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Physical Examination and Clinical Course of Poisoning and Drug Overdose

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ABSTRACT

Poisoning refers to the development of dose-related adverse effects following exposure to drugs, chemicals, or other xenobiotics. To paraphrase Paracelsus, the dose creates the poison. Although most poisons have predictable dose-related effects, individual responses to a given dose may vary due to inhibition in the presence of other xenobiotics, genetic polymorphism, enzymatic induction, or acquired tolerance. Poisoning may be local (e.g. lungs, skin, eyes) or systemic depending on the route of exposure, the physical and chemical properties of the poison, and its mechanism of action. The reversibility and severity of poisoning also depend on the functional reserve of the target organ or individual which is influenced by preexisting disease and age. The history should include the route, duration, time, and circumstances (surrounding events, location, and intent) of exposure; the amount and name of each chemical, drug, or ingredient involved; the severity of symptoms, time of onset, nature of symptoms; the time and type of first-aid measures given; and the medical and psychiatric history. In most cases, the patient is unaware of exposure, confused, comatose, or unable or unwilling to admit to one. Suspicious circumstances include unexplained sudden disease in a previously healthy person or a group of healthy people; a history of psychiatric problems (especially depression); current changes in health, social relationships, economic status, or the onset of disease during work with chemicals or after ingestion of drink (especially ethanol), food, or medications. When patients become sick soon after arriving from a foreign country or being arrested for criminal activity, “body packing” or “body stuffing” (ingesting or concealing illicit drugs in a body cavity) should be suspected. Relevant information may be available from friends, paramedics, family, police, pharmacists, physicians, and employers, who should be queried regarding the patient’s, behavioral changes, habits, hobbies, available medications, and antecedent events. Patients have to be asked explicitly concerning their prescribed drugs and recreational medication use. Drugs previously considered “illicit” such as cannabinoids are now legal in many places and prescribed for therapeutic purposes. A search of belongings, clothes, and places of discovery may unveil a suicide note or a container of chemicals or drugs. Without an apparent history in a patient clinically suspected to be poisoned, all drugs available anywhere in the patient’s home or belongings should be considered as possible agents, including drugs for pets.

The label on chemical products or the imprint code on drugs may be used to identify the potential toxicity of a suspected poison by consulting the manufacturer, a reference text, a computerized database, or a regional poison information center (800-222-1222). However, poisoning can mimic other illnesses, the correct diagnosis can usually be established by the history, physical examination, routine and toxicologic laboratory evaluations, and characteristic clinical course.

Keywords: drug overdose; poisoning; physical examination

INTRODUCTION & BACKGROUND

Drug overdose is a serious, harmful, and sometimes, fatal toxic reactions following ingestion of a certain drug. Chemical or drug overdose can occur accidentally or intentionally. It happens when a person takes more than the medically recommended dose of a prescription or over-the-counter drug. However, a few number of people may be very sensitive to particular medications so that the high-end of the therapeutic range of a drug may be toxic for them [1]. Illegal drugs, used to get high, may be taken in overdose amounts when a person's metabolism cannot detoxify the medication fast enough to avoid unintended side effects. Exposure to toxic substances, plants and chemicals that can cause harm are called poisonings. The longer the exposure or the higher the dose, the worse the poisoning. Drug overdose can involve people of any age [1]. It is easily occurred in very young children (from crawling age to about 5 years) and among teenagers to those in their mid-30s. Poisoning is a common reason for intensive care unit admission for both children and adults, and most poisoning victims are effectively treated using standard decontamination measures and supportive care. For a very small number of poisons, acceleration of toxin removal with hemofiltration or hemodialysis is indicated [2]. Also, specific antidotes are indicated in a few selected circumstances. Patients rarely benefit from more aggressive supportive techniques such as cardiopulmonary bypass [1].

Factors that can push one to drug overdose include:

• Drugs not kept out of the reach of children:
Inappropriately stored drugs can be an easy targets for children, who are curious and tend to put things in their mouth [2]. It is easy for them to get into and accidentally overdose on drugs that are not properly sealed or stored away from them.

• Not following Doctor's prescription:
Adults can also overdose on drugs if they fail to follow the instructions given to them [3]. Mistakenly taking drugs in excess or taking drugs sooner than directed can actually lead to an overdose of a substance that is otherwise safe for you.

