Approach to the Poisoned Patient

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I have no relevant financial disclosures related to this presentation.

Objectives
Identify commonly abused agents
Identify common presentations of intoxicated patients
Identify critical findings suggestive of specific agents
Discuss treatment options for toxins
Discuss outcomes and disposition for common agents

Epidemiology
Accidental and intentional poisoning remains a major cause of morbidity and mortality worldwide.

In the United States, the American Association of Poison Control Centers (AAPCC) reported over 2.3 million human exposure calls in 2011.

While the overall mortality rate reported by the AAPCC was 0.05 percent, 26.4 percent of cases required management at a healthcare facility and 7.1 percent required hospital admission.

There were an estimated 907,000 emergency department (ED) visits related to poisoning in 2005, accounting for approximately 0.79 percent of all ED visits.

Source: http://www.uptodate.com/

General Approach to the Poisoned Patient

Presentation depends upon the agent ingested, whether the ingestion is acute or chronic, baseline agents a patient may be taking, and whether the ingestion involves a single drug or several coingestants.

Initial management is focused on acute stabilization. ABCDE, IV/O2/Monitor, etc.

The history and physical examination are of great importance in recognizing which type of poisoning has occurred.

Management is directed to the provision of supportive care, prevention of poison absorption, and, when applicable, the use of antidotes and enhanced elimination techniques.

Source: http://www.uptodate.com/
Primary Assessment

A brief initial screening examination should be performed on all patients to identify immediate measures required to stabilize and prevent deterioration of the patient.

Assess vital signs, mental status, and pupil size, skin temperature and moisture, and perform a brief examination, electrocardiogram monitoring, and an electrocardiography if intravenous access and a fitter stick are available.

In patients with suspected occult trauma, maintain in-line cervical immobilization. Assess the airway and perform endotracheal intubation if there is significant doubt about the patient’s ability to protect their airway and avoid aspiration. Provide advanced cardiac life support measures as required.

Source: http://www.uptodate.com/

Do the DONT

Dextrose
Oxygen
Naloxone
Thiamine

Patients with altered consciousness should prompt administration of intravenous thiamine (100 mg) to prevent Wernicke’s encephalopathy and 25 g of dextrose (50 mL of a 50 percent solution) to treat hypoglycemia, unless these diagnoses can be rapidly excluded. Administer intravenous naloxone (0.4-2 mg) if signs or symptoms of opioid intoxication are present. Wernicke’s encephalopathy can be caused by alcoholism, and the administration of thiamine should be delayed until the patient has recovered from alcohol withdrawal. Uptake of thiamine into cells is slower than that of dextrose, and withholding dextrose until the administration of thiamine is complete may prove detrimental to those with actual hypoglycemia.


The Tox History MATTERS

Medication
Amount
Time Taken
Emesis (pill fragments)
Reason
Symptoms

And Remember...

Trust No One
Physical Examination

The mental status, vital signs, and pupillary examination are the most useful elements and allow classification of the patient into either a state of physiologic excitation or depression.

Physiologic excitation, manifested by increased rate and depth of respiration, and increased pulse, blood pressure, temperature, is most commonly caused by anticholinergic, sympathomimetic, or central hallucinogenic agents, or drug withdrawal states.

Physiologic depression, manifested by decreased mental status, blood pressure, pulse, respiration rate, and temperature, is most commonly precipitated by ethanol, other sedative-hypnotic agents, opiate or opioid agonists, sympatholytics, or toxic alcohols (methanol or ethylene glycol).

Mixed physiologic effects may occur in polydrug overdoses or following exposure to certain metabolic poisons (eg, hypoglycemic agents, salicylates, cyanide), membrane-active agents (eg, volatile inhalants, antiarrhythmic drugs, local anesthetic agents), heavy metals (eg, iron, arsenic, mercury, lead), or agents with multiple mechanisms of action (eg, tricyclic antidepressants).

