



General Management of Poisoned Patients

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INTRODUCTION



- * Poisoning is a worldwide problem
- * U.S. poison control centers documented **2.38** million human exposures in 2010, with 1146 associated deaths
- * **Prevention** is the key to reducing unintentional poisoning deaths :
 - **Pharmacists** , **parents** , **Teachers** and **healthcare** providers ??



INTRODUCTION

* Exposures occur:

-ingestion

-injection

-inhalation

-cutaneous and mucous membrane exposure

-...



INTRODUCTION

Criteria's for **nontoxic** exposure are:

- (1) an **unintentional** exposure to a clearly identified single substance,
- (2) where an estimate of **dose** is known, and
- (3) a recognized information source (e.g., a poison control center) confirms the substance as **nontoxic**.

Asymptomatic patients with nontoxic exposures may be discharged after a short period of observation, providing they have access to further consultation and a safe discharge destination.

RESUSCITATION

- **Resuscitation** (first priority)
- a structured **risk assessment** (benefit from an antidote, decontamination, or enhanced elimination techniques)
- ✓ Treatment of cardiac arrest :
Advanced Cardiac Life Support + interventions

Patients : Young +minimal preexisting organ dysfunction



Prolonged resuscitation

- * Stabilization of **a**irway, **b**reathing, and **c**irculation represents initial priorities
- * **IV crystalloid** bolus (**10 to 20** mL/kg) is first-line treatment of hypotension
- * Persisting hypotension may respond to a specific **antidote**.
- * Otherwise, cautious administration of an **inotropic** agent is indicated. (knowledge of the toxin's toxicodynamic properties)

ANTIDOTES

- * Stabilization of airway, breathing, and circulation allows further assessment of **blood glucose** concentration, **temperature**, and **conscious** state.
- * Although the proper use of **antidotes** is important, only a few are indicated **before** cardiopulmonary stabilization :
 - **naloxone** for opiate toxicity,
 - **cyanide antidotes** for cyanide toxicity
 - **atropine** for organophosphate poisoning

HYPOGLYCEMIA

- * Treat hypoglycemia with **IV dextrose** (glucose).
- * **Altered mental status** when hypoglycemia cannot be excluded is an indication for IV dextrose.
- * Supplemental oxygen, thiamine, glucose, and naloxone are often administered empirically as a **cocktail** in cases of altered mental status. (not cost-effective)
- * The **decision** to administer an **antidote** should be made after a rapid collateral **history** is obtained and targeted **examination** completed.

CARDIAC ARRHYTHMIAS

- * Antiarrhythmic drugs are **not** first-line treatment for toxin induced arrhythmias.
- * Most toxin-induced arrhythmias respond to correction of **hypoxia**, **metabolic/acid–base** abnormalities, and administration of an **antidote** (e.g., digoxin Fab).
- * **Sodium bicarbonate** is administered for sodium-channel blocker toxicity with cardiovascular complications, such as **wide QRS** complex tachyarrhythmia's.
Ventricular tachyarrhythmia may respond to overdrive pacing

SEIZURES

- * Drug-induced seizures are treated with **IV benzodiazepines**
 - isoniazid-induced seizures require **pyridoxine**
 - hypoglycemia and hyponatremia, should be rapidly **excluded**.
- * **Barbiturates** are second-line agents for benzodiazepine-resistant seizures
- * There is no role for **phenytoin** in the treatment of toxin-induced seizures; it has neither theoretical nor proven **efficacy**, and may **worsen** toxicity

AGITATION

- * treated with titrated doses of **benzodiazepines**.
- * **antipsychotic** agents are often used as second-line agents for toxin-induced agitation (disadvantages, including anticholinergic and extrapyramidal effects)
- * **Droperidol** has been associated (rarely) with **QT** interval **prolongation** and cardiac arrhythmias

HYPERTHERMIA AND HYPOTHERMIA

- * temperatures of **>39°C** (>102.2°F) require aggressive active cooling measures to prevent complications such as **rhabdomyolysis**, **organ failure**, and **DIC**
- * Sedation, neuromuscular paralysis, and intubation are required if active measures are ineffective.
- * specific pharmaceutical agents:
 - sympathomimetic (**benzodiazepines**)
 - serotonin (**cypheptadine**)
 - neuromuscular malignant syndrome (**bromocriptine**).

