CHAPTER 6

EMERGENCY TOXICOLOGY

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Abstract

Managing toxic emergencies requires a systematic approach beginning with rapid stabilization followed by specific interventions. Initial management follows the ABCDE (Airway, Breathing, Circulation, Disability, Exposure) framework, with particular attention to securing compromised airways, supporting ventilation, establishing vascular access, and addressing life-threatening dysrhythmias or hypotension. Simultaneously, attempts to identify the toxin involve examining vital sign patterns, recognizing toxidromes, and gathering exposure history from patients, bystanders, or prehospital personnel. Critical care management addresses organ system complications including hepatotoxicity, nephrotoxicity, cardiotoxicity, neurotoxicity, and metabolic disturbances. Targeted interventions for specific toxins are initiated while monitoring progresses from basic vital signs to advanced parameters including continuous ECG, capnography, invasive hemodynamic measurements, and serial laboratory studies tailored to the suspected agent. Particular toxins require specialized monitoring such as QRS duration in sodium channel blocker toxicity or anion gap in toxic alcohol ingestions. Comprehensive documentation encompasses initial presentation, interventions, response to treatment, consultation with poison centers or toxicologists, laboratory and diagnostic findings, and disposition decisions. This structured approach optimizes outcomes in poisoned patients while facilitating continuity of care, quality improvement, risk management, and potential medicolegal review.

Keywords: *Toxidromes, Critical Care, Antidotal Therapy, Toxicological Monitoring, Airway Management, Toxicology*

Learning Objectives

After completion of the chapter, the learners should be able to:

- Apply the ABCDE approach to initial stabilization of acutely poisoned patients with emphasis on toxin-specific airway and hemodynamic considerations.
- Recognize common toxidromes and vital sign patterns to guide early management decisions and toxin identification.
- Implement appropriate critical care interventions for organ-specific toxic effects including hepatotoxicity, nephrotoxicity, cardiotoxicity, and neurotoxicity.
- Select appropriate monitoring parameters for specific toxic exposures and interpret findings to guide ongoing management.
- Develop comprehensive documentation of toxic exposures that captures essential elements for clinical care, quality improvement, and potential legal proceedings.
- Formulate disposition plans for poisoned patients based on clinical stability, toxin characteristics, anticipated clinical course, and available monitoring capabilities.

INITIAL MANAGEMENT

The approach to acutely poisoned patients demands systematic assessment and intervention, addressing life-threatening complications while establishing foundations for definitive care.

Triage and Risk Assessment

Effective triage of potentially poisoned patients requires structured evaluation focusing on exposure characteristics, presenting symptoms, and risk factors for severe outcomes. Initial telephone triage, often conducted through poison control centers, evaluates exposure circumstances, substance identity and quantity, time elapsed since exposure, current symptoms, and pre-existing conditions affecting risk. This preliminary assessment determines whether home observation with monitoring suffices or healthcare facility evaluation becomes necessary. For patients presenting directly to healthcare facilities, rapid triage assessment focuses on vital sign abnormalities, level of consciousness, specific high-risk symptoms like seizures or dysrhythmias, and exposure to substances associated with delayed toxicity or rapid deterioration.

Risk stratification incorporates both toxicant-specific and patient-

specific factors. High-risk substances include calcium channel blockers, beta-blockers, cyclic antidepressants, opioids, hypoglycemic agents, and toxins with delayed onset of life-threatening effects like acetaminophen, certain mushrooms, and paraquat. High-risk exposure characteristics include large estimated doses, sustained-release formulations, multiple substance exposure, and unusual routes like injection or inhalation that bypass normal protective barriers. Patient risk factors include extremes of age, pregnancy, significant comorbidities affecting metabolic or eliminative functions, and lack of reliable observation capability for home management plans. This multifactorial assessment guides both triage categorization and initial resource allocation, including immediate bedside evaluation, continuous monitoring requirements, and anticipatory preparation for potential deterioration.

Table 6.1: Initial Management in Toxicological Emergencies

Priority	**Actions**	**Potential Complications**
Airway	Assessment, positioning, intubation if needed	Aspiration, respiratory arrest
Breathing	Oxygen, ventilatory support	Hypoxemia, hypercarbia
Circulation	IV access, fluid resuscitation, vasopressors if needed	Hypotension, dysrhythmias
Decontamination	Appropriate method based on route and timing	Aspiration, further absorption
Enhanced elimination	Consider in severe cases of dialyzable toxins	Complications of procedure
Targeted antidotes	Administration when indicated	Adverse effects of antidotes
Diagnostic testing	Targeted based on clinical presentation	Delayed diagnosis, missed toxins

Triage systems for poisoned patients increasingly incorporate structured toxicological triage tools alongside general emergency severity indices. The Poisoning Severity Score categorizes cases from asymptomatic (grade 0) through mild (grade 1), moderate (grade 2), and severe (grade 3), based on standardized criteria across multiple organ systems. Additional standardized assessment tools evaluate specific toxidrome presentations, pediatric poisoning severity, and risk for

delayed toxicity manifestation. These structured approaches enhance consistency in initial assessment and disposition decision-making across providers with varying toxicological experience levels.