• History of misuse or addiction:
Drug abuse or ingestion of illicit drugs can actually put you at risk of drug overdose, especially if it happens often or if you become addicted. This risk increases if you use multiple drugs, use them with alcohol or mix different drugs [3].

• History of mental disorders:
Mental diseases can also be risk factors for a drug overdose. Depression and suicidal thoughts can trigger overdose. This is extremely true if the symptoms are not being treated. Overdose, giving naloxone to patients with opioid overdose [2,3].

Physical examination and clinical course

The physical examination should concentrate originally on vital signs, neurologic system, and cardiopulmonary system.

The neurologic assessment should involve documentation of neuromuscular abnormalities like dyskinesia, dystonia, fasciculations, myoclonus, rigidity, and tremors [2]. The patient should furthermore be evaluated for evidence of trauma and underlying diseases. Focal neurologic findings are unusual in poisoning, and their existence should prompt examination for a structural central nervous system lesion. Assessment of the eyes for pupil size and reactivity, nystagmus, abdomen for bowel activity and bladder size, and skin for burns, bullae, color, warmth, moisture, pressure sores, and puncture marks, may reveal findings of diagnostic value [3]. When the history is not clear, all orifices should be examined for the presence of chemical burns and drug packets. The smell of breath and vomitus can provide significant diagnostic clues. Same as the color of skin, urine and nails. The diagnosis of poisoning in cases of unidentified etiology mainly depends on pattern recognition. The next step is to evaluate the, blood pressure, respiratory rate, pulse, temperature, and neurologic status and to define the overall physiologic state as stimulated, depressed, discordant, or normal. Obtaining a complete set of vital signs and reassessing them often are important. Measuring core temperature is particularly important, even in tough or aggressive patients, since temperature elevation is the most reliable prognosticator of poor finding in poisoning from stimulants e.g., cocaine or drug withdrawal e.g., alcohol or GHB [3]. Another step is to consider the underlying causes of the physiologic state and to attempt to recognize a pathophysiologic pattern or toxic syndrome (toxidrome) based on the observed findings. Assessing the stringency of the physiologic derangements is helpful in this regard and also for monitoring the clinical course and response to treatment [1]. The very last step is to attempt to discover the particular agent involved by looking for unique or relatively poison-specific physical or ancillary test abnormalities. Differences among toxidromes based on the physiologic state are outlined next.

• The Stimulated Physiologic State:
Increased blood pressure, pulse rate, temperature, neuromuscular activity, and respiratory rate characterize the stimulated physiologic state, which can indicate, anticholinergic, sympathetic, or hallucinogen poisoning or medication withdrawal. Mydriasis, a typical feature of all stimulants, is greatly marked in anticholinergic poisoning since pupillary reactivity depends on muscarinic control [2]. In cocaine poisoning (sympathetic poisoning), pupils are enlarged, but some reactivity to light remains. The anticholinergic toxidrome is also differentiated by hot, dry, flushed skin; reduced bowel sounds; and urinary retention [4].

• The Depressed Physiologic State:
Reduced, blood pressure, pulse rate, respiratory rate, temperature, and neuromuscular activity indicate the depressed physiologic state because of functionalsympatholytics (agents that reduce cardiovascular function and vascular tone as well as sympathetic activity), cholinergic (muscarinic and nicotinic) agents, opioids, and sedative-hypnotic γ-aminobutyric acid (GABA)-ergic agents. Miosis is common and is most noticeable in cholinergic and opioid poisoning [2,4].

Miosis is differentiated from other depressant syndromes by nicotinic and muscarinic signs and symptoms. Pronounced cardiac depression in the absence of notable CNS depression indicates a peripherally or direct acting sympatholytic [4]. However, in sedative-hypnotic and opioid poisoning, vital sign changes are usually secondary to depression in CNS respiratory and cardiovascular centers, and important abnormalities in these parameters do not happen until there is a marked reduction in the height of consciousness (grade 3 or 4 physiologic depression). Additional clues that indicate the cause of physiologic depression are conduction disturbances and cardiac arrhythmias (secondary to, calcium channel blockers, digitalis glycosides, antiarrhythmics, β-adrenergic antagonists, propoxyphene, and cyclic antidepressants), mydriasis (due to meperidine, tricyclic antidepressants, some antiarrhythmics, and diphenoxylate-atropine [Lomotil]), nystagmus (caused by sedative-hypnotics), and seizures (from cyclic antidepressants, cholinergic agents, propoxyphene) [4].

