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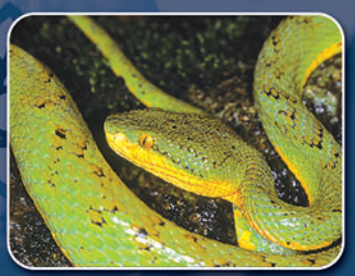
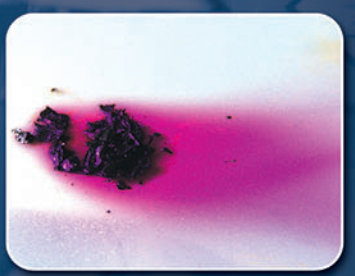
MODERN MEDICAL TOXICOLOGY

Completely Updated, Revised and Profusely Illustrated

VV Pillay



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CHAPTER

2 Diagnosis of Poisoning

JAYPEE BROTHERS

INTRODUCTION

A poisoning case can present to a doctor or hospital in any one of a number of ways. Broadly, there are four types of presentation:

1. **Fulminant:** Produced by a massive dose. Death occurs very rapidly, sometimes without preceding symptoms, the patient appearing to collapse suddenly.
2. **Acute:** Produced by a single dose or several small doses taken in a short period. Onset of symptoms is abrupt.
3. **Chronic:** Produced by small doses taken over a long period. Onset is insidious.
4. **Subacute:** Characterized by a mixture of features of acute and chronic poisoning.

The majority of poisoned patients presenting to the casualty (emergency) department are victims of acute exposure. Most of them are usually coherent enough to tell the doctor what the problem is, and indeed what they have taken or been exposed to. However, in an unconscious or uncooperative patient the diagnosis will have to be made on the basis of circumstantial or third party evidence. It is important to interrogate the persons accompanying the patient (relatives, friends, ambulance personnel, etc.), and to contact his or her family doctor as soon as possible. In spite of all this, unfortunately, in a significant proportion of cases the diagnosis remains uncertain. This is because unlike in other clinical conditions arising out of natural disease, there are only a very few syndromes (**toxidromes**)* characterized by specific signs and symptoms (**Table 2.1**). In most cases, the poisoned patient presents with one or more of the following nonspecific features:

- Impairment of consciousness
- Respiratory/Cardiovascular depression
- Dehydration due to vomiting/diarrhea
- Hypothermia
- Convulsions
- Cardiac arrhythmias

However, there are some valuable clues afforded on detailed clinical examination, which can help narrow down the differential diagnosis. Most of these will be dealt with in a subsequent section (**Chapter 3: General Management**), but a few are discussed here for the sake of convenience.

- **Ocular clues**: Several drugs/poisons affect the pupils of the eyes producing either miosis or mydriasis. A few produce nystagmus. These have been laid out in **Table 2.2**. Normally, both the pupils are equal in size, 3–4 mm under typical conditions, round, and react directly as well as consensually to increased light intensity by constricting. Pupillary constriction also occurs as part of the near reflex when a person focuses on near objects. All these functions result from the balance between cholinergic innervation of the iris sphincter (constrictor) by the oculomotor nerve, and sympathetic innervation of the radial muscle of the iris (dilator). Mydriasis can occur due to increased sympathetic stimulation by endogenous catecholamines or from systemic or ocular exposures to sympathomimetic drugs. Mydriasis can also result from inhibition of cholinergic- mediated pupillary constriction. Because pupillary constriction in response to light is a major determinant of pupil size, blindness from ocular, retinal, or optic nerve disorders also leads to mydriasis. Pupillary constriction or miosis can result from increased cholinergic stimulation, or inhibition of sympathetic dilation. Other ophthalmological manifestations along with their respective causes are mentioned in **Table 2.3.**
- **Olfactory clues**: Some poisons have distinctive odors, which may be perceived in the vicinity of a poisoned patient, especially in the breath. Some important examples are mentioned in **Table 2.4**.
- **Dermal clues**: Some poisons have characteristic dermal manifestations in acute toxicity, while certain others demonstrate skin signs on chronic exposure (**Table 2.5**).

*Some toxicologists prefer to restrict the term "***toxidrome***" to a group of signs and symptoms that is consistently encountered in relation to a specific toxin, e.g., paracetamol, salicylates, methanol, carbon monoxide, etc.

Table 2.1: Toxidromes (Toxic syndromes).

