General approach to drug poisoning in adults

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INTRODUCTION — Accidental and intentional poisonings or drug overdoses constitute a significant source of aggregate morbidity, mortality, and health care expenditure worldwide. Millions of poisonings and drug overdoses occur annually in the United States alone [1,2].

The general approach and initial management of patients with suspected or confirmed poisoning will be reviewed here. Specific issues relating to the management of common drug overdoses are discussed separately (see appropriate topic reviews). A topic devoted to the management of the critically ill patient with an unknown overdose is found separately. (See "Initial management of the critically ill adult with an unknown overdose").

EPIDEMIOLOGY — Accidental and intentional poisoning from both licit and illicit substances remains a major cause of morbidity and mortality worldwide [3-7]. In the United States, the American Association of Poison Control Centers (AAPCC) reported over 2.1 million human exposure calls in 2014. While the overall mortality rate reported by the AAPCC was 0.07 percent, 28.3 percent of cases required management at a health care facility and 7.9 percent of cases required hospital admission [2]. Between 2008 and 2011 in the United States, there were an estimated 1.1 million annual emergency department (ED) visits related to drug poisoning, or 35.4 visits per 10,000 persons; 24.5 percent of these patients presenting with drug poisoning required hospital admission, compared with 12.7 percent for non-poisoning related presentations [8]. Rates of poisoning cases among ED patients appear similar in other industrialized nations [9].

As of 2008, poisoning has become the leading cause of injury-related death in the United States, surpassing motor vehicle collisions. The majority of poisoning fatalities were related to drugs, with 36,500 cases in 2008 [10]; most overdoses involved prescription drugs [11]. Patients aged 35 to 54 years accounted for the highest number of poisonings, but patients between the ages of 18 and 20 had the highest rate. The contribution of poisoning to suicide cases varies by region: suicidal poisoning is especially prevalent in Scandinavian countries and the United Kingdom, while the burden of suicidal poisonings is relatively less in most of Eastern Europe and Central and South America [12].

Among adults, the AAPCC reported the most common exposures were due to analgesics (11.3 percent), sedatives and antipsychotics (5.9 percent), and antidepressants (4.4 percent). A review of a toxicology case registry noted similar findings, with sedatives, analgesics and antidepressants accounting for the most frequently mentioned exposures [13]. Reviews of self-poisoning cases in the United Kingdom and Spain found that analgesics, benzodiazepines, and antidepressants were the most commonly encountered drugs [9,14]. However, trends vary in other geographic regions. In a study of Norwegian patients, the most prevalent drugs (aside from ethanol) were acetaminophen, opioids, and gamma hydroxybutyrate (GHB) [15]. A German study of intensive care unit (ICU) admissions found benzodiazepines, antidepressants and antihistamines were the most commonly encountered drugs, again excluding ethanol [16]. A report of Israeli poison center data found that antimicrobials were the most frequently reported drugs, second only to analgesics (including opioids) [17].

OVERVIEW OF APPROACH — Clinicians who treat poisoned patients should have a systematic and consistent approach to evaluation and management. It is important to note that drug poisoning can produce a wide range of symptoms and clinical findings. Presentation depends upon the agent ingested, whether the ingestion is acute or chronic, baseline prescription medications a patient may be taking, and whether the ingestion involves a single drug or several coingestants. Initial management is focused on acute stabilization. The history and physical examination are of great importance in recognizing that 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 [18].

INITIAL EVALUATION AND TREATMENT — 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 pulse oximetry, continuous cardiac monitoring, and an electrocardiogram. Obtain intravenous access and a finger-stick glucose measurement. 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. (See "Initial management of the critically ill adult with an unknown overdose" and "Initial management of trauma in adults" and "The decision to intubate" and "Rapid sequence intubation for adults outside the operating room" and "Advanced cardiac life support (ACLS) in adults".)