- The Discordant Physiologic State:

This state is characterized by mixed vital signs and neuromuscular abnormalities, as seen in poisoning by membrane-active agents, asphyxiants, CNS syndromes, and anion-gap metabolic acidosis (AGMA) inducers [4]. In these conditions, manifestations of physiologic stimulation and physiologic depression arise together or at different times during the clinical course. For example, membrane-active agents can result in hypotension, simultaneous coma, seizures, and tachyarrhythmias [2,4,5].

- The Normal Physiologic State:

This may be due to a psychogenic illness, nontoxic exposure or poisoning by toxic time-bombs: agents that absorb slowly, they are slowly distributed to their sites of action, require metabolic activation, or disrupt metabolic processes [5]. Because so many drugs have now been reformulated into once-a-day preparations for the patient's convenience and adherence, toxic time bombs are increasingly very common.

FIGURE 1: A photograph showing a person who had over dosed.

Laboratory assessment

Laboratory examination is very helpful in the differential diagnosis. A high anion gap metabolic acidosis (AGMA) is the most common in advanced salicylate, methanol and ethylene glycol Intoxication but can occur with each poisoning that leads to respiratory, hepatic, renal failure; seizures; or shock [4]. The serum lactate concentration is commonly low (less than the anion gap) in the former and high (nearly equal to the anion gap) in the latter. A low level of anion gap can be because of increased blood levels of magnesium, bromide, calcium, iodine or lithium. An elevated osmolal gap-a difference of >10 mmol/L between serum osmolality (measured by freezing-point depression) and osmolality calculated from serum sodium, glucose, and blood urea nitrogen levels-suggests the presence of a low-molecular-weight solute such as acetone; an alcohol (benzyl, ethanol, isopropanol, methanol); a glycol (diethylene, ethylene, propylene); ether (ethyl, glycol); or an unmeasuredcation (calcium, magnesium) or sugar (glycerol, mannitol, sorbitol). Ketosis suggests acetone, isopropyl alcohol, salicylate poisoning, or alcoholic ketoacidosis [6]. Hypoglycemia can be secondary to poisoning with quinine, salicylates, β- adrenergic blockers, ethanol, insulin and oral hypoglycemic agents, whereas hyperglycemia can occur in poisoning with N-3-pyridylmethyl-N′-p- nitrophenylurea (PNU [Vacor]), acetone, β-adrenergic agonists, caffeine, calcium channel blockers, iron and theophylline. Hypokalemia can be due to theophylline, barium, β-adrenergic agonists, caffeine, diuretics, or toluene; hyperkalemia suggests poisoning with a fluoride, α-adrenergic agonist, a β- adrenergic blocker or cardiac glycosides [7]. Hypocalcemia is often seen in oxalate, ethylene glycol, fluoride poisoning. PT and INR are useful for risk stratification in cases of warfarin or rodenticide poisoning, but are not to be relied on when evaluating overdose or complications from new anticoagulant pharmaceuticals (e.g., dabigatran). The electrocardiogram (ECG) may be useful for rapid diagnostic purposes [8-10]. Bradycardia and atrioventricular block may occur in patients poisoned by calcium channel blockers, α-adrenergic agonists, antiarrhythmic agents, beta blockers, cholinergic agents (carbamate and organophosphate insecticides), cardiac glycosides, lithium, or tricyclic antidepressants. QRS- and QT-interval prolongation may be caused by hyperkalemia, various antidepressants, and other membrane- active drugs [10]. Ventricular tachyarrhythmias is seen in poisoning with sympathomimetics, cardiac glycosides, fluorides, membrane-active drugs, methylxanthines, antidepressants, and agents that cause hyperkalemia or potentiate the effects of endogenous catecholamines (e.g., chloral hydrate, aliphatic and halogenated hydrocarbons) [8]. Radiologic investigations may be essential. Pulmonary edema (adult respiratory distress syndrome [ARDS]) can be caused by poisoning with a sedative-hypnotic, carbon monoxide, cyanide, an opioid, paraquat, phencyclidine, or salicylate; by inhalation of irritant gases, fumes, or vapors (acids and alkali, ammonia, aldehydes, chlorine, hydrogen sulfide, isocyanates, metal oxides, mercury, phosgene, polymers); or by prolonged anoxia, hyperthermia, or shock [4]. Aspiration pneumonia is common in patients with petroleum distillate aspiration, coma, and seizures. Chest x-ray is helpful for identifying complications from elemental mercury, or metal fume fever. The presence of radiopaque densities on abdominal CT scan or abdominal X-rays indicates the ingestion of potassium salts, enteric-coated tablets, calcium salts, chloral hydrate, chlorinated hydrocarbons, heavy metals, illicit drug packets, iodinated compounds or salicylates. Toxicologic examination of blood and urine (and occasionally of chemical samples and gastric contents) can sometimes confirm or rule out suspected poisoning [9]. Interpretation of laboratory data requires knowledge of the qualitative and quantitative tests used for screening and confirmation (fluorescence polarization, enzyme-multiplied and radio-immunoassays; colorimetric and fluorometric assays; gas-liquid, thin-layer, or gas chromatography; mass spectrometry; high-performance liquid chromatography; their sensitivity (limit of detection) and specificity, the preferred biologic specimen for analysis, and the optimal time of specimen sampling [8].

