain from gastritis, cramping, and hematemesis. Neurologically, the alcohol can cause ataxia, areflexia, dizziness, headache, and muscle weakness. Finally, alcohol poisoning can result in hypotension, bradypnea, and hypothermia. Lab tests will show ketones in the blood and urine, but the pH, HCO<sub>3</sub><sup>-</sup> and PCO<sub>2</sub> will be normal.

*Harry is admitted to the unit and given supportive care. His lab values show consistently high levels of isopropyl alcohol, and the physicians decide to hemodialyze Harry.*

## Methanol

The potentially fatal dose of methanol is 30 mL of a 40% solution -- although fatalities have been reported with 15 mL.

The breakdown product of methanol is initially formaldehyde, which is further metabolized into formate. This metabolite accumulates and causes metabolic acidosis. Visual disturbances are typical for methanol poisoning, including blindness, blurred vision, and dimmed vision (like a snowstorm). The patient may seem to be inebriated, and may have seizures, dizziness, headache, nausea, vomiting, and abdominal pain.

## Ethylene Glycol

Ethylene glycol is metabolized into glycolate, which causes the metabolic acidosis seen in these patients, and calcium oxalate precipitate. Symptoms include decreased level of consciousness, lethargy, seizures, slurred speech, coma, abdominal pain, nausea, and vomiting. Lab results show hypoglycemia, metabolic acidosis, hypocalcemia, and calcium oxalate crystals in the urine. Renal failure may result.

### Treatment for Methanol and Ethylene Glycol Poisoning

Initial neutralization of the poisons is done with gastric lavage. Charcoal does not absorb either alcohol, so is not used unless there was a poly-drug overdose. Treatment is focused on maintaining hydration, maintaining a normal pH with sodium bicarbonate as necessary, and preventing methanol and ethylene glycol from being metabolized by using an ethanol drip.

To compete for the metabolism sites, an ethanol drip may be started to maintain the blood level at 100 - 150 mg/dL. This prevents liver and kidney damage and neurological changes caused by the metabolites of both of these substances. The ethanol drip is continued until the blood level of the toxic agent decreases to a safer level (<20 mg/dL), which may occur over several days. Hemodialysis may also be considered.

Fomepizole, a drug recently approved by the FDA, inhibits alcohol dehydrogenase, and was developed specifically to treat ethylene glycol poisoning. The Ad Hoc Committee on the Treatment Guidelines for Ethylene Glycol Poisoning recommends that fomepizole may be

given IV every 12 hours to prevent renal damage and metabolic problems related to ethylene glycol poisoning.<sup>20</sup>

Finally, thiamine 100 mg IV and pyridoxine 50 mg PO are given every six hours.

## ORGANOPHOSPHATES

*Tom J. is a 43-year-old farmer who is admitted to the ER via ambulance. He had one tonic-clonic seizure en route and is flaccid and non-responsive at this time. He was emergently intubated and is being bagged with 100% oxygen. Copious secretions are draining from his mouth; he is being suctioned frequently through the endotracheal tube for copious clear white secretions.*

The organophosphates bind with acetylcholinesterase, which is the chemical that stops the action of acetylcholine. The actions of acetylcholine just go on and on, producing the following symptoms:

- S:** salivation
- L:** lacrimation
- U:** urination
- D:** defecation
- G:** gastric cramping
- E:** emesis

Tom has been poisoned with an organophosphate insecticide, probably through skin contact with the dust on the crops and his clothing. Be aware that patients with pesticide toxicity often have the dust still on their clothing. You should take care to handle all clothing as hazardous material, so that you aren't exposed to the same thing!

*Tom is placed on a mechanical ventilator and is oxygenated at 60% with adequate ABG's. He is given 1 mg Atropine IV initially, followed by 1 gram of pralidoxime.*

Atropine is an anti-**cholinergic** drug that stops the action of acetyl**choline**. Pralidoxime (Protopam<sup>®</sup>) is a drug that reactivates cholinesterase, so that the cholinesterase can attach to acetylcholine and stop its action. Repeated doses of both atropine and pralidoxime may be necessary.

Further treatment of the patient with organophosphate poisoning is symptomatic: airway management, removal of secretions, correction of acidosis, and maintenance of adequate PaO<sub>2</sub>.

## POISONING FROM ILLEGAL DRUGS

---

Coke, horse, ice, X-TC, and angel dust: they are all names for some of the most dangerous drugs in use today. This section will describe the use of each of these drugs, along with the effects, symptoms of overdose, symptoms of withdrawal, and treatment.