Patients with altered consciousness should prompt administration of intravenous thiamine 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 to patients with signs, symptoms, or a history suggestive of opioid intoxication [18,19]. (See "Wernicke encephalopathy", section on 'Treatment' and "Acute opioid intoxication in adults", section on 'Basic measures and antidotal therapy'.)

The notion that thiamine must be given prior to dextrose to avoid precipitating Wernicke's encephalopathy is largely unsupported [20]. Uptake of thiamine into cells is slower than that of dextrose [21], and withholding dextrose until the administration of thiamine is complete may prove detrimental to those with actual hypoglycemia. Rapid bedside glucometers are the most expedient method to determine the presence of hypoglycemia. Empiric administration of dextrose is recommended for the patient with altered consciousness when bedside glucose measurements are low or borderline-low, not immediately available, or the accuracy of the results is questionable. Follow-up finger-stick or serum glucose measurements should be obtained in patients with a persistent altered mental status, as hypoglycemia may develop during the later stages of some poisonings.

Be certain to expose the patient completely and look for signs of trauma, drug use, infection, or extremity swelling. Measure the core body temperature. Patient decontamination should then be initiated (if indicated). Obtain an electrocardiogram to assess for drug-related cardiotoxicity (see section on electrocardiogram below). Search clothing, wallets, and pocket books for pills, pill bottles, or drug related equipment, but take care when doing so to avoid a needle stick. A more detailed diagnostic evaluation can then ensue. (See "Gastrointestinal decontamination of the poisoned patient".)

DIAGNOSIS OF POISONING — The history, physical examination, and routine and toxicological laboratory evaluations are used to establish and confirm the diagnosis of poisoning.

History — The history, although intuitively the source of the most helpful information for identifying the etiology of poisoning, is often unreliable when provided by a patient following intentional ingestion [22,23]. In one prospective survey, the initial clinical history fully correlated with confirmatory testing in only 27 percent of cases [24]. The patient's ability to provide a reliable history is often impaired by direct drug effects or psychiatric illness [18]. Therefore, the patient's history should be confirmed whenever possible and correlated with the signs, symptoms, and laboratory data expected from poisoning with the agent(s) implicated by history. When the patient is unable or unwilling to give a reliable history regarding poison exposure, information should be sought from paramedics, police, and the patient's employer, family, friends, primary care clinician, and pharmacist.

A thorough search of the exposure environment should be conducted for pill bottles or a suicide note which may provide clues to etiologic agent(s). Knowledge of drugs prescribed for the patient or the patient's family or friends to which he or she could have had access may also prove important. Unknown pills or chemicals may be identified by consultation with a regional poison control center, computerized drug or poison identification system, or product manufacturer (eg, material data safety sheet). United States poison control centers can be reached through a toll-free line (1-800-222-1222). The World Health Organization (WHO) provides a listing of international poison centers at its website.

It is critical to inquire specifically about the use of over-the-counter medications, traditional or herbal remedies, and...
dietary supplements as these are often not considered to be medications by the patient and may not be volunteered during routine questioning about "drugs." In addition, keep in mind that patients or secondary sources providing history may misidentify drugs. Over-the-counter products may be confused (eg, acetaminophen versus aspirin) or prescription medications may be mistaken for each other (eg, clonazepam versus clonidine). Lastly, drugs of abuse may only be identified by colloquial or slang terms (eg, "ecstasy" for MDMA, or "bath salts" for synthetic cathinones) [18].

Physical examination — The physical examination of symptomatic poisoned patients may provide invaluable clues to the agent involved. 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 [25].

- Physiologic excitation, manifested by central nervous system stimulation and increased pulse, blood pressure, respiratory rate and depth, and temperature, is most commonly caused by anticholinergic, sympathomimetic, or central hallucinogenic agents, or by drug withdrawal states. (See "Anticholinergic poisoning" and "Cocaine: Acute intoxication" and "Methamphetamine intoxication" and "Acute amphetamine and synthetic cathinone ("bath salt") intoxication" and "Phencyclidine (PCP) intoxication in adults" and "Intoxication from LSD and other common hallucinogens" and "Management of moderate and severe alcohol withdrawal syndromes".)