Communication with regional poison centers provides critical triage support and management guidance, particularly valuable for uncommon exposures or complex scenarios. These centers maintain comprehensive toxicant databases, case registries documenting previous similar exposures, and specialists experienced in risk assessment for diverse poisonings. Early poison center consultation enables appropriate resource utilization through evidence-based recommendations regarding necessary observation periods, laboratory evaluations, and interventions based on specific toxicant characteristics and exposure circumstances. This collaboration enhances both individual patient care and public health surveillance for emerging toxicological threats or product hazards.

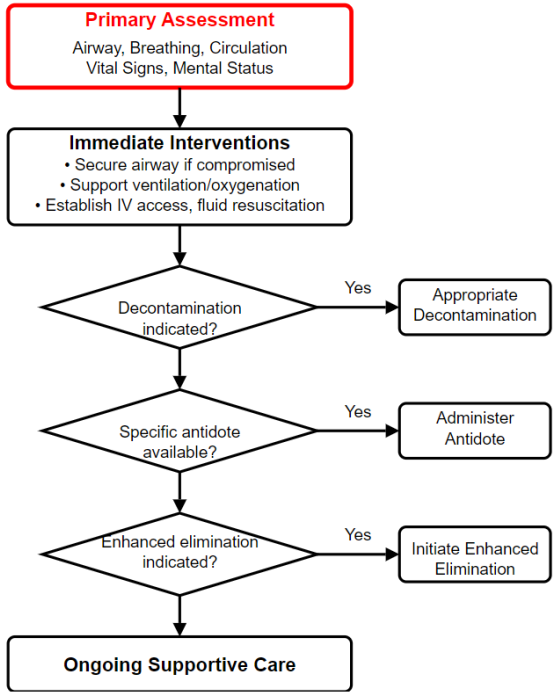

Figure 6.1: Initial Management Algorithm for Toxicological Emergencies

Resuscitation Priorities

Initial stabilization of poisoned patients follows modified advanced life support principles addressing toxicant-specific pathophysiology. Airway assessment carries particular importance given the frequency of mental status alteration and protective reflex impairment in toxicological emergencies. Indications for early intubation include Glasgow Coma Scale below 8, inability to maintain patent airway, progressive deterioration in respiratory status, severe agitation requiring sedation, and anticipation of complications requiring airway protection based on specific toxicant properties. Rapid sequence intubation protocols for poisoned patients typically employ etomidate or ketamine for induction, minimizing additional hemodynamic compromise in potentially unstable patients, with special consideration for potential drug interactions with specific toxicants.

Remember

Initial management of poisoned patients follows the ABCDE approach (Airway, Breathing, Circulation, Disability, Exposure) with emphasis on early airway protection in patients with decreased consciousness or risk of aspiration

Respiratory support addresses multiple pathophysiological processes including central respiratory depression from sedative-hypnotics or opioids, bronchospasm from cholinergic or irritant exposures, and non-cardiogenic pulmonary edema from various toxicants including salicylates and opioids. Oxygen administration targets saturation above 94% in most scenarios, with 100% oxygen indicated for carbon monoxide poisoning to accelerate carboxyhemoglobin dissociation. Ventilation strategies require toxicant-specific modifications, particularly in salicylate poisoning where hyperventilation provides compensatory alkalosis that should be maintained during mechanical ventilation to prevent paradoxical acidosis and increased central nervous system toxicity.

Circulatory support begins with establishing reliable vascular access, typically through two large-bore peripheral intravenous lines, with central venous access considered for patients requiring vasopressor support or specialized monitoring. Hypotension management follows a stepwise approach beginning with crystalloid fluid resuscitation (typically 20 mL/kg initially), followed by vasopressor support if inadequate response occurs. Vasopressor selection considers toxicant-specific mechanisms: norepinephrine for distributive shock from vasodilatory toxins, epinephrine for bradycardia-associated hypotension from beta-blockers or calcium channel blockers, and pure

alpha-agonists like phenylephrine for toxicants causing both vasodilation and tachycardia. Specific antidotal therapy often proves more effective than conventional hemodynamic support alone, exemplified by calcium, glucagon, and high-dose insulin therapy for calcium channel blocker and beta-blocker toxicity.

Neurological emergencies in poisoned patients require aggressive management, with seizures representing a common complication of numerous toxicants. Initial treatment employs benzodiazepines at standard or increased doses, recognizing that toxicant-induced seizures often prove more resistant to standard anticonvulsant regimens. Second-line agents include phenobarbital or propofol rather than phenytoin, which shows limited efficacy against most toxicant-induced seizures. Specific antidotal therapy addresses underlying mechanisms in certain poisonings, exemplified by pyridoxine administration for isoniazid-induced seizures refractory to conventional anticonvulsants. Status epilepticus unresponsive to standard measures may require rapid progression to general anesthesia with continuous electroencephalogram monitoring, particularly when neuromuscular blockade masks motor manifestations of ongoing seizure activity.