### COCAINE

In 1997, the Office of National Drug Control Policy estimated that there were 3.6 million chronic cocaine users in the United States. Of those, approximately 604,000 used crack cocaine.<sup>21</sup> Let's put that into perspective. That's the same as every man, woman, and child in the state of Oregon using cocaine, and every person in the state of North Dakota using crack cocaine.

#### What cocaine is...

Cocaine is a drug that is extracted from the coca bush, found primarily in Peru and Bolivia. It has been used for thousands of years, and abused for more than 100. There are two forms of cocaine: the salt and "freebase," which has not been neutralized by an acid.

The salt form of cocaine can be dissolved in water and taken either intravenously or intranasally. The freebase form can be smoked. Crack cocaine is a freebase form of cocaine that has been processed from the salt form into a smokable form. The processing usually involves ammonia or sodium bicarbonate, water, and heat. "Crack" is the crackling sound that the substance makes when smoked.

The faster the drug enters the bloodstream, the faster the effect. The fastest "high" will be obtained by smoking. On the other hand, the faster the high, the shorter the

duration of the high. The duration of the high can be as little as five minutes and as long as 10-20 minutes.

### Why do people use cocaine?

Cocaine gives people a sensation of euphoria, relaxation, and being carefree. They feel as if they are in control and are powerful. The sensations come from the action of the cocaine on the neurotransmitter dopamine in a region in the brain called the ventral tegmental area (VTA). This area of the brain is connected to the brain's key pleasure center. The cocaine does not allow the dopamine to be recycled, so the stimulation of the pleasure center goes on longer than normal.

### So what's so bad about it?

Larger amounts of cocaine (several hundred milligrams and up) intensify the high, but lead to violent, bizarre, or erratic behavior, tremors, muscle twitches, paranoia, and anxiety. With these neurological changes come the cardiovascular effects of cocaine: increased blood pressure, heart rate, constricted blood vessels, and increased temperature.

*The first dose -- or any dose after -- can kill. Most deaths are the result of cardiac arrest or respiratory arrest caused by seizures.*

### In the End...

Here are the medical consequences of using cocaine:

- Cardiac: Dysrhythmias, hypertension, heart attacks
- Pulmonary: respiratory failure
- Neurological: strokes, seizures, headaches
- GI: abdominal pain, nausea

### What are the symptoms of cocaine overdose?

Although cocaine users may enter the health care system because of related problems, such as a heart attack, many other people need care directly related to an overdose of cocaine. Here is a case of one such person:

*Jeremy I. is a 26-year-old male who is admitted to the MICU, under arrest for suspicion of cocaine dealing. According to police, they came across Jeremy in the midst of suspicious activity. When the police were seen, Jeremy*

*swallowed a packet of unknown substance. His physical examination in your unit is:*

*Cardiovascular: HR 115; ST. BP 146/88. Denies chest pain.*

*Pulmonary: RR 24. Lung sounds clear. SaO2 99%.*

*Neurological: Agitated, moving restlessly in bed. Denies using cocaine, but has smoke odor to breath. Alert and oriented; responsive to commands. No auditory or visual hallucinations. He is shackled to the bed.*

*GI/GU: Abdomen soft and nontender. Bowel sounds active. No bowel movements. Voided X1 for 350 cc.*

*Toxicology screen: Positive for cocaine*

*One hour after admit to the unit, Jeremy began to yell loudly. His BP climbed to 198/110; his HR jumped to 168 in an SVT. Jeremy's RR was 34 and shallow. Jeremy screamed out "those bugs, the bugs, kill the bugs." He was agitated and attempting to get out of bed. He required four-point leather restraints for safety, and was restrained by six people.*

*Shortly after, Jeremy had a generalized tonic-clonic seizure with a respiratory arrest. He was emergently intubated and given a loading dose of Dilantin and IV diazepam. His skin after the seizure was clammy, although his temperature was 103 degrees F.*

*Twenty-four hours after admission, Jeremy was alert and oriented. His heart rate was 100; BP 140/76. He admitted to ingesting a packet of cocaine to avoid arrest. A small baggy was excreted rectally in the morning. Jeremy was transferred to a treatment center for detoxification and withdrawal before appearing in court on charges of cocaine possession and intent to distribute.*

The major manifestations for cocaine toxicity are:

1. Hyperthermia from increased motor activity and vasoconstriction (the body can't get rid of the heat it generates)
2. Anxiety and agitation, potential for seizures, CVA's, and varied psychiatric manifestations

3. Myocardial ischemia and infarction related to atherosclerosis and vasospasm
4. Tachydysrhythmias, including ventricular tachycardia and ventricular fibrillation

### Treatment for Cocaine Overdose

Because of the incredibly fast action of cocaine, lavage is rarely indicated. In a large overdose, activated charcoal may be given. The treatment for "body packing" and "body stuffing" is different. Body packing is when a person swallows or inserts an object filled with an illegal drug with the intent of smuggling it. "Body packers" usually place the drug in latex or plastic to avoid breaking open during transit.

Body stuffing usually occurs when a person is attempting to avoid arrest and swallows the object. No extra care is taken to avoid rupture. With both types of ingestion, the goal is to get the packet out of the person as soon as possible. Activated charcoal and/or whole bowel irrigation are tried first. In more extreme cases, the packet may be removed endoscopically or surgically.

After neutralizing as much cocaine as possible, the main goal is to decrease the psychomotor agitation -- uniformly, drugs such as the benzodiazepines are used to sedate the patient. Diazepam (Valium), 5-10 mg IV, or lorazepam (Ativan) 2-4 mg IV, may be given initially and on a PRN basis to keep the patient calm. This should decrease the hyperthermia, anxiety, and agitation. Other measures for hyperthermia include oxygen administration, ice water baths, and a cool environment with minimal activity.

Cocaine users, often between 19 and 40 years of age, can experience atypical chest pain hours to days after their last use. The 12-lead will show non-specific ST segment elevation and T wave inversion that persists. Although definitive treatment for cocaine related chest pain is not known, research has shown that propranolol (Inderal), and probably esmolol, will cause further problems. Vasodilating agents such as nitroglycerine or nitroprusside can be used for hypertension.

Atrial tachydysrhythmias that do not respond to benzodiazepines may respond to one of the calcium channel blockers like verapamil or diltiazem. For ventricular dysrhythmias, lidocaine is used. Lidocaine will replace cocaine at the heart's sodium channels.

## HEROIN

Heroin is a narcotic that is processed from morphine. Morphine is a derivative of the opium poppy. About 600,000 people in the U.S. need treatment for heroin addiction -- a trend that seems to be on the increase, particularly among younger people. The National Institute on Drug Abuse reported that there was a 19 percent increase in heroin-related emergency department visits between 1994 and 1995. The Drug Enforcement Agency reports that there were 81,000 new users in 1997 -- nearly all of them under 26 years of age.

### Why do people use heroin?

With a single dose -- either injected, sniffed, or smoked -- the user feels a surge of euphoria -- a "rush". This effect, combined with a warm feeling in the skin, dry mouth, and heavy extremities, lasts for several hours. After this, the user has a period where he alternates between sleepiness and wakefulness -- "on the nod."

### The long term effects of heroin use...

Heroin is a drug that has been around for a very long time, and has been abused by some very famous people. Chronic users can expect to develop collapsed veins, endocarditis, abscesses, cellulitis, and liver disease from the drug itself. Pneumonia can occur because of poor health as well as the respiratory depressant effect of heroin.

Chronic users develop an addiction to heroin quickly. With addiction and physical dependence comes tolerance. The user needs more and more of the drug to achieve the same kind of rush; in the later stages, the user needs more and more just to keep from withdrawing.

### Emergency! Emergency!

A single dose of heroin can cause fulminant non-cardiogenic pulmonary edema. The alveoli fill with fluid and prevent gas exchange from occurring. The person can develop extreme hypoxemia and may die if not treated. The person who experiences this will present with shortness of breath (if not mentally obtunded), a low PaO<sub>2</sub> and SaO<sub>2</sub>, and copious amounts of pink, frothy sputum.

The person with a heroin overdose can experience a number of symptoms, all classic for opioid toxicity:

- Mental status depression

- Hypoventilation
- Miosis (abnormal pupillary constriction)
- Reduced bowel motility

### Treatment for Heroin Overdose

Naloxone (Narcan) is an opioid antagonist that is commonly used for actual or suspected heroin overdose. Naloxone competes with the opioid for receptor sites, and will effectively reverse the symptoms of an opioid. Giving naloxone to a person addicted to heroin has risks. Abrupt reversal can lead to abrupt withdrawal, causing agitation, hypertension, and tachycardia. In some research, naloxone administration has been implicated in causing non-cardiogenic pulmonary edema. If deemed necessary, a small dose of naloxone will be given as a bolus (0.1-0.4 mg), and possibly continued as an infusion.