- Physiologic depression, manifested by a depressed mental status, blood pressure, pulse, respiratory rate and depth, and temperature, is most commonly precipitated by ethanol, other sedative-hypnotic agents, opiates, cholinergic (parasympathomimetic) agents, sympatholytics, or toxic alcohols (methanol or ethylene glycol). (See "Organophosphate and carbamate poisoning" and "Acute opioid intoxication in adults" and "Benzodiazepine poisoning and withdrawal" and "Ethanol intoxication in adults" and "Methanol and ethylene glycol poisoning".)

- 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). (See "Metformin poisoning" and "Sulfonylurea agent poisoning" and "Salicylate (aspirin) poisoning in adults" and "Cyanide poisoning" and "Acute iron poisoning" and "Tricyclic antidepressant poisoning".)

Following the initial diagnostic evaluation and stabilization, other physical findings should be sought to further define a particular toxic syndrome (toxidrome) and to narrow the potential etiologies of poisoning. The following table describes the major characteristics of the common toxidromes and was created to make comparisons easier and assist with diagnosis (table 1). However, a particular patient may not manifest all the symptoms or findings typically associated with a given toxidrome. The diagnosis may be assisted by [18,26]:

- Characteristic odors (table 2)
- Pupillary findings (table 3)
- Neuromuscular abnormalities (table 4)
- Mental status alterations (table 5)
- Skin findings (table 6)
- Temperature alterations (table 7)
- Blood pressure and heart rate alterations (table 8)
- Respiratory disturbances (table 9)

Discrepancies between the physical examination and the history may reflect an inaccurate ingestion history or a brief or prolonged time interval between exposure and physical examination. The physical examination, particularly the evaluation of mental status and vital signs, should be repeated frequently (approximately every hour, depending upon the patient's condition) to determine the course of poisoning and the need for further intervention.

Electrocardiography — 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 ([table 10] [27]. Particular attention should be paid to the duration of the QRS and QTc intervals. Toxin-induced QRS interval prolongation, of the type seen with tricyclic antidepressant poisoning, may warrant immediate intervention. (See "Tricyclic antidepressant poisoning".)

**Radiographic studies** — Imaging studies are not required in every patient but may be useful in several situations [28,29]:

- Certain radiopaque toxins (summarized by the mnemonic "CHIPES") may be visualized by plain film radiographs ([table 11] and [image 1]).
- Ingested drug packets of "body packers" may be seen on plain films ([image 2]). (See "Internal concealment of drugs of abuse (body packing)".)
- Abdominal ultrasound does not appear to be a reliable method of detecting ingested medications [30,31].
- 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 ([table 12]).

**Toxicology screens (drug testing)** — Toxicology screening is rarely necessary when patients with a non-intentional ingestion are asymptomatic or have clinical findings that are consistent with the medical history. However, 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 [32]. Other less common "toxic time bombs" for which diagnosis demands a high index of suspicion are listed in the table ([table 13]). (See "Acetaminophen (paracetamol) poisoning in adults: Pathophysiology, presentation, and diagnosis" and "Acetaminophen (paracetamol) poisoning in adults: Treatment" and "Salicylate (aspirin) poisoning in adults".)