- 
- * Drug-induced coma with subsequent immobility and environmental exposure or inherent drug toxicity (**opioids, phenothiazines, ethanol**) may produce **hypothermia**.
 - * A core temperature **<32°C** (<90°F) is an indication for active rewarming

NALOXONE

- * **Naloxone** is a nontoxic, diagnostic, and therapeutic antidote. (IV, IM, or intranasally)
- * Naloxone can be used as a **diagnostic** (**respiratory rate of <12** breaths/min) suggest possible opioid exposure.
- * Naloxone is titrated to clinical effect using bolus doses, typically **0.1 to 0.4** milligrams.
- * Large initial bolus doses may precipitate **vomiting** and **aspiration**, acute opioid **withdrawal**, or an **uncooperative, agitated** patient.
- * **Miosis** is an **unreliable** indicator of naloxone's adequate clinical effect

- * Doses are titrated to achieve desirable ventilation and conscious state (adequate **respiratory rate**, normal arterial oxygen **saturations** on **room air**, and **verbal** or **motor** response to **voice**).

- * Although naloxone may reverse the effects of opioids for **20** to **60** minutes, the effect of many opioids will outlast this time frame with possible return of **respiratory depression**.

- * Patients should be observed for **2 to 3 hours** after administration of IV naloxone

RISK ASSESSMENT

- * Following initial resuscitation and stabilization, a **risk assessment** is performed to **predict** course of clinical toxicity, **interventions** required, and patient **disposition**.
- * Risk assessment is formulated using **history**, **examination**, and ancillary **test results**.
- * Acute poisoning is a **dynamic** process; therefore, risk assessment may change with **time** and requires **ongoing review**

HISTORY

- * no clear history due to
 - **psychiatric** illness,
 - clinical **effects** of exposure
 - fear of **arrest** or **repercussions** from family or friends.
- * Information including identity of **substances**, **doses**, **route** of exposure , **time** , **Emesis** , **Reason** and **Symptoms** (from family, friends, previous medical records, and usual healthcare provider)

HISTORY

Prehospital emergency services can provide information regarding **empty medication containers** or the **scene** environment (**smells**, particular **materials** or substances present).

EXAMINATION

- * A systematic physical examination can yield important clues to the nature and potential severity of an exposure
- * Examine the **skin folds**, body cavities if appropriate, and **clothing** for retained **tablets** or substances

TABLE 176-3**Examination of the Poisoned Patient**

Organ System	Examination	Example of Finding (Possible Significance)
General appearance	General demeanor and dress Signs of injury Odors Mental state Nutritional state Temperature	Unkempt (psychiatric illness) Scalp hematoma (intracranial injury) Malnourished (IV drug use, HIV infection) Smell of bitter almonds (cyanide toxicity)
Central nervous	Conscious state Pupil size and reactivity Eye movements Cerebellar function/gait	Miosis (opioids, organophosphates, phenothiazines, clonidine intoxication) Nystagmus/ataxia (anticonvulsant and ethanol toxicity)

Cardiovascular	Heart rate/blood pressure Cardiac auscultation	Murmur (endocarditis/IV drug abuse)
Respiratory	Oxygen saturation Respiratory rate Chest auscultation	Fever/crepitations/hypoxia (aspiration pneumonia) Bronchorrhea/crepitations/hypoxia (organophosphate toxicity)
Gastrointestinal	Oropharynx Abdomen Bladder	Urinary retention (anticholinergic toxicity) Oral cavity burns (corrosive ingestion) Hypersalivation (cholinergic toxidrome)
Peripheral nervous	Reflexes Tone Fasciculations Tremor Clonus	Tremor/fasciculations (lithium toxicity) "Lead pipe" rigidity (neuromuscular malignant syndrome) Clonus/hyperreflexia (serotonin toxicity)

BOX 139.1

Agents Affecting Pupil Size

MIOSIS (COPS)

Cholinergics, clonidine, carbamates

Opioids, organophosphates

Phenothiazines (antipsychotics), pilocarpine, pontine hemorrhage

Sedative-hypnotics

MYDRIASIS (SAW)

Sympathomimetics

Anticholinergics

Withdrawal syndromes

Agents Causing Coma or Seizures

COMA (LETHARGIC)

Lead, lithium

Ethanol, ethylene glycol, ethchlorvynol

Tricyclic antidepressants, thallium, toluene

Heroin, hemlock, hepatic encephalopathy, heavy metals, hydrogen sulfide, hypoglycemics

Arsenic, antidepressants, anticonvulsants, antipsychotics, antihistamines

Rohypnol (sedative hypnotics), risperidone

Gamma-hydroxybutyrate (GHB)