### Withdrawal from Heroin

Withdrawal from heroin can occur as early as two hours after the last use; symptoms peak in 48 to 72 hours and may last as long as one week. Withdrawal from heroin is not considered as dangerous as withdrawal from alcohol, but still can be fatal.

The symptoms of withdrawal are:

- ◆ drug craving
- ◆ watery eyes and runny nose
- ◆ muscle and abdominal cramps
- ◆ restlessness and irritability
- ◆ muscle and bone pain
- ◆ insomnia
- ◆ loss of appetite, diarrhea and vomiting
- ◆ cold flashes with goose bumps -- "cold turkey"
- ◆ kicking movements -- "kicking the habit"

## AMPHETAMINE/ METHAMPHETAMINE

In 1998, the Toxic Exposure Surveillance System (TESS) study reported over 15,000 toxic exposures to amphetamines, including 31 deaths.<sup>22</sup> An estimated 4.7 million people had used methamphetamine in 1998.<sup>23</sup> Although both amphetamines and methamphetamine can be prescribed legally for weight reduction, attention-deficit disorder, and narcolepsy, the majority of toxic exposures come from illegal use.

### What do amphetamines do?

No one knows exactly what the mechanism of action for amphetamines is, but most research indicates that amphetamines cause the release of dopamine and block the re-uptake of the catecholamines (epinephrine and norepinephrine). Amphetamines can cause the following effects:

- Increased alertness
- Excitation
- Euphoria
- Tachycardia
- Hypertension
- Insomnia
- Anorexia

### Methamphetamine = Ice, Speed, Crystal, Meth, Crank

"Ice" is very similar to amphetamine in structure. It is the one of the top drugs of abuse, particularly on the West Coast and in the Midwest. Why? Because methamphetamine is easily and cheaply produced in "home" labs. Ice can be made very pure -- 80-90% -- leading to an increased effect. Ice can be snorted, smoked, injected, or taken orally. Crystal meth is smoked much like crack cocaine.

Beyond the "usual" effects of an amphetamine, ice can cause hyperthermia, pupil dilation, paranoia, and violent behavior. Violent and erratic behavior is seen with high-dose, chronic abusers, especially those who binge and have been without sleep for 3-15 days.

### MDMA = 3,4-methylenedioxyamphetamine = Ecstasy

MDMA is another form of methamphetamine which is mixed with a hallucinogen (usually mescaline). It is taken orally as a tablet or capsule. MDMA lasts between 4 and 6 hours. The effects of MDMA are slightly different: people say they are more empathetic, more positive, have decreased anxiety, and are very relaxed. Users can also go for days without eating, drinking, or sleeping, leading to severe dehydration and exhaustion. MDMA users may first take the drug at a "rave." A rave is an all night (or longer!) party. The adverse effects include nausea, hallucinations, chills, sweating, hyperthermia, tremors, muscle cramping, blurred vision, anxiety (after use), and paranoia.

An overdose of MDMA can result in heart failure or heat stroke from hypertension, faintness, confusion, panic attacks, loss of consciousness, seizures, and hyperthermia.

### What are the clinical manifestations of toxicity?

• hypertension	• anorexia
• tachycardia	• choreoathetoid movements
• dysrhythmias	• hyperreflexia
• myocardial ischemia	• paranoid psychosis
• vasospasm	• diaphoresis
• hyperthermia	• tachypnea
• agitation	• mydriasis
• seizures	• tremor
• intracerebral hemorrhage	• nausea
• headache	• rhabdomyolysis
• euphoria	• muscle rigidity
	• pulmonary edema

### What is the treatment for amphetamine toxicity?

After initial assessment and neutralization, the patient is treated symptomatically. Benzodiazepines (particularly diazepam) are given to decrease agitation and the potential for seizures. Barbiturates may be necessary to stop seizures.

The patient may be severely hyperthermic. A rectal temperature should be taken on a routine basis, especially if the patient continues to be very agitated. Cooling blankets or tepid water baths can also help bring down the temperature. Rhabdomyolysis may result from both agitation and hyperthermia. The patient should have adequate IV hydration and monitoring for a urine output of 1-2 ml/kg/hour.