Source: http://www.uptodate.com/

Toxidromes

<table>
<thead>
<tr>
<th>Toxidrome</th>
<th>Mental Status</th>
<th>Pupils</th>
<th>Vital Signs</th>
<th>Other Manifestations</th>
<th>Examples of Toxic Agents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sympathomimetic</td>
<td>Hyperalert, agitation, hallucinations, paranoia</td>
<td>Mydriasis</td>
<td>Hyperthermia, tachycardia, hypertension, tachypnea</td>
<td>Dry flushed skin, diaphoresis, tremors, hyperreflexia, seizures</td>
<td>Cocaine, amphetamines, cathinones, ephedrine, pseudoephedrine, phenylpropanolamine, theophylline, caffeine</td>
</tr>
<tr>
<td>Anticholinergic</td>
<td>Hypervigilance, agitation, hallucinations, delirium</td>
<td>Mydriasis</td>
<td>Hyperthermia, tachycardia, hypertension, tachypnea</td>
<td>Dry flushed skin, dry mucous membranes, decreased bowel sounds, urinary retention, myoclonus, choreoathetosis, picking behavior, seizures (rare)</td>
<td>Antihistamines, tricyclic antidepressants, cyclobenzaprine, orphenadrine, antiparkinson agents, antispasmodics, phenothiazines, atropine, scopolamine, belladonna alkaloids (eg, Jimson Weed)</td>
</tr>
<tr>
<td>Hallucinogenic</td>
<td>Hallucinations, perceptual distortions, depersonalization, synesthesia, agitation</td>
<td>Mydriasis</td>
<td>Hyperthermia, tachycardia, hypertension, tachypnea</td>
<td>Nystagmus</td>
<td>Phencyclidine, LSD, mescaline, psilocybin, designer amphetamines (eg, MDMA [&quot;Ecstasy&quot;], MDEA)</td>
</tr>
<tr>
<td>Opioid</td>
<td>CNS depression, coma</td>
<td>Miosis</td>
<td>Hypothermia, bradycardia, hypotension, apnea, bradypnea</td>
<td>Hyporeflexia, pulmonary edema, needle marks</td>
<td>Opioids (eg, heroin, morphine, methadone, oxycodone, hydromorphone), diphenoxylate</td>
</tr>
<tr>
<td>Sedative-hypnotic</td>
<td>CNS depression, confusion, stupor, coma</td>
<td>Variable</td>
<td>Hypothermia, bradycardia, hypotension, apnea, bradypnea</td>
<td>Hyporeflexia</td>
<td>Benzodiazepines, barbiturates, carisoprodol, meprobamate, glutethimide, alcohols, zolpidem</td>
</tr>
<tr>
<td>Cholinergic</td>
<td>Confusion, coma</td>
<td>Miosis</td>
<td>Bradycardia, hypertension or hypotension, tachypnea or bradypnea</td>
<td>Salivation, urinary and fecal incontinence, diarrhea, emesis, diaphoresis, lacrimation, GI cramps, bronchoconstriction, muscle fasciculations and weakness, seizures</td>
<td>Organophosphate and carbamate insecticides, nicotine, pilocarpine, physostigmine, edrophonium, bethanechol, urecholine</td>
</tr>
<tr>
<td>Serotonin syndrome</td>
<td>Confusion, agitation, coma</td>
<td>Mydriasis</td>
<td>Hyperthermia, tachycardia, hypertension, tachypnea</td>
<td>Tremor, myoclonus, hyperreflexia, clonus, diaphoresis, flushing, trismus, rigidity, diarrhea</td>
<td>MAOIs alone or with: SSRIs, meperidine, dextromethorphan, TCAs, L-tryptophan</td>
</tr>
</tbody>
</table>

Source: http://www.uptodate.com/
Odors

<table>
<thead>
<tr>
<th>Odor</th>
<th>Agent(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetone</td>
<td>fruity</td>
</tr>
<tr>
<td>Ethanol, isopropyl alcohol, chloroform</td>
<td></td>
</tr>
<tr>
<td>Salicylates</td>
<td></td>
</tr>
<tr>
<td>Bitter almonds</td>
<td></td>
</tr>
<tr>
<td>Cyanide</td>
<td></td>
</tr>
<tr>
<td>Garlic</td>
<td>Arsenic, organophosphates, phosphorus, thallium, selenium</td>
</tr>
<tr>
<td>Mothballs</td>
<td>Naphthalene, paradichlorobenzene</td>
</tr>
<tr>
<td>Kerosene</td>
<td>Organophosphates, parathion</td>
</tr>
<tr>
<td>Freshly mown hay</td>
<td></td>
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<tr>
<td>Phosgene</td>
<td></td>
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<tr>
<td>Rotten eggs</td>
<td>Hydrogen sulfide</td>
</tr>
<tr>
<td>Wintergreen</td>
<td>Methyl salicylate</td>
</tr>
</tbody>
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Sources:
https://images.google.com
and
http://www.uptodate.com/