"Drugs of abuse" immunoassay screens can be used to detect opioids, benzodiazepines, cocaine metabolites, barbiturates, tricyclic antidepressants, tetrahydrocannabinol, and phencyclidine in urine ([table 14]). These assays are inexpensive and provide rapid results, usually within one hour. Positive and negative screens for drugs do not absolutely confirm or refute poisoning diagnoses and require further evaluation. 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 in assays screening for tricyclic antidepressants [33]. In some instances, a positive test may reflect an earlier ingestion that does not account for the patient's presentation. Testing for drugs of abuse is reviewed in detail separately. (See "Testing for drugs of abuse (DOA)", and "Substance use disorder: Principles for recognition and assessment in general medical care")

In contrast to the rapid immunoassay screens, comprehensive qualitative toxic screening of urine, blood, or other body fluids (commonly by liquid and gas chromatography and mass spectrometry) is expensive, commonly requires six hours for results, often does not predict or define the severity of poisoning, detects unsuspected drugs in only a minority of patients, rarely leads to changes in patient management and disposition, and is unlikely to affect patient outcome [24,34-36]. Thus, comprehensive toxicology screening should be reserved for patients with severe or unexplained toxicity. Urine is the optimal matrix for analysis (as opposed to serum or whole blood) due to the longer window for detection and higher concentrations of drugs or their metabolites.

Quantitative assays have defined roles in the management of poisoned patients. They are useful in guiding the management of certain intoxications when interpreted in conjunction with clinical status and the timing of poisoning ([table 15]).

**Other laboratory studies** — Certain laboratory abnormalities are characteristic of specific agents (table 16 and table 17). Symptomatic patients and those with an unreliable or unknown history should, at a minimum, undergo urinalysis and measurement of serum electrolytes, BUN, creatinine, and glucose. Measurements of serum osmolality, ketones, creatine kinase, liver function tests, lipase, ionized calcium, and magnesium should also be performed in most significantly ill patients. Routine urine pregnancy testing is strongly recommended in all women of
childbearing age.

The ordering of other laboratory studies should be individualized and is somewhat dependent upon the results of initial laboratory studies:

- Arterial blood gas, co-oximetry, and serum lactate measurements may be necessary in patients with acid-base, cardiovascular, neurologic, or respiratory disturbances. Co-oximetry can aid in the rapid diagnosis of carbon monoxide poisoning and methemoglobinemia. (See "Carbon monoxide poisoning" and "Clinical features, diagnosis, and treatment of methemoglobinemia".)

- Any patient with an acid-base disturbance, increased serum osmolal gap, or oxygen saturation gap (>5 percent difference between measured and calculated value) should have a toxic etiology ruled out (table 18 and table 19). Detection of a plasma osmolal gap with any alcohol intoxication occurs only when the plasma osmolality is measured by freezing point depression; the osmotic contribution of volatile alcohols is not included when using a vapor pressure osmometer, which assumes that only water is in the vapor phase. (See "Serum osmolal gap" and "Methanol and ethylene glycol poisoning".)

- The presence of an anion gap metabolic acidosis may be the first clue to a toxic ingestion and should prompt consideration of etiologies such as salicylates, ethylene glycol, and methanol; serum creatinine, glucose, ketones, and lactate also should be measured to detect other causes of the anion gap acidosis. (See "Approach to the adult with metabolic acidosis" and "Salicylate (aspirin) poisoning in adults" and "Methanol and ethylene glycol poisoning".)

- The presence of an abnormally elevated serum creatinine with a normal BUN may be seen with isopropyl alcohol poisoning (or with diabetic ketoacidosis). High serum levels of acetone, which are formed as a metabolite of isopropyl alcohol, interfere with colorimetric creatinine assays, resulting in falsely elevated values [37]. (See "Isopropyl alcohol poisoning".)

- Measurement of isopropyl alcohol concentration in blood should be obtained in patients with an elevated osmolal gap without metabolic acidosis.

POISONING MANAGEMENT — Optimal management of the poisoned patient depends upon the specific poison(s) involved, the presenting and predicted severity of illness, and elapsed time between exposure and presentation. Treatment variably includes supportive care, decontamination, antidotal therapy, and enhanced elimination techniques. Assistance can be obtained from regional poison control centers. United States poison control centers can be reached through a toll-free line (1-800-222-1222). The World Health Organization (WHO) provides a listing of international poison centers at its website.