The patient who is still experiencing the effects of toxicity is probably a danger to him or herself. Physical restraints may be necessary, as well as side-rail padding. Special care should be taken to monitor the skin and circulation around the restraints, as the person may not recognize when he or she is injuring him or herself.

If the patient is hallucinating, haloperidol (Haldol) or droperidol may be given after a benzodiazepine. These neuroleptic agents antagonize some of the effects of amphetamines.

## FOR MORE INFORMATION

---

The table at the end of this booklet, "Commonly Abused Drugs," is another resource for you to review as you have time. The potential for dependence, routes of administration, duration of action, signs of overdose, and management of overdose are all briefly outlined.

There are several websites and books that have a lot of information on alcohol and drug toxicity:

- National Institutes on Drug Abuse (NIH) at [www.nida.nih.gov](http://www.nida.nih.gov)
- U.S. Department of Justice -- Drug Enforcement Administration Website at [www.usdoj.gov/dea](http://www.usdoj.gov/dea)
- National Institute on Alcohol Abuse and Alcoholism at [www.niaaa.nih.gov](http://www.niaaa.nih.gov)
- The National Clearinghouse for Alcohol and Drug Information at [www.health.org](http://www.health.org)
- The American Association of Poison Control Centers at [www.aapcc.org](http://www.aapcc.org)
- Goldfrank, L. R. et al. (2002). Goldfrank's Toxicologic Emergencies, McGraw-Hill.
- Leikin, J.B. & Paloucek, F.P. (1999). Poisoning and Toxicology Compendium, Lexi-Comp, Inc.: Hudson, OH.
- Olson, K.R. (2004). Poisoning and Drug Overdose. New York: Lange Medical Books/McGraw-Hill.

## SUMMARY

---

The statistics are all there -- the health care practitioner can expect to see patients who are experiencing problems with drug toxicity at some point. It is vital that you understand the priorities in assessing and managing the patient with drug toxicity for that patient who will hit your door. This independent-learning program was designed to help you understand some of the different toxins that patients may present with in the health care setting. The assessment, neutralization of the toxin, and management of selected toxins were described.

---

## REFERENCES

---

1. The Economic Costs of Alcohol and Drug Abuse (1992). Executive Report for the National Institutes on Drug Abuse and Alcohol Abuse and Alcoholism.
2. Goldfrank, L. R. et al. (1998). Goldfrank's Toxicologic Emergencies, Appleton and Lange: Stamford, page 366.
3. Ibid, page 366
4. Ibid, page 369.
5. Ibid, page 359.
6. Ibid, page 362.
7. Ibid, page 356.
8. Viccellio, P. (1998). Emergency Toxicology (2nd ed.). Lippincott-Raven Publishers: Philadelphia, page 64.
9. Ibid, page 67.
10. Linden, C.H. & Rumack, B.H. (1984). Acetaminophen overdose, Emergency Medicine Clinics of North America, 2, 103-119.
11. Goldfrank, L. R. et al. (1998). Goldfrank's Toxicologic Emergencies, Appleton and Lange: Stamford, page 929.
12. Litovitz, T.L., et.al. (1999). 1998 Annual report of the American Association of Poison Control Centers Toxic Exposure Surveillance System, The American Journal of Emergency Medicine, 17(5), 435-487.
13. Goldfrank, L. R. et al. (1998). Goldfrank's Toxicologic Emergencies, Appleton and Lange: Stamford, pages 570-571.
14. Ibid, page 576.
15. Litovitz, T.L., et.al. (1999). 1998 Annual report of the American Association of Poison Control Centers Toxic Exposure Surveillance System, The American Journal of Emergency Medicine, 17(5), 435-487.
16. Goldfrank, L. R. et al. (1998). Goldfrank's Toxicologic Emergencies, Appleton and Lange: Stamford, pages 818-819.
17. Litovitz, T.L., et.al. (1999). 1998 Annual report of the American Association of Poison Control Centers Toxic Exposure Surveillance System, The American Journal of Emergency Medicine, 17(5), 435-487.
18. Ibid.
19. Fourth Special Report to Congress on Alcohol and Health, U.S. Department of Health and Human Services, 1981.
20. Barceloux, D.G. (1999). American Academy of Clinical Toxicology practice guidelines on the treatment of ethylene glycol poisoning, Journal of Toxicology-Clinical Toxicology, 37(5).
21. Office of National Drug Control Policy.
22. Litovitz, T.L., et.al. (1999). 1998 Annual report of the American Association of Poison Control Centers

Toxic Exposure Surveillance System, The American Journal of Emergency Medicine, 17(5), 435-487.