Diagnosis

Initial diagnostic evaluation incorporates focused history-taking, physical examination targeting toxidrome recognition, and selected laboratory and imaging studies guided by clinical presentation. History acquisition, often challenging in poisoned patients due to altered mental status or intentional deception, emphasizes toxicant identification, quantity, timing, coingestants, and available medical history. Collateral information sources including family members, emergency medical services personnel, prescription databases, and scene observations often provide critical details unavailable from the patient. Physical examination focuses on vital sign patterns and characteristic toxidrome manifestations, with particular attention to pupillary response, mucous membrane moisture, bowel sounds, skin characteristics, and neuromuscular findings that may suggest specific toxicant classes.

Laboratory evaluation begins with essential studies indicated for most significant poisonings: comprehensive metabolic panel assessing electrolytes, renal and hepatic function; complete blood count; coagulation studies; arterial blood gas analysis; urinalysis; and pregnancy testing for women of childbearing potential. Toxicant-specific testing follows a targeted approach guided by clinical presentation and suspected exposures. Quantitative levels for specific toxicants like acetaminophen, salicylates, digoxin, lithium, iron, carbamazepine, and valproic acid provide critical management guidance when corresponding toxicity is suspected. Toxicology

screening may include immunoassay-based urine drug screens for common substances of abuse, recognizing the significant limitations in sensitivity, specificity, and substance coverage of these tests. Expanded toxicological testing requires consultation with laboratory services and consideration of send-out testing to reference laboratories when results would meaningfully impact management.

Electrocardiography provides essential assessment in suspected poisonings, with specific attention to rate, rhythm, intervals, and morphological abnormalities suggesting particular toxicants. QRS prolongation (>100 ms) suggests sodium channel blockade from agents like tricyclic antidepressants or class Ia antiarrhythmics. QT prolongation indicates potassium channel effects from numerous medications including antipsychotics, methadone, and class III antiarrhythmics. Specific dysrhythmias may suggest particular toxicants: bidirectional ventricular tachycardia suggesting digitalis glycoside toxicity; torsades de pointes indicating QT-prolonging agents; and bradydysrhythmias suggesting beta-blockers, calcium channel blockers, or cholinergic excess. Serial electrocardiograms provide valuable trending information, particularly for cardiotoxic substances with delayed manifestations.

Imaging studies serve targeted purposes in selected poisoning scenarios. Abdominal radiographs may identify radio-opaque tablets, foreign body ingestions, or characteristic patterns like the "string of pearls" appearance in iron tablet ingestion. Chest radiography evaluates for aspiration pneumonitis, non-cardiogenic pulmonary edema, and pneumothorax from inhalational exposures. Computed tomography of the brain remains the preferred neuroimaging modality for altered mental status evaluation, excluding structural lesions mimicking toxicological presentations and identifying complications like cerebral edema in specific poisonings. Bedside ultrasonography increasingly provides valuable assessment of cardiac function in cardiotoxic exposures, volume status in conditions with significant fluid shifts, and presence of foreign bodies in selected ingestions.

Early Interventions

Time-sensitive interventions often prove critical in poisoned patients, with early administration of certain antidotes substantially improving outcomes. Naloxone administration for suspected opioid toxicity presenting with respiratory depression follows a titrated approach, beginning with 0.4 mg intravenously in adults with escalation as needed to restore adequate respiratory status without precipitating severe withdrawal in opioid-dependent individuals. Higher initial doses (2-4 mg) may be warranted with suspected potent synthetic opioid exposures like fentanyl analogs. Dextrose administration (25 g

intravenously in adults) addresses hypoglycemia, a common contributor to altered mental status either independently or concurrent with toxicant exposure. Thiamine supplementation (100 mg intravenously) should precede dextrose administration in malnourished or alcoholic patients to prevent precipitation of Wernicke's encephalopathy.

Table 6.2: Critical Toxicological Syndromes Requiring Immediate Intervention

Syndrome	Priority Interventions	Monitoring Parameters
Severe sympathomimetic toxicity	Benzodiazepines, cooling, fluid resuscitation	Temperature, BP, HR, CK, renal function
Opioid overdose	Airway management, naloxone, ventilatory support	Respiratory rate, oxygen saturation, level of consciousness
Organophosphate poisoning	Atropine, pralidoxime, benzodiazepines	Secretions, respiratory status, cholinesterase levels
Calcium channel blocker toxicity	Calcium, high-dose insulin euglycemia therapy, vasopressors	BP, HR, glucose, calcium, cardiac output
Toxic alcohol poisoning	Fomepizole, bicarbonate, hemodialysis	Anion gap, osmolal gap, pH, methanol/ethylene glycol levels
Salicylate toxicity	Alkalinization, fluid resuscitation, hemodialysis in severe cases	Salicylate level, pH, mental status, temperature
Cyanide toxicity	Hydroxocobalamin or sodium thiosulfate	Lactate, vital signs, pH, cardiac function

Decontamination procedures, when indicated, require prompt implementation for maximal effectiveness. Activated charcoal administration (typically 1 g/kg orally or via nasogastric tube) provides greatest benefit when administered within 1-2 hours of ingestion for most toxicants, with possibly extended efficacy for sustained-release preparations or substances undergoing enterohepatic circulation.