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. Copious water or saline irrigation for topical exposures and administration of activated charcoal for ingestions are the preferred methods of decontamination in most cases. In certain circumstances, other methods of gastrointestinal decontamination may be warranted, such as gastric lavage, whole bowel irrigation, endoscopy, surgery, dilution, and cathartics. (See "Topical chemical burns" and "Gastrointestinal decontamination of the poisoned patient", section on 'Activated charcoal'.)

The role of decontamination in the management of specific toxins is reviewed in topics devoted to the poisoning in question; general discussions of methods for decontamination, including the evidence for their effectiveness, are found separately. (See "Gastrointestinal decontamination of the poisoned patient" and "Enhanced elimination of poisons".)

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. Antidotes dramatically reduce morbidity and mortality in certain intoxications, but they are unavailable for most toxic agents and therefore are used in only a small fraction of cases [38].
Antidotes reduce or reverse poison effects by a variety of means. They may prevent absorption, bind and neutralize poisons directly, antagonize end-organ effects, or inhibit conversion to more toxic metabolites.

The pharmacokinetics of both the toxin and the antidote must be considered. Toxicity may recur if the antidote is eliminated more rapidly than the ingested substance, particularly if the antidote acts by antagonizing end-organ effects or inhibiting conversion to toxic metabolites. As an example, naloxone reverses opioid-induced somnolence and respiratory depression, but symptoms recur in approximately one-third of cases because the elimination half-life of naloxone is only 60 to 90 minutes [19,39] (see "Acute opioid intoxication in adults", section on 'Basic measures and antidotal therapy'). Thus, in certain situations antidotes may require repeated administration or continuous infusion.

Although a response to empirically administered antidotes can be used to confirm a suspected diagnosis, their indiscriminate use can potentially increase patient morbidity. As an example, routine administration of flumazenil to comatose patients suspected of benzodiazepine overdose may precipitate seizures, particularly if a proconvulsant drug has also been ingested, and is therefore NOT recommended. (See "Benzodiazepine poisoning and withdrawal", section on 'Antidote (flumazenil)').

**Enhanced elimination techniques** — Procedures to enhance elimination of poisons include forced diuresis, urine ion trapping, hemodialysis, hemoperfusion, hemofiltration, and exchange transfusion. Various measures are useful in selected circumstances. (See "Enhanced elimination of poisons").

**Supportive care** — Supportive care is the most important aspect of treatment and frequently is sufficient to effect complete patient recovery. Supportive care for the poisoned patient is generally similar to that utilized for other critically ill patients, but certain issues are managed slightly differently:

- **Airway protection** — Airway protection by endotracheal intubation should be performed early in the poisoned patient with depressed mental status, unless the cause is easily reversible (eg, opioid intoxication or hypoglycemia), because of the high risk for aspiration and its associated complications, particularly when gastric decontamination procedures need to be undertaken [40]. Tracheal intubation with mechanical ventilation is also indicated in the presence of severe acid-base disturbances or acute respiratory failure. Particularly when intubating a severely acidemic patient, it is important to prevent the development of a respiratory acidosis through inadequate minute ventilation. Occasionally, the management of high-grade physiologic stimulation may require sedation and/or paralysis with mechanical ventilation to limit the extent of complications such as hyperthermia, acidosis, and rhabdomyolysis. One rare exception to this important principle of aggressive airway management is salicylate poisoning, in which mechanical ventilation should be avoided unless absolutely necessary. (See "Salicylate (aspirin) poisoning in adults").

- **Hypotension** — Hypotension should be managed initially with boluses of isotonic intravenous fluids. Vasopressors are required when hypotension does not resolve with volume expansion. In general, direct-acting vasopressors, such as norepinephrine, are favored over indirect-acting agents, such as dopamine. The superiority of direct-acting agents has been demonstrated in the setting of tricyclic antidepressant poisoning [41,42]. (See "Tricyclic antidepressant poisoning").