23. 1998 National Household Survey on Drug Abuse, National Institutes of Health.

## DIRECTIONS FOR SUBMITTING YOUR POST TEST FOR CONTACT HOURS

To obtain a certificate of completion for this home study program, please complete the post-test and evaluation on the next few pages. The date on your certificate of completion will be the date that your home study is received. **Any materials received with a postmark after the expiration will be discarded.**

### **HealthEast, HCMC, & MVAMC Employees**

If you are an employee of HealthEast, HCMC, or MVAMC, you may send the post-test and evaluation to TCHP for processing. Your post-test will be returned to you through your hospital. It cannot be mailed to your home.

### **Paid Participants**

If you are not an employee of one of the TCHP hospitals, please send the post-test and evaluation to TCHP with a check for \$12.00. Please make check payable to **TCHP Education Consortium** and mail to:

**TCHP Education Consortium  
Capitol Office Building  
525 Park Street, Suite 120  
St. Paul, MN 55103**

Your post-test will be returned to you with the certificate of completion.



## COMMON DRUGS OF ABUSE

Drug	Dependence Physical/Psychological	<i>O = oral</i>	<i>S = smoked</i>	<i>I = injected</i>	Treatments
		How Used	Duration (hours)	Signs of Overdose	
Narcotics					
Opium	High/High	O, S	3-6	<ul style="list-style-type: none"> <li>• Slow, shallow breathing</li> <li>• Clammy skin</li> <li>• Seizures</li> <li>• Coma</li> <li>• Constricted pupils (miosis)</li> <li>• Non-cardiogenic pulmonary edema</li> </ul>	<ol style="list-style-type: none"> <li>1. Gastric lavage</li> <li>2. MDAC</li> <li>3. Respiratory support</li> <li>4. Naloxone cautiously</li> </ol>
Morphine	High/High	O, S, I	3-6		
Codeine	Mod/Mod	O, I	3-6		
Heroin	High/high	S, I, Sniffed	3-6		
Hydromorphone	High/High	O, I	3-6		
Meperidine	High/High	O, I	3-6		
Methadone	High/High	O, I	12-24		
Depressants					
Barbiturates	High/Mod	O	1-16	<ul style="list-style-type: none"> <li>• Tense vesicular skin lesions (6%)</li> <li>• Nystagmus</li> <li>• Fluid overload</li> <li>• Cardiorespiratory depression</li> <li>• Depressed reflexes</li> <li>• Prolonged coma</li> </ul>	<ol style="list-style-type: none"> <li>1. Respiratory support</li> <li>2. Gastric lavage</li> <li>3. MDAC</li> <li>4. Flumazenil for benzodiazepine toxicity</li> <li>5. Urinary alkalization for phenobarbital toxicity</li> </ol>
Benzodiazepines	High/High	O	1-8	<ul style="list-style-type: none"> <li>• Muscle relaxation</li> <li>• Decreased LOC</li> <li>• Respiratory depression</li> </ul>	
Methaqualone	High/High	O	4-8	<ul style="list-style-type: none"> <li>• Increased reflexes</li> <li>• Tonic-clonic spasms</li> <li>• Prolonged coma</li> <li>• Internal bleeding</li> <li>• Depressed platelet function</li> <li>• Slow respirations</li> </ul>	
Chloral hydrate	Mod/Mod	O	5-8	<ul style="list-style-type: none"> <li>• Pear-like odor</li> <li>• Cardiac arrhythmias</li> <li>• Opacities on abd. X-ray</li> </ul>	