Isoniazid, insulin

Carbon monoxide, cyanide, clonidine

SEIZURES (OTIS CAMPBELL)

Organophosphates, oral hypoglycemics

Tricyclic antidepressants

Isoniazid, insulin

Sympathomimetics, strychnine, salicylates

Camphor, cocaine, carbon monoxide, cyanide, chlorinated hydrocarbons

Amphetamines, anticholinergics

Methylxanthines (theophylline, caffeine), methanol

Phencyclidine (PCP), propranolol

Benzodiazepine withdrawal, botanicals (water hemlock, nicotine), bupropion, GHB

Ethanol withdrawal, ethylene glycol

Lithium, lidocaine

Lead, lindane

Agents With a Characteristic Odor

ODOR	POSSIBLE SOURCE
Bitter almonds	Cyanide
Carrots	Cicutoxin (water hemlock)
Fruity	Diabetic ketoacidosis, isopropanol
Garlic	Organophosphates, arsenic, dimethyl sulfoxide (DMSO), selenium
Gasoline	Petroleum distillates
Mothballs	Naphthalene, camphor
Pears	Chloral hydrate
Pungent aromatic	Ethchlorvynol
Oil of wintergreen	Methylsalicylate
Rotten eggs	Sulfur dioxide, hydrogen sulfide
Freshly mowed hay	Phosgene

Agents Causing Skin Findings

DIAPHORETIC SKIN (SOAP)

Sympathomimetics

Organophosphates

Acetylsalicylic acid or other salicylates

Phencyclidine (PCP)

DRY SKIN

Antihistamines, anticholinergics

BULLOUS LESIONS OR BLISTERS

Barbiturates and other sedative-hypnotics

Mustard gas

Snakes and spiders

FLUSHED OR RED APPEARANCE

Anticholinergics, niacin

Boric acid

Carbon monoxide (in morbid states)

Cyanide (rare)

CYANOSIS

Ergotamine

Nitrates

Nitrites

Aniline dyes

Phenazopyridine

Dapsone

Agent causing hypoxemia, hypotension, or methemoglobinemia

ACNEIFORM RASH

Bromides

Chlorinated aromatic hydrocarbons

TOXIDROMES

- * Substances belonging to a particular pharmaceutical/chemical class often produce a **cluster of symptoms** and **signs**, or “**toxidrome**” enabling the identification of potential toxins when a clear history is unavailable

Toxidrome Symptoms

CHOLINERGIC

Muscarinic (DUMBELLS)

Diarrhea, diaphoresis

Urination

Miosis

Bradycardia

Bronchorrhea

Emesis

Lacrimation

Lethargic

Salivation

Nicotinic: Days of Week

Mydriasis

Tachycardia

Weakness

Tremors

Fasciculations

Seizures

Somnolent

ANTICHOLINERGIC

Hyperthermia (HOT as a hare)

Flushed (RED as a beet)

Dry skin (DRY as a bone)

Dilated pupils (BLIND as a bat)

Delirium, hallucinations (MAD as a hatter)

Urinary retention (DRY as a bone)

Tachycardia

OPIOID

Miosis

Hypoventilation

Depressed mental status/coma

Withdrawal

Diarrhea

Mydriasis

Goose flesh

Tachycardia

Lacrimation

Hypertension

Yawning

Cramps

Hallucinations

Seizures (with ethyl alcohol [ETOH] and benzodiazepine withdrawal)

Sympathomimetic

Hyperthermic

Flushed

Diaphoretic

Mydriatic

Agitated

Tachycardic

Seizures

Predicting Toxicity From Vital Signs

BRADYCARDIA (PACED)

Propranolol (β -blockers), poppies (opioids), propoxyphene, physostigmine
Anticholinesterase drugs, antiarrhythmics
Clonidine, calcium channel blockers
Ethanol or other alcohols
Digoxin, digitalis

TACHYCARDIA (FAST)

Free base or other forms of cocaine, Freon
Anticholinergics, antihistamines, antipsychotics amphetamines, alcohol withdrawal
Sympathomimetics (cocaine, caffeine, amphetamines, phencyclidine [PCP]), solvent abuse, strychnine
Theophylline, tricyclic antidepressants (TCAs), thyroid hormones

HYPOTHERMIA (COOLS)

Carbon monoxide
Opioids
Oral hypoglycemics, insulin
Liquor (alcohols)
Sedative-hypnotics

HYPERTHERMIA (NASA)