Treatment

The treatment purposes include support of vital signs, prevention of further poison absorption (decontamination), enhancement of poison elimination, administration of specific antidotes, and prevention of preexposure [11].

The specific treatment depends on the identity of the poison, the amount and route of exposure, the time of presentation relative to the time of exposure, and the severity of poisoning. Knowledge of the offending agents pharmacodynamics and pharmacokinetics is crucial [11]. In the course of the pretoxic phase, before the beginning of poisoning, decontamination is the highest priority, and treatment is based solely on the history. The maximal potential toxicity based on the greatest possible exposure should be determined. Since decontamination is more effective when the patient is asymptomatic and accomplished shortly after exposure, the initial history and physical examination should be brief and focused. Also, it is advisable to initiate cardiac monitoring, particularly in patients with potentially severe ingestions or unclear history, and establish IV access [12]. When a detailed history is not obtainable and a poison causing delayed toxicity (i.e., a toxic time-bomb) or irreversible damage is suspected, urine and blood should be sent for appropriate quantitative analysis and toxicologic screening [11]. During poison absorption and distribution, the level of blood may be greater than those in tissue and may not correlate with toxicity. However, high blood levels of agents whose metabolites are more toxic than the parent compound (methanol, acetaminophen or ethylene glycol) may suggest the necessity for supplementary interventions (dialysis, antidotes) [12-15]. A lot of patients who stay asymptomatic or who become asymptomatic 6hr after ingestion are improbable to develop subsequent toxicity and can be discharged safely. Longer observation will be crucial for patients who have ingested toxic time-bombs. During the toxic phase-the interval between the onset of poisoning and its peak effects-management is based primarily on clinical and laboratory findings [14]. Effects after an overdose usually begin sooner, peak later, and last longer than they do after a therapeutic dose [13,14]. A medications published pharmacokinetic profile in standard references such as the Physicians Desk Reference (PDR) is usually different from its toxicokinetic profile in overdose.

Resuscitation and stabilization are the initial priority [13]. Symptomatic patients should have an IV line placed and should undergo cardiac monitoring, oxygen saturation determination, and continuous observation. ECG, baseline laboratory and x-ray evaluation may also be appropriate. An Intravenous glucose, thiamine and naloxone should be considered in patients with altered mental status, especially those with seizures or coma. Decontamination should also be done, but it is unlikely to be effective in this phase than in the pretoxic phase. Measures which enhance poison elimination may reduce the duration and severity of the toxic phase. However, they are not without risk, which must be weighed against the potential benefit [13]. Diagnostic certainty (usually through laboratory confirmation) is generally a prerequisite. Intestinal dialysis with repetitive doses of activated charcoal can enhance the elimination of selected poisons e.g. carbamazepine, or theophylline. Urinary alkalinization can enhance the elimination of salicylates and a few other poisons [16-18]. Chelation therapy can also enhance the elimination of selected metals. Extracorporeal elimination methods are helpful for several poisons, but their risk and expense make their use reasonable only in patients who would otherwise have an unfavorable outcome [13]. In the resolution phase of poisoning, monitoring and supportive care should continue until clinical, laboratory, and ECG abnormalities have resolved. Because chemicals are eliminated sooner from the blood than from tissues, blood levels are generally lower than tissue levels during this phase and again may not correlate with toxicity. This variation applies particularly when extracorporeal elimination procedures are done. Redistribution from tissues may cause a rebound rise in the blood level after termination of these procedures (e.g., lithium) [14].