Drug	Dependence Physical/Psychological	<i>O = oral</i>		<i>S = smoked</i>	<i>I = injected</i>	Treatments
		How Used	Duration (hours)	Signs of Overdose		
Stimulants						
Cocaine	Possible/High	S, I, Sniffed	1-2	<ul style="list-style-type: none"> <li>• Dilated pupils</li> <li>• Tachycardia</li> <li>• Hypertension</li> <li>• Hallucinations/psychosis</li> </ul>	<ol style="list-style-type: none"> <li>1. Respiratory support</li> <li>2. Gastric lavage</li> <li>3. MDAC</li> <li>4. Whole bowel irrigation for packets of drug</li> <li>5. Benzodiazepines for s/s of agitation</li> <li>6. Management of hyperthermia</li> <li>7. Vasodilators (NTG, nitroprusside) for hypertension</li> </ol>	
Amphetamines	Possible/High	O, I	2-4	<ul style="list-style-type: none"> <li>• Toxic psychosis/hallucinations</li> <li>• Hyperthermia</li> <li>• Hypertension</li> <li>• Seizures</li> <li>• Tachycardia</li> </ul>		
Ice	High/High	O, I, S, inhaled	4-14	<ul style="list-style-type: none"> <li>• Toxic psychosis/hallucinations</li> <li>• Hyperthermia</li> <li>• Hypertension</li> <li>• Seizures</li> <li>• Tachycardia</li> </ul>		
Hallucinogens						
PCP	Unknown/High	S, O	Up to days injected	<ul style="list-style-type: none"> <li>• Hallucinations, psychosis</li> <li>• Dizziness, confusion, anxiety</li> <li>• Extreme changes in behavior</li> <li>• Hypertension</li> <li>• Longer, more intense "trips"</li> <li>• Seizures, coma, death</li> </ul>	<ol style="list-style-type: none"> <li>1. Respiratory support</li> <li>2. Gastric lavage</li> <li>3. MDAC</li> <li>4. Supportive care</li> </ol>	
LSD	None/Unknown	O	8-12			
Mescaline/Peyote	None/Unknown	O, I	8-12			
Psilocybin	None/Unknown	O, I, S, Sniffed	Variable			
Ecstasy	Unknown/Unknown		Variable			

Drug	Dependence Physical/Psychological	<i>O = oral</i>	<i>S = smoked</i>	<i>I = injected</i>	Treatments
		How Used	Duration (hours)	Signs of Overdose	
Alcohols					
Ethanol	Possible/Possible	O	1-4	<ul style="list-style-type: none"> <li>• Depressed LOC</li> <li>• Respiratory depression</li> <li>• Delayed deep tendon reflexes</li> </ul>	<ol style="list-style-type: none"> <li>1. Respiratory support</li> <li>2. Thiamine 100 mg IV</li> <li>3. Supportive care</li> </ol>
Methanol	None	O		<ul style="list-style-type: none"> <li>• Blindness</li> <li>• Blurred vision</li> <li>• Snowstorm vision</li> <li>• Seizures</li> <li>• Dizziness</li> <li>• Headache</li> <li>• Abdominal pain</li> </ul>	<ol style="list-style-type: none"> <li>1. Gastric lavage if ingestion within minutes</li> <li>2. Ethanol drip</li> <li>3. Thiamine 100 mg IV</li> <li>4. Pyridoxine 50 mg PO every 6 hours</li> </ol>
Ethylene Glycol	None	O		<ul style="list-style-type: none"> <li>• Decreased LOC</li> <li>• Lethargy</li> <li>• Seizures</li> <li>• Slurred speech</li> <li>• Abdominal pain</li> <li>• Nausea/vomiting</li> <li>• Calcium oxalate crystals in urine</li> </ul>	<ol style="list-style-type: none"> <li>1. Respiratory support</li> <li>2. Ethanol IV drip or fomepizole IV every 12 hours</li> <li>3. Thiamine 100 mg IV</li> <li>4. Pyridoxine 50 mg PO every 6 hours</li> </ol>

# Post -Test: Drug Overdose

Please print all information clearly and sign the verification statement:

Name \_\_\_\_\_  
(please print legal name above)

**Birth date (required)**

Format: 01/03/1999

M	M	D	D	Y	Y	Y	Y

*For HealthEast, HCMC, or MVAMC, employees only:*

Hospital \_\_\_\_\_ Unit \_\_\_\_\_

**Personal verification of successful completion of this educational activity (required):**

*I verify that I have read this home study and have completed the post-test and evaluation.*