Neuroleptic malignant syndrome (NMS), nicotine
Antihistamines, alcohol withdrawal
Salicylates, sympathomimetics, serotonin syndrome
Anticholinergics, antidepressants, antipsychotics

HYPOTENSION (CRASH)

Clonidine, calcium channel blockers
Rodenticides (containing arsenic, cyanide)
Antidepressants, aminophylline, antihypertensives
Sedative-hypnotics
Heroin or other opioids

HYPERTENSION (CT SCAN)

Cocaine
Thyroid supplements
Sympathomimetics
Caffeine
Anticholinergics, amphetamines
Nicotine

RAPID RESPIRATION (PANT)

PCP, paraquat, pneumonitis, phosgene
Acetylsalicylic acid (ASA) and other salicylates
Noncardiogenic pulmonary edema, nerve agents
Toxin-induced metabolic acidosis

SLOW RESPIRATION (SLOW)

Sedative-hypnotics (barbiturates, benzodiazepines)
Liquor (alcohols)
Opioids
Weed (marijuana)

SEGMENT/INTERVAL	APPEARANCE	AGENT(S)
QT/QTc	Prolonged	Antipsychotics (typical and atypical), citalopram, hydrofluoric acid, methadone, ethylene glycol (oxalate byproduct)
T wave	Peaked	Hydrofluoric acid (hyperkalemia)
	Flattened	Lithium
U wave		Barium, beta-agonists, lithium, methylxanthines (caffeine, theophylline), toluene

DIAGNOSTIC TESTING

- * A serum **acetaminophen** concentration is a routine screening test in poisoned patients.
- * An **electrocardiogram** is a useful test to detect cardiac conduction abnormalities
- * Toxicologic screening tests seldom directly influence patient management, and toxicology screening has **limitations**
- * Toxicologic screening may be appropriate for **medicolegal** reasons, especially in pediatric cases
- * A positive **urine drug** screen for an **illicit** substance is an indication to involve local **child protection services**

Toxicologic Electrocardiogram Manifestations

SEGMENT/INTERVAL	APPEARANCE	AGENT(S)
P wave	Absent	Digoxin Cholinergics Hyperkalemia
	Notched	Quinidine
PR interval	Prolonged	Beta-antagonists, calcium-channel antagonists, magnesium
QRS interval	Prolonged	Type 1 antidysrhythmics, cocaine, diphenhydramine, tricyclic antidepressants
ST segment	Scooped	Digoxin ("Salvador Dali's moustache")

DECONTAMINATION

- * **Decontamination** is required for toxic exposures affecting **large dermal areas**.
- * Healthcare providers wearing personal protective equipment (if indicated) or observing universal precautions (**gown, gloves, eye protection**) should assist with undressing and washing the patient using copious amounts of water.
- * Contaminated clothing is **collected, bagged**, and properly **disposed**.
- * Decontamination ideally occurs in a **separate area** adjacent to the ED, minimizing cross-contamination

OCULAR DECONTAMINATION

- * Eye exposures may require local **anesthetic** (e.g., 0.5% tetracaine) instillation and lid **retractors** to facilitate copious irrigation with crystalloid solution.
- * **Alkalis** produce greater injury than acids due to **deep** tissue penetration via liquefaction so that prolonged irrigation (**1 to 2** hours) may be required.
- * **Ten minutes** after irrigation (allowing equilibration of crystalloid and conjunctival sac pHs), conjunctival sac pH is tested.
Irrigation continues until **pH is <7.4**.
- * **Ophthalmologic consultation** is indicated for all ocular alkali injuries

GASTROINTESTINAL DECONTAMINATION

- * **Gastric** decontamination is not a routine part of poisoned-patient management
- * Gastric decontamination may be considered in individual patients after a three-question risk-benefit analysis:
 - (1) Is this exposure likely to cause **significant toxicity**?
 - (2) Is gastrointestinal decontamination likely to **change** clinical **outcome**?
 - (3) Is it possible that gastrointestinal decontamination will cause **more harm** than good

Emesis

- * Traditionally, **ipeccac** syrup was administered to induce vomiting, theoretically emptying the stomach of poisons.
- * ipecac may be used in rare circumstances in **remote locations**, but this recommendation has been questioned.