When a metabolite is responsible for toxic effects, continued treatment may be necessary in the absence of clinical toxicity or abnormal laboratory studies [16].

SPECIFIC TOXIC SYNDROMES AND POISONINGS

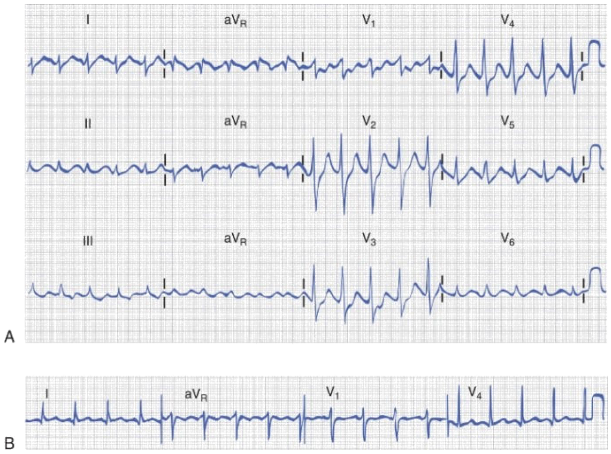
TABLE1: Analgesic and sedative toxin exposures.

Toxin Exposure	Basic Information	Clinical picture	Diagnosis	Treatment
Acetaminophen	Toxicity caused by reactive metabolite. Metabolite detoxified by glutathione	Hepatotoxicity: if ingestion > 7.5 g, less in alcoholics	Acetaminophen level Nomogram helpful in determining potential for toxicity over time	N- Acetlycystenine. Best results within 8 hours of exposure. May be beneficial up to 24 hours
Salicylate	Uncouples oxidative phosphorylation	Acute toxicity: Respiratory alkalosis, anion gap acidosis, Hyperthermia, coagulopathy, pulmonary edema, hyper- or hypoglycemia, seizures	Salicylate level: > 40 mg/dl in acute ingestion. >30 mg dl in chronic ingestion. potential toxicity by dose: <150 mg/kg low risk. >300 mg/kg severe toxicity. >500 mg/kg may be lethal	Hydration alkalinization of urine. Gastric lavage, activated charcoal hemodialysis (for seizures, refractory acidosis, or levels> 90 to 100 mg dl)
Opiates	Blind opiate receptors	Hypotension, respiratory depression, CNS depression	Urine testing for opiate metabolites	Naloxone Opiate antagonist Short- acting
Benzodiazepines	Blind the GABA receptor	Hypotension, respiratory depression, CNS depression, synergistic effect with opiates	Urine testing for metabolites	Flumazenil, Benzodiazepine receptor antagonist short acting. Use with caution. May precipate seizures in patients with long-term benzodiazepine use or concomitant TCA overdose.

Cardioactive Medications

i. Tricyclic antidepressants:

Possess α-adrenergic blocking, anticholinergic and adrenergic uptake-inhibiting properties. Ingestion of 10 to 20 mg/kg may result in moderate to severe toxicity, and 30 to 40 mg/kg may be life-threatening [14]. Best predictor of toxicity is QRS interval greater than 100ms. It presents with hypotension, arrhythmias, and anticholinergic effects (e.g., flushing, hyperthermia, dilated pupils, intestinal ileus, urinary retention, and sinus tachycardia) [10,14].



Central nervous system effects are common, with initial agitation followed by depressed consciousness and seizures. Treatment: Alkalinization to serum pH of 7.4 to 7.5 with IV sodium bicarbonate is indicated to reduce occurrence of arrhythmias [16]. If a patient is on the ventilator, may also hyperventilate. Carefully monitor pH during therapy (pH greater than 7.55 increases risk of seizures) [16].

ii. β-Blockers:

Has negative inotropic and chronotropic effects. Non-β1-selective agents may have respiratory effects, and lipophilic agents (e.g., propranolol) can cross blood-brain barrier, causing CNS depression. It presents with hypotension, bradycardia, and varying degrees of heart block. Treatment is with glucagon, IV fluids if hypotensive. If needed, pacing and vasopressor or inotrope with β1 activity [14].

iii. Calcium channel blockers:

Has negative inotropic and chronotropic effects. It presents with hypotension, bradycardia, and low cardiac output. Treatment is with 10% calcium chloride. If needed, pacing and vasopressor with β and α activity [18].

iv. Digitalis:

This is a therapeutic inotropic agent/antiarrhythmic with a narrow therapeutic window. It presents with nausea, malaise, weakness, bradycardia, heart block, hyperkalemia, visual complaints of seeing yellow halos around lights [17]. Treatment with digoxin-specific antibody is indicated for hemodynamically compromising conduction disturbances or for hyperkalemia. Calcium is relatively contraindicated because hypercalcemia can potentiate digitalis toxicity. Cardiac monitor done at least 6 hours after ingestion is necessary [19].

v. Theophylline:

Methylxanthine bronchodilator with a narrow therapeutic window. It presents with nausea and vomiting, agitation, seizures and arrhythmias, especially supraventricular [14]. Treatment with hemodialysis or charcoal hemoperfusion may be initiated for markedly elevated levels (more than 60 to 100 mg/mL), seizures, or persistently unstable hemodynamics. Supraventricular arrhythmias may be treated with β-blockers [20].

Toxins That Alter Hemoglobin/O2-Binding Properties

i. Carbon monoxide poisoning:

Colorless, odorless gas emitted by faulty heaters. It is seen primarily in winter months. It presents with dizziness, headache, weakness, confusion, dyspnea, chest pain. In severe cases; there maybe loss of consciousness, focal CNS symptoms, cardiac ischemia [21]. Lips and skin may appear cherry red, but this is an insensitive sign and diagnosis is made by measurement of carboxyhemoglobin. Treatment is by removing patient from the source, deliver 100% FiO2 by nonrebreathing mask. Hyperbaric O2 appears to prevent delayed neurologic sequelae in high-risk patients (loss of consciousness, confusion, lactic acidosis) [21-23].

ii. Methemoglobinemia:

Occurs when the ferrous ions (Fe2+) of heme are oxidized to the ferric (Fe3+) state that has a lower affinity for O2. Methemoglobin formation may be caused by exogenous agents, including sulfonamides, nitrates, topical anesthetics, antimalarials, and occupational exposures [23]. Higher levels of methemoglobin occur in individuals with deficiency of the cytochrome b5 reductase enzyme, which reduces Fe3+ to the Fe2+ state. It presents with hypoxia, cyanosis, and chocolate color of arterial blood [19]. It is suggested in a patient with a low pulse oximetry reading, but a normal arterial blood gas PaO2. Diagnosis is made by measurement of methemoglobin level by co-oximetry. Treatment: Discontinuation of the offending agent and administration of methylene blue to reduce the methemoglobin to hemoglobin [22].

TABLE 3: Alcohol-induced Toxicity.

Alcohol	Example	Clinical Presentation	Anion Gap	Osmolar Gap	Treatment
Methanol	Antifreeze Bootleg whiskey	Abdominal pain Nausea/vomiting Loss of vision	+	+	Fomepizole (alcohol dehydrogenase inhibitor).
Ethylene glycol	Antifreeze Windshield deicer	Ataxia Seizures Abdominal pain Nausea/vomiting	+	+	Fomepizole
Isopropanol	Rubbing alcohol	Abdominal pain Nausea/vomiting Headache Ataxia Coma	-	+	Fomepizole

CONCLUSIONS

A lot of poisoned patients seen in the accident and emergency department are adults with acute oral drug overdoses. However, other common clinical scenarios are accidental poisoning in children; drug abuse through smoking, snorting, or injection; chronic poisoning from drug abuse or from environmental, industrial, and agricultural chemical exposure; medication reactions or interactions; and envenomation. The clinical course of recovery of a poisoned patient depends on the quality of care received within the first few hours in the emergency setting, the etiologic substance, dose, underlying health condition of the person and age. With prompt and good medical care, most recover fully. The quick diagnosis and immediate intervention needed in patients with drug overdose or poisoning makes it pertinent that a clinician should be able to perform a meticulous clinical evaluation using the history, physical examination, and available laboratory tests to make a tentative diagnosis and commence life-saving treatment. Physical examination findings of importance include altered blood pressure, altered pulse, altered respiration and altered body temperature. Different poisonings and drug overdoses can affect those vital signs by either increasing or decreasing them. A neurological exam is indispensable as it can show the presence of coma, nystagmus, agitation, delirium and muscular weakness. The examination of the eye is also of importance in the acutely poisoned patient. Examination of the mouth may show signs of burns and you may note typical odors. The presence of flushing, increased diaphoresis and discoloration of the skin on examination might be the first clue to the toxicologic diagnosis.

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