Syndrome	*Causes*	*Symptomatology*
Anticholinergic syndrome	Antihistamines, antiparkinsonian drugs, atropine, scopolamine, amantadine, antipsychotic drugs, antidepressants, antispasmodics, skeletal muscle relaxants, many plants (especially Datura), and fungi (e.g., *Amanita muscaria*)	Delirium, tachycardia, dry hot skin, mydriasis, myoclonus, urinary retention, decreased bowel sounds, convulsions, and arrhythmias
Cholinergic syndrome	Organophosphates, carbamates, parasympathomimetic drugs, and some mushrooms	Confusion, CNS depression, salivation, lacrimation, urinary and fecal incontinence, vomiting, sweating, fasciculations, seizures, miosis, pulmonary edema, tachy/ bradycardia
Sympathomimetic syndrome	Cocaine, amphetamines, upper respiratory decongestants (phenylpropanolamine, ephedrine, and pseudoephedrine)	Paranoia, delusions, tachycardia, hypertension, hyperpyrexia, sweating, mydriasis, seizures, arrhythmias
Sedative-hypnotic syndrome	Opiates, barbiturates, benzodiazepines, ethanol, methaqualone, meprobamate, ethchlorvynol, glutethimide, clonidine	Miosis, hypotension, bradycardia, hypothermia, CNS depression, hyporeflexia, coma, convulsions (rarely)
Opioid syndrome	Morphine, codeine, pethidine (meperidine), pentazocine,buprenorphine, propoxyphene, tramadol, butorphanol, dextromethorphan, diphenoxylate, loperamide, heroin, fentanyl	Miosis, hypotension, bradycardia, bradypnea, hypothermia, CNS depression, hyporeflexia, decreased peristalsis, mild diaphoresis, urinary retention (occasionally), coma, convulsions (rarely)
Salicylate syndrome	Salicylic acid, methyl salicylate, homosalate (homomenthyl salicylate), trolamine salicylate, acetyl salicylic acid, sodium salicylate, sodium aminosalicylate, bismuth subsalicylate, mesalamine (5-aminosalicylic acid), olsalazine (sodium azodisalicylate), sulfasalazine (salicylazosulfapyridine), diflunisal, benorylate (4-acetamidophenyl-o-acetylsalicylate)	Tachycardia, tachypnea, hyperthermia, sweating, delirium, decreased peristalsis, tinnitus, and convulsions
Hallucinogen syndrome	LSD (lysergic acid diethylamide), phencyclidine, ketamine, designer drugs: MDA (adam), MDEA (eve), MDMA (ecstasy), α-methyl fentanyl, tryptamines (dimethyl tryptamine, psilocin, psilocybin), mescaline, cannabinols	Tachycardia, hypertension, hyperthermia, agitation, psychotic behavior, miosis or mydriasis, hallucinations
Serotonin syndrome	Drugs that inhibit serotonin breakdown (non-selective MAOIs, e.g., phenelzine, moclobemide, clorgyline, isocarboxazid), drugs that block serotonin reuptake (SSRIs, e.g., fluoxetine, citalopram, paroxetine, fluvoxamine, and sertraline; as well as cocaine, dextromethorphan, pethidine, pentazocine, clomipramine, trazodone, venlafaxine), serotonin precursors or agonists (LSD, tryptophan), drugs that enhance serotonin release (cocaine, amphetamines, lithium, mirtazapine, buspirone)	Tachycardia, hypertension, hyperthermia, tachypnea, agitation, confusion, mydriasis, sweating, flushed skin and mocosae, tremor, myoclonus, hyper-reflexia, rigidity, trismus
Ethanol withdrawal syndrome	Withdrawal reaction in an alcoholic	Tachycardia, hypertension, hyperthermia, tachypnea, agitation, confusion, disorientation, mydriasis, sweating, diarrhea, tremor, convulsions
Opioid withdrawal syndrome	Withdrawal reaction in an opiate/opioid addict	Tachycardia, hypertension, fever, anxiety, mydriasis, sweating, vomiting, diarrhea, rhinorrhea, piloerection

Table 2.2: Drugs/poisons producing pupillary changes.

Miosis	*Mydriasis*	*Nystagmus*
Barbiturates	Alcohol (constricted in coma)	Alcohol
Benzodiazepines	Amphetamines	Barbiturates
Caffeine	Antihistamines	Carbamazepine
Carbamates	Carbon monoxide	Phencyclidine
Carbolic acid (Phenol)	Cocaine	Phenytoin
Clonidine	Cyanide	
Methyl dopa	Datura (Atropine)	
Nicotine	Ephedrine	
Opiates		
Organophosphates		
Parasympathomimetics		

Table 2.3: Toxic ophthalmological manifestations.

Feature	*Cause*
Diplopia	Barbiturates, cannabis, ethanol, opiates, phenytoin, tetracycline, vitamin A
Blurred vision	Alcohol, anticholinergics, botulism, ethanol, lithium, MAOIs, methanol
Altered color perception	Cannabis, CO, digitalis, hydrocarbons, ibuprofen, nalidixic acid
Corneal deposits	Chloroquine, vitamin D
Oculogyric crisis	Phenothiazines, butyrophenones, metoclopramide
Optic neuritis	Chloroquine, digitalis, disulfiram, ergot, heavy metals, methanol, penicillamine, quinine

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Table 2.4: Diagnostic odors.

Odor	*Substance*
Acetone (apple-like)	Chloroform, ethanol, isopropanol, lacquer
Acrid (pear-like)	Chloral hydrate, paraldehyde
Bitter almond	Cyanide
Burnt rope	Marijuana (Cannabis)
Coal gas	Carbon monoxide (CO)
Disinfectant (hospital odor)	Carbolic acid, creosote
Garlicky	Arsenic, dimethylsulfoxide, organophosphates, phosphorus, selenium, tellurium, thallium
Mothballs	Camphor, naphthalene
Musty (fishy)	Aluminum phosphide, zinc phosphide
Rotten egg	Carbon-di-sulfide, disulfiram, hydrogen sulfide, mercaptans, N-acetylcysteine
Shoe polish	Nitrobenzene
Vinegar	Acetic acid
Wintergreen	Methyl salicylate

Table 2.6: Drug-induced oral manifestations.