**There is no role for the induction of emesis
in the ED**

Orogastric Lavage

- * **orogastric lavage** is now rarely indicated. changes outcome ? complications ??
- * may be considered in cases of:
 - ingestion of a **life-threatening amount** of poison within the **previous hour** where institution of **supportive** care and **antidotal** therapy would not ensure full recovery.
- * When orogastric lavage is performed in a resuscitation area:
 - Ensure a **protected airway** if consciousness level is reduced.
 - Use a **36 to 40F-gauge** orogastric tube (22 to 24F in children).
 - Position the patient on the left side with the **head down 20** degrees.
 - Pass lubricated tube down the esophagus a distance equal to that between **chin and xiphoid** process.
 - Confirm tube position by **insufflation of air**.
 - Gently lavage with **200 mL** (10 mL/kg in children) of warm **tap water**.
 - Continue until returned fluid is **clear**.
 - Consider administration of **activated charcoal** via orogastric tube **before** removal

Single-Dose Activated Charcoal

- * Toxins within the GI lumen are adsorbed onto the activated charcoal.
- * Activated charcoal does **not** effectively **adsorb metals**, **corrosives**, and **alcohols**.
- * The decision to give activated charcoal requires individual patient **risk assessment** and is **not considered routine** management.
- * may be effective when given **>60** minutes after ingestion of substances known to **slow GI motility** (e.g., **anticholinergics**) or after massive ingestion of a substance associated with **bezoar** formation (e.g., **salicylates**).
- * There are **insufficient** published **data** supporting the routine use of a **cathartic** agent **added** to activated **charcoal**

Substances That Do *Not* Bind to Activated Charcoal

PHAILS

Pesticides

Heavy metals

Acids/alkalis

Iron

Lithium

Solvents

Whole-Bowel Irrigation

- * **Polyethylene glycol** can be administered orally to **cooperative, awake** patients, but consider formal airway control if consciousness is likely to deteriorate.
- * **Minimize** risk of pulmonary **aspiration** during whole-bowel irrigation by:
 - patient positioning (**head up 30** degrees), ensuring **bowel sounds** are present during fluid administration, and utilization of **cuffed endotracheal** tubes.
- * Nonsurgical treatment of asymptomatic **body drug packers** using **whole-bowel** irrigation is increasingly common
- * **metoclopramide** may be required to control polyethylene glycol–induced gastric **distension** and **vomiting**.
- * The endpoint is **clear** rectal effluent and imaging demonstrating **absence** of **foreign bodies**

ENHANCED ELIMINATION

- * MULTIDOSE ACTIVATED CHARCOAL
- * URINARY ALKALINIZATION

MULTIDOSE ACTIVATED CHARCOAL

* **Indications:**

- **Carbamazepine** coma (reduces duration of coma)
- **Phenobarbital** coma (reduces duration of coma)
- **Dapsone** toxicity with significant methemoglobinemia
- **Quinine** overdose
- **Theophylline** overdose if hemodialysis/hemoperfusion unavailable

URINARY ALKALINIZATION

- * **Alkaline** urine favors **ionization** of acidotic drugs within renal tubules, preventing **resorption** of the ionized drug (most effective for **weak acids**)
- * **Hypokalemia** will reduce the effectiveness of urinary alkalization.
- * Indication: **moderate to severe salicylate** toxicity when criteria for hemodialysis have not been met.
- * Urinary alkalization for adult patients can be instituted as follows:
 - **Correct** any existing **hypokalemia**.
 - Administer a **1 to 2 mEq/kg** IV **sodium bicarbonate** bolus.
 - Infuse **100** mEq of sodium bicarbonate mixed with **1 L of D5W** at **250 mL/h**.

* 20 mEq of potassium chloride may be added to the solution to maintain normokalaemia.

- Monitor serum potassium and bicarbonate every 2 to 4 hours to detect

hypokalemia or excessive serum alkalinization.

- Check urine pH regularly (every 15 to 30 minutes), aiming for a pH of 7.5 to 8.5

* **GOAL :**

serum pH of approximately 7.5 and a urinary pH of approximately 8.0

DISPOSITION

- * should be part of initial risk assessment.
- * **Admission** is indicated if the patient has **persistent** and/or **severe toxic** effects
- * In most cases, a **6-hour observation** period is sufficient to exclude the development of serious toxicity.
- * Onset of clinical toxicity can be delayed after :
 - **calcium** channel **antagonists**
 - **tramadol**, **venlafaxine**
 - newer antipsychotics (**amisulpride**)

hence a period of extended observation is indicated.
- * Patients who have deliberately **self-poisoned** require appropriate **mental health assessment** before disposition

از توجهتان متشکرم