Skin Findings

<table>
<thead>
<tr>
<th>Red and flushed</th>
<th>Cyanotic</th>
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</thead>
<tbody>
<tr>
<td>Anticholinergic agents</td>
<td>Methemoglobinemia</td>
</tr>
<tr>
<td>Antihistamines</td>
<td>Sulfhemoglobinemia</td>
</tr>
<tr>
<td>TCAs</td>
<td>Hypoxemia</td>
</tr>
<tr>
<td>Atropine</td>
<td>Desquamation</td>
</tr>
<tr>
<td>Scopolamine</td>
<td>Stevens-Johnson syndrome</td>
</tr>
<tr>
<td>Belladonna alkaloids</td>
<td>Toxic epidermal necrolysis</td>
</tr>
<tr>
<td>Phenothiazines</td>
<td>Boric acid</td>
</tr>
<tr>
<td>Disulfiram</td>
<td>Heavy metals</td>
</tr>
<tr>
<td>Disulfiram/ethanol</td>
<td>Arsenic</td>
</tr>
<tr>
<td>Cephalosporins</td>
<td>Salicylates</td>
</tr>
<tr>
<td>Ethanol</td>
<td></td>
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<tr>
<td>Solvents</td>
<td></td>
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<tr>
<td>Coprinus mushrooms</td>
<td></td>
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<tr>
<td>Monosodium glutamate</td>
<td></td>
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<tr>
<td>Scombroid fish poisoning</td>
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<tr>
<td>Rifampin</td>
<td></td>
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<tr>
<td>Carbon monoxide</td>
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</table>

EKG Findings

Electrocardiographic abnormalities may provide diagnostic and prognostic information, and an ECG should be performed on all patients who are symptomatic or who have been exposed to potentially cardiotoxic agents. Particular attention should be paid to the duration of the QRS and QTc intervals, abnormalities of the QRS shape, and QRS prolongation. Any variant increases interventricular and intraventricular conduction delays and cardiac arrhythmia.

Imaging

Imaging studies are not required in every patient but may be useful in a few situations:

- Certain radiopaque toxins may be visualized by plain film radiographs.
- Ingested drug packets of "body packers" may be visualized on plain films.
- Noncardiogenic pulmonary edema and/or the acute respiratory distress syndrome due to exposure to certain toxic agents may be suggested by the appearance of the chest radiograph.

Labs

"Overdose Panel" or "Tox Panel" is always a matter of debate. What I do: CBC, CMP, EtOH, Tyl, Sal, UA, UTox, UPreg for females of childbearing age, drug levels if indicated, Osm if any indication of toxic alcohol ingestion. IL poison center often asks for INR/APTT as an indicator of hepatic dysfunction, so I usually order this as well. For critically ill patients, consider Mg, Phos, Serum Ketones, CK, Lipase, Ionized Ca, Lactate, and ABG.

Screening for acetaminophen and salicylates is strongly recommended for patients with an uncertain history or intentional poisoning; few early signs may be present following lethal doses of these agents, and specific treatments are available and highly effective if implemented early. One retrospective study found detectable serum acetaminophen concentrations in 9.6 percent of all overdose patients; almost one-third of this subset denied ingestion of acetaminophen.

"Drugs of abuse" immunoassay screens can be used to detect opioids, benzodiazepines, cocaine metabolites, barbiturates, tricyclic antidepressants, tetrahydrocannabinol, and phencyclidine in urine. These assays are inexpensive and provide rapid results, usually within one hour. Positive and negative screens do not confirm or refute poisoning diagnoses and further confirmation is needed by GC/MS for forensic purposes. As an example, a negative screen may reflect a drug concentration below the threshold limit of detection due to timing of the specimen before or after peak concentration. Conversely, high concentrations of certain drugs may be due to a false positive result. As an example, diphenhydramine can cause false-positive results, especially with tricyclic antidepressants. In some instances, a positive test result may be an earlier indicator that does not accord with the patient's presentation.
Drug Levels to Consider

- Acetaminophen
- Carboxyhemoglobin
- Methemoglobin
- Digoxin
- Lithium
- Theophylline
- Salicylate
- Paraquat
- Iron
- Methanol
- Ethylene glycol
- Antiepileptic agents
  - Carbamazepine
  - Phenytoin
  - Valproic acid
- Heavy metals
  - Lead
  - Mercury