- **Hypertension** — Hypertension in agitated patients is best treated initially with nonspecific sedatives such as benzodiazepines [43]. When hypertension necessitates specific therapy because of associated end-organ dysfunction, preferred treatments include calcium-channel blocking agents, phentolamine, labetalol, or nitroprusside. The use of beta-blockers alone for patients with sympathetic hyperactivity (eg, cocaine intoxication) is generally not recommended because it may result in unopposed alpha-adrenergic stimulation and intensified vasoconstriction [43,44]. The use of a beta-blocker after vasodilation has been achieved through alpha blockade (eg, phentolamine) or another vasodilator (eg, nitroprusside) is acceptable in these circumstances. In addition, a positive drug screen result for cocaine or amphetamines in an otherwise asymptomatic patient should not be considered a contraindication for beta-blockers. (See "Cocaine: Acute intoxication" and "Acute amphetamine and synthetic catathine ("bath salt") intoxication" and "Methamphetamine intoxication").

- **Ventricular tachycardia** — Sodium bicarbonate is first line therapy for ventricular tachycardias when they occur in the context of intoxication with tricyclic antidepressants or other membrane-active agents. Types IA (eg,
DISPOSITION

Following initial evaluation, treatment, and a short period of observation, disposition of the patient is based upon the observed and predicted severity of toxicity. Patients who develop only mild toxicity and who have only a low predicted severity can be observed in the emergency department until they are asymptomatic. An observation period of four to six hours is usually adequate for this purpose. Patients with moderate observed toxicity or those who are at risk for such on the basis of history or initial laboratory data should be admitted to an intermediate-care floor or an appropriate observation unit for continued monitoring and treatment. Patients with significant toxicity should be admitted to an intensive care unit (ICU) (table 20). All patients with intentional overdose require psychiatric assessment prior to discharge.

One retrospective study of 209 patients with drug overdoses suggested that clinical assessment in the emergency department could reliably identify patients who are at high risk for complications and require ICU care [49]. The presence of any of eight clinical criteria predicted a complicated hospital course that could be best managed in an ICU:

- PaCO2 >45 mmHg
- Need for emergency intubation
- Post-ingestion seizures

Bradyarrhythmia — Bradyarrhythmias associated with hypotension should be treated in the standard fashion with atropine and/or temporary pacing. However, in patients with calcium channel blocker or beta blocker intoxication, the administration of calcium, glucagon, or high dose insulin may obviate the need for further measures. Patients who have ingested clonidine are an exception to the general rule that direct-acting vasopressors are preferred over indirect-acting agents. Such patients have decreased sympathomimetic tone due to decreased release of sympathomimetic neurotransmitters, which is overcome by dopamine. (See "Calcium channel blocker poisoning" and "Beta blocker poisoning" and "Clonidine and related imidazoline poisoning").

Seizures caused by certain agents may require specific antidotes for their successful termination (eg, pyridoxine for isoniazid toxicity, glucose for hypoglycemic agents). (See "Beta blocker poisoning" and "Theophylline poisoning" and "Isoniazid (INH) poisoning" and "Metformin poisoning" and "Sulfonylurea agent poisoning").

Agitation — Drug-associated agitated behavior is generally best treated with benzodiazepines, followed by barbiturates if necessary. Phenytoin is typically not recommended to control seizures in poisoned patients [46]. Seizures caused by certain agents may require specific antidotes for their successful termination (eg, pyridoxine for isoniazid toxicity, glucose for hypoglycemic agents). (See "Beta blocker poisoning" and "Theophylline poisoning" and "Isoniazid (INH) poisoning" and "Metformin poisoning" and "Sulfonylurea agent poisoning").