Feature	*Drug /Poison*
Glossitis	Trimethoprim–sulfamethoxazole, diclofenac, naproxen, metronidazole, amoxycillin, erythromycin, piroxicam
Stomatitis	Cytotoxic drugs, penicillamine, gold salts, gentian violet dye
Sialadenitis	Phenylbutazone, isoproterenol, nitrofurantoin, iodine
Parotitis	Methyl dopa, clonidine, phenyl and oxyphenbutazone, thioridazine
Gingival hyperplasia	Phenytoin, sodium valproate, phenobarbitone, nifedipine, diltiazem, verapamil
Pigmentation	Cisplatin, oral contraceptives, antimalarials
Dental discoloration	Fluorides, tetracycline, chlorhexidine, iron tonic syrups
Dental caries	Cough and vitamin syrups, antibiotic suspensions
Xerostomia	Antipsychotics, tricyclics, antihistamines, anticholinergics, anticonvulsants, narcotics, diuretics, centrally acting antihypertensives
Sialorrhea	Parasympathomimetics, iodides

Table 2.5: Dermal manifestations of poisoning.

Acute		*Chronic*	
Poison/Drug	*Feature*	*Poison/Drug*	*Feature*
Datura, atropine	Dry, hot skin	Heroin, barbiturates, morphine, phencyclidine	Needle marks
Organophosphates, salicylates, arsenic, LSD, dinitrophenol	Profuse sweating	Bromides, iodides, coal tar products, phenytoin	Acne, brown color
Carbon monoxide (CO)	Cherry pink color	Arsenic	Rain drop pigmentation, hyperkeratosis, exfoliative dermatitis
Cyanide	Brick red color	Chlorinated hydrocarbons	Eczematous dermatitis
Barbiturates, CO, imipramine, methadone, nitrazepam	Blisters	Chloroquine, busulfan, clofazimine, phenothiazines, phenytoin	Dark pigmentation
Warfarin	Petechiae and purpuric spots	Bromides, iodides, oral contraceptives, penicillin, salicylates, tetracycline	Erythema nodosum
Clonidine, ergot, niacin, sympathomimetics, theophylline	Flushing		

There have been cases reported of unexpected dermal manifestations from ingestion of chemicals such as kerosene. Several therapeutic drugs produce irritant dermatitis even in nontoxic doses, e.g., most antibiotics, INH, phenothiazines, sulfonamides, thiazides, NSAIDs, etc.

- **Oral clues**: Careful examination of the mouth can afford valuable information about the etiology of poisoning in some cases (**Table 2.6**).

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Highlights

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- Only book that features in the WHO list of recommended books on Medical Toxicology for the Indian sub-continent
- New edition has been completely revamped with brand new current information presented in a totally different format
- Every commonly encountered xenobiotic (poison/toxin/drug/chemical) has been dealt with in a comprehensive manner regarding to source, physical appearance, toxicokinetics, fatal dose, signs and symptoms, diagnostic methods and treatment modalities, besides forensic and medico-social issues
- Incorporation of about 500 color images and several hundred tables, boxes and bulleted lists
- Each chapter ends with 10–100 or more recent references for further reading and research
- Recent developments on the diagnosis and management of poisoning, including the use of extracorporeal membrane oxygenation (ECMO), lipid emulsion therapy, etc., have been added
- The most trusted handbook on poisoning/overdose for physicians and medical students, now in a new *Avatar*!

VV Pillay MD DCL is currently the Chief of Poison Control Centre; Head, Analytical Toxicology, and Professor and Head, Department of Forensic Medicine and Medical Toxicology, Amrita School of Medicine (Amrita Vishwa Vidyapeetham), Kochi, Kerala, India. He obtained his MBBS and MD from Gandhi Medical College, Hyderabad, Telangana and worked in various capacities in medical colleges across India before settling down in Kochi. He has more than hundred publications in scientific journals. He is also the Editor of the hugely popular *Textbook of Forensic Medicine and Toxicology*, which is now entering the 20th edition. In 2003, Dr Pillay published *Comprehensive Medical Toxicology* which is the most exhaustive reference book on toxicology ever written by an Indian author, now in its 3rd edition. He has also contributed to several other books, including a section on Indian poisonous plants for the *Oxford Textbook of Medicine*.

In a career spanning more than 3 decades, Dr Pillay has been conferred with several accolades, including an award from Society of Toxicology, USA at the 57th Annual Meeting in Texas, USA in March 2018, and Lifetime Achievement Award for Contribution to Medical Toxicology by Indian Society of Toxicology in December 2021. He is a much sought-after orator on medical toxicology and has travelled extensively to deliver more than 200 invited lectures in India and abroad.

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