\_\_\_\_\_  
Signature

- 1) Which drug does not cause AV conduction abnormalities?
  - a) epinephrine
  - b) calcium channel blocking agent
  - c) beta blocking agent
  - d) digoxin
- 2) Which of the following statements about gastric lavage is true?
  - a) a 40 french tube is appropriate for an adult.
  - b) a 30 french tube is appropriate for a child.
  - c) lavage is contraindicated when the patient has a decreased LOC and has ingested lye.
  - d) all of the above
- 3) Which of the following toxins is NOT eliminated by activated charcoal?
  - a) Acetaminophen
  - b) Diazepam
  - c) Methanol
  - d) Valproic acid
- 4) Which patient would have a higher chance of seizures following a tricyclic antidepressant overdose?
  - a) The patient with hallucinations
  - b) the patient with a QRS complex > 160 msec
  - c) the patient with a serum level of < 1,000
  - d) none of the above
- 5) Sodium bicarbonate is given for what reason in a cyclic antidepressant overdose?
  - a) to diminish the effect of the antidepressant on the myocardium
  - b) to correct metabolic acidosis
  - c) to prevent seizures
  - d) to interact with dopamine
- 6) What is the greatest risk of giving flumazenil to the patient who has benzodiazepine toxicity?
  - a) re-sedation
  - b) withdrawal symptoms
  - c) a seizure
  - d) pain
- 7) What acid-base derangement is seen with a salicylate overdose?
  - a) mixed respiratory alkalosis and metabolic acidosis
  - b) respiratory alkalosis late in the course
  - c) metabolic acidosis with respiratory compensation
  - d) there is no acid-base problem
- 8) The treatment of choice for a calcium channel blocker overdose is:
  - a) dopamine
  - b) calcium
  - c) intra-aortic balloon pumping
  - d) atropine
- 9) Which of the following lab results will be seen in an isopropyl alcohol poisoning?
  - a) positive serum and urine ketones
  - b) elevated pH
  - c) decreased glucose
  - d) elevated serum sodium
- 10) The main symptoms of methanol poisoning are related to:
  - a) hearing
  - b) vision
  - c) smell
  - d) touch
- 11) Which of the following may be administered to the patient who has been poisoned with an organophosphate?
  - a) epinephrine
  - b) atropine
  - c) pralidoxime
  - d) b & c

- 12) Which of the following are the major manifestations of cocaine toxicity?
- a) hyperthermia
  - b) anxiety and agitation
  - c) tachydysrhythmias
  - d) all of the above
- 13) What drugs have been most effective in decreasing psychomotor agitation?
- a) barbiturates
  - b) tricyclic antidepressants
  - c) benzodiazepines
  - d) diltiazem
- 14) The signs and symptoms of heroin overdose include:
- a) non-cardiogenic pulmonary edema
  - b) mental status depression
  - c) miosis
  - d) all of the above
- 15) What is the opioid antagonist that may be given in a heroin overdose?
- a) Flumazenil
  - b) Atropine
  - c) Rogaine
  - d) Naloxone

**Expiration date:** The last day that post tests will be accepted for this edition is **December 31, 2017**—your envelope must be postmarked on or before that day.

## Evaluation: Drug Overdose

Please complete the evaluation form below by placing an "X" in the box that best fits your evaluation of this educational activity. Completion of this form is required to successfully complete the activity and be awarded contact hours.

At the end of this home study program, I am able to:	Strongly Agree	Agree	Neutral	Disagree	Strongly Disagree
1. Identify the scope of alcohol and drug abuse, overdose, and withdrawal in the United States.					
2. Describe the initial and ongoing assessment of the patient experiencing a drug or alcohol overdose.					
3. Describe the initial management of the patient who has overdosed on drugs or alcohol.					
4. Identify indications, contraindications, and methods of poison neutralization.					
5. Identify the contributing factors, signs and symptoms, and interventions for overdose on legal drugs and poisons.					
6. Describe the assessment and management of the person with acute or chronic alcohol poisoning.					
7. Identify desired effect of drug, signs, and interventions for overdose on selected illicit drugs.					
8. The teaching / learning resources were effective. <i>If not, please comment:</i>					

The following were disclosed in writing prior to, or at the start of, this educational activity (please refer to the first 2 pages of the booklet).

	Yes	No
9. Notice of requirements for successful completion, including purpose and objectives		
10. Conflict of interest		
11. Disclosure of relevant financial relationships and mechanism to identify and resolve conflicts of interest		
12. Sponsorship or commercial support		
13. Non-endorsement of products		
14. Off-label use		
15. Expiration Date for Awarding Contact Hours		
16. Did you, as a participant, notice any bias in this educational activity that was not previously disclosed? <i>If yes, please describe the nature of the bias:</i>		

17. How long did it take you to read this home study and complete the post test and evaluation:

\_\_\_\_\_ hours and \_\_\_\_\_ minutes.

18. Did you feel that the number of contact hours offered for this educational activity was appropriate for the amount of time you spent on it?

\_\_\_ Yes

\_\_\_ No, more contact hours should have been offered

\_\_\_ No, fewer contact hours should have been offered.

Expiration date: December 31, 2017
------------------------------------