Hyperthermia — Severe hyperthermia secondary to drug toxicity (eg, sympathomimetic overdose, serotonin syndrome, or neuroleptic malignant syndrome) may require aggressive treatment, possibly including ice water immersion [48]. Descriptions of such cooling techniques, including ice water immersion, are provided separately. (See "Severe nonexertional hyperthermia (classic heat stroke) in adults", section on 'Cooling measures' and "Exertional heat illness in adolescents and adults: Management and prevention", section on 'Cooling measures'.)
None of the 151 patients who lacked these risk factors developed a high-risk condition after admission and none required transfer to the ICU. The authors estimated that use of these predictors in similar patient populations could eliminate over one-half of intensive care days for poisoning without compromising the quality of care. A subsequent prospective study confirmed the importance of several of the criteria listed above (respiratory depression, hypotension, arrhythmia), and also noted that older age (over 61 years), abnormal body temperature, and suicidal intent were associated with an increased risk of death following poisoning [50].

ADDITIONAL RESOURCES — Regional poison control centers in the United States are available at all times for consultation on patients who are critically ill, require admission, or have clinical pictures that are unclear (1-800-222-1222). In addition, some hospitals have clinical and/or medical toxicologists available for bedside consultation and/or inpatient care. Whenever available, these are invaluable resources to help in the diagnosis and management of ingestions or overdoses. The World Health Organization (WHO) provides a listing of international poison centers at its website.

SUMMARY AND RECOMMENDATIONS — While poisonings can be fatal, the vast majority of patients presenting with a toxic exposure suffer minimal morbidity and recover fully. As a result, it is important to weigh the risks of interventions against the potential benefits which, for many patients, are relatively small. Assistance can be obtained from regional poison control centers. United States poison control centers can be reached through a toll-free line (1-800-222-1222). The World Health Organization (WHO) provides a listing of international poison centers at its website.

The essential elements of care for the poisoned patient include the following:

- Unresponsiveness to verbal stimuli
- Non-sinus cardiac rhythm
- Second- or third-degree atrioventricular block
- Systolic blood pressure less than 80 mmHg
- QRS duration ≥0.12 seconds

Institute stabilization procedures in patients with a compromised airway, inadequate gas exchange, or marginal hemodynamics. (See 'Initial evaluation and treatment' above.)

Obtain a thorough history of actual and potential exposures. Use information from friends, family, and prehospital personnel when available. Always inquire specifically about the use of over-the-counter drugs and traditional or herbal remedies in cases of intentional overdose. (See 'History' above.)

Perform a thorough physical examination to ascertain signs of a potential toxidrome as well as complications of the toxic exposure. Look for signs of trauma. The topic contains multiple tables that describe the association between particular physical findings and potential toxins. (See 'Physical examination' above.)

Perform a focused laboratory and radiographic evaluation that is guided by the severity of the patient's clinical status and the suspected toxins. Symptomatic patients and those with an unknown or unreliable history should, at a minimum, have measurements of serum electrolytes, renal function, and blood glucose. A urinalysis should also be performed. Calculation of the osmolal and anion gaps may help guide further evaluation of the toxic agent. An electrocardiogram should be performed on all patients who are symptomatic or who have been exposed to potentially cardiotoxic agents. Obtain a pregnancy test in women of child-bearing age. (See 'Radiographic studies' above and 'Other laboratory studies' above and 'Electrocardiography' above.)

Obtain a "toxic screen" of blood and/or urine in patients with severe or unexplained toxicity. Limited immunoassay screens are adequate for most cases of intentional overdose and provide less expensive and more timely results than comprehensive qualitative assays using gas chromatography or mass spectrometry. Screening for acetaminophen and salicylates is strongly recommended for patients with an uncertain history, intentional poisoning, or unexplained toxicity. Obtain quantitative drug concentrations if the results will help guide management. (See 'Toxicology screens (drug testing)' above.)

Supportive care in conjunction with decontamination procedures is sufficient for the vast majority of patients with toxic exposures. The decision to admit a patient with a toxic exposure to an intensive care setting should be based upon clinical criteria that relate to the stability of the airway, respiratory system, cardiovascular...
REFERENCES


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