Sources:
- https://images.google.com
- http://www.uptodate.com/

Decontamination

Following initial patient stabilization, patient decontamination may be performed if indicated. The sooner decontamination is performed, the more effective it is at preventing poison absorption. Exposure to oral or skin ingested substances can be managed by several methods, including gastric emptying (by inducing vomiting, lavages, and cathartic administration) and percutaneous administration in patients with ingestions. Systemic decontamination in patients with ingestions may be achieved by administering activated charcoal, but may not be efficacious in the setting of a delayed ingestion with Fixed Clinical or the TracStar System. If systemic decontamination is contraindicated or unanticipated ingestion is noted, gastrointestinal decontamination may be warranted, such as gastric lavage, oral or nasal irrigation, endoscopy, surgery, dialysis, and cathartics (consult with Poison Control or Medical Toxicologist for consultation procedures).

Sources:
- https://images.google.com
- http://www.uptodate.com/

Antidotes

Supportive care is the cornerstone of the treatment of the poisoned patient, and the adage “treat the patient, not the poison” is the guiding principle of medical toxicology. However, there are instances in which prompt administration of a specific antidote is potentially life-saving.

Antidote administration is appropriate when there is a poisoning for which an antidote exists, the actual or predicted severity of poisoning warrants its use, expected benefits of therapy outweigh its associated risk, and there are no contraindications to the use of the specific antidote. Antidotes can be effective in preventing morbidity and mortality in certain situations, but they are unable to reverse those effects that are caused by the poison. Antidotes are used in only a small fraction of cases.

Although a response to empirically administered antidotes can be used to confirm a suspected diagnosis, their indiscriminate use can potentially increase patient morbidity. Antidote administration should be reserved for specific instances, such as poisoning that has resulted in coma, respiratory arrest, or other life-threatening conditions.

The most common causes of death from poisoning are cardiac arrest, respiratory failure, and seizures. Antidotes can be used to treat these conditions, but they are not effective in reversing the underlying poison.

Enhanced Elimination

Procedures to enhance elimination of poisons include forced diuresis, urine ion trapping, hemodialysis, hemoperfusion, hemofiltration, and exchange transfusion. Each of these techniques has its own indications and limitations, and it is necessary to tailor the treatment plan accordingly.
Supportive Care

History and physical examination should be performed in all patients. High risk for aspiration is indicated when the patient is unconscious, sedated, intubated, has a history of increased intracranial pressure or respiratory depression, and for those with severe hypotension or bradycardia. When a patient has severe hypotension or bradycardia, the use of vasopressors for support is recommended. The patient may also require intubation to facilitate mechanical ventilation to limit the extent of complications such as hyperthermia, acidosis, and rhabdomyolysis. One can also consider hyperthermia secondary to drug toxicity (e.g., sympathomimetic overdose, serotonin syndrome, or neuroleptic malignant syndrome) may require aggressive treatment, possibly including ice water immersion.

Supportive Care

- Seizures generally are best treated with benzodiazepines, followed by barbiturates if necessary.
- Drug-associated agitated behavior is generally best treated with benzodiazepine administration.
- Severe hyperthermia secondary to drug toxicity (e.g., sympathomimetic overdose, serotonin syndrome, or neuroleptic malignant syndrome) may require aggressive treatment, possibly including ice water immersion.

Supportive Care

- Hypotension should be managed initially with boluses of isotonic intravenous fluids. Vasopressors should be used only if necessary.
- Hypertension in agitated patients is best treated initially with nonspecific sedatives such as benzodiazepines.
- Drug-associated tachycardia is best managed with nonspecific beta-blockers.

Disposition

Following initial evaluation, treatment, and a short period of observation, disposition of the patient is determined. Patients who are at high risk for complications or who are failing to improve may require admission to the ICU. Other factors considered in the decision to discharge the patient include the patient’s ability to participate in their care and the availability of follow-up. Patients who are not at high risk for complications and who are improving may be discharged to an intermediate-care floor or an appropriate observation unit for continued monitoring and treatment. Patients with significant toxicity should be admitted to an intermediate-care floor or an appropriate observation unit for continued monitoring and treatment. Patients who develop only mild toxicity and who have only minor symptoms should be allowed to return home.

References

UpToDate Online
Goldfrank’s Toxicologic Emergencies, 10th ed.
Personal Instruction from Carst<br>Harris, MD