CME MODULE ON TOXICOLOGY

FOR MEDICAL OFFICERS OF UTTAR PRADESH

SIHFWUP

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CME MODULE ON
TOXICOLOGY
FOR MEDICAL OFFICERS OF UTTAR PRADESH

**STATE INSTITUTE OF HEALTH AND FAMILY WELFARE,
UTTAR PRADESH**

**In collaboration with State Medico Legal Cell,
Medical & Health Services, Uttar Pradesh**

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Processed and Realization


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MESSAGE

Shri Brajesh Pathak
Hon'ble Deputy Chief Minister
Hon'ble Minister of
Medical Health and Family Welfare
Department
Government of Uttar Pradesh

Forensic science is a boon to the crime field investigations in today's world. With the help of this branch of science it becomes so easy or accurate to solve a mystery of cases like suicidal, homicidal accidental or other crime cases. By using techniques of forensic science we can also identify various poisons. Toxicological clinician such as Medical Officers posted in the districts needs to know how to identify various poisons and poisonous conditions to treat the patients.

This CME Module on Toxicology reviews the introduction, application of various toxicological tests with upgraded scientific innovations in field of forensic medicine. This Module on Toxicology reveals the international scientific methods and tools of investigation that will not only enhance the skills of Medical Officers of Uttar Pradesh Health Services but also bring standard forensic investigation practices in Uttar Pradesh

I congratulate Director, SIHFW for his continuous efforts in shaping the skills of Medical Officers of Uttar Pradesh Health Services matching with international standards through this continuing in-service medical education and enhancing core competencies of medical officers related to Medical and Health Services.

(Brijesh Pathak)


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MESSAGE

Shri Mayankeshwar Sharan Singh

Hon'ble State Minister
Medical Health and Family Welfare Department
Government of Uttar Pradesh

Forensic toxicology is a scientific discipline which studies the medical and legal aspects of the harmful effects of a substance on organisms, and by chemical and analytical methods helps to establish facts in forensic investigations. Forensic practice is particularly significant for fatal poisoning, as well as those that may be related to the commission of criminal acts.

The common investigative mishaps that occur in major autopsies and postmortem are due to lack of knowledge with new and recent updates in toxicological methods and investigative tools used internationally under forensic protocols.

I thank team SIHFW and State Medico-Legal Cell for developing CME on Toxicology for Medical Officer's of Uttar Pradesh to conduct international and standardize toxicological tools at the best of their abilities and take part in the continuing contribution in the field of public health services.

(Mayankeshwar Sharan Singh)


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FOREWARD

Shri Partha Sarthi Sen Sharma
Principal Secretary
Department of
Medical Health and Family Welfare
Government of Uttar Pradesh

India is a developing nation and presents demographic features quite similar to other developing countries. Emerging occupational and epidemiological health problems are major priorities that need to be tackled along with existing traditional public health problems like communicable diseases, malnutrition, poor environmental sanitation, and inadequate medical care. Although environmentalists and toxicologists in general, and the Government of India in particular, have expressed deep concern and have prioritized these issues, public awareness, toxicological databases, and suitable preventive measures are still lacking.

Therefore, this CME Module on Toxicology is an essential learning material to enhance the skills and core competencies of our Medical Officers posted in field. I am sure these investigative methods in the Module with international approach to toxicology will benefit our officers in the field to identify various poisons and poisonous conditions to treat the patients accurately.

(Partha Sarthi Sen Sharma


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MESSAGE

Dr. Renu Srivastava Varma
Director General
Department of
Medical Health and Family Welfare
Government of Uttar Pradesh

One of the foremost requirements to improve the existing knowledge regarding the deleterious effects of toxicants and their preventive or therapeutic measures is to have the latest technologies, amenities, and resources for detecting toxic substances or their derivatives at nanogram (ng) levels. This step would aid to precisely quantify various toxicants and further facilitate research to develop (i) better therapeutic or preventive regimes and (ii) biomarkers to examine effects at the molecular level

The State Medico Legal Experts and faculties at State Institute of Health & Family Welfare, Uttar Pradesh have taken a great deal of care in preparing this Module for Continuing Medical Education CME) to help Government Medical Officers to stay up-to-date with the latest regulations and best practices in the field of toxicology, whilst ensuring that they are in compliance with all relevant laws and standards.

(Dr. Renu Srivastava Varma)


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MESSAGE

Dr. Anita Joshi
Director General Family Welfare
Directorate of Family Welfare
Government of Uttar Pradesh

Toxicology is the study of the adverse effects of chemical, physical or biological agents on living organisms and the ecosystem, including the prevention and amelioration of such adverse effects. Toxicology has emerged as an important area of biomedical research today, the importance of which has already been felt more and more in every corner of the world.

This major area of health research provides information not only on how to safeguard our own health but also the health of the environment around us. The personnel working in this area cut across a broad spectrum of scientific and technological disciplines, from biology to medicine, engineering to molecular biology, chemistry to mathematical modeling.

I congratulate the team SIHFW and Team State Medico Legal Cell for developing such a resourceful document blended with latest technological and chemical research.

(Dr. Anita Joshi)


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MESSAGE

Dr. Deepa Tyagi
Director General (Training)
Medical, Health & Family Welfare
Government of Uttar Pradesh

In recent years, it has been realized that most of the diseases related to toxicants are incurable; therefore, the best course of action in dealing with them is prevention. The economic benefits and incurable nature of these diseases need to be highlighted, besides proposing investment in safety and remedial programs. In the Indian context, one of the major obstacles to circumventing the above problem is literacy. Most of the population that suffers from these diseases are illiterate and are unaware of the hazards associated with the toxicants. Because of a gradual increase in the literacy rate in recent years, the general population in India is becoming aware of the toxicological consequences of substances around them. This Module highlights the current issues related to the status of toxicology and efforts in the betterment of this field.

This Module on Toxicology caters to broad spectrum of scientific and technological disciplines, from biology to medicine, engineering to molecular biology, chemistry modeling that will benefit the Medical Officers to consider the best therapeutic treatment to their patients.

(Dr. Deepa Tyagi)


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ACKNOWLEDGEMENT

Dr. Rajaganapathy R.
Director
State Institute of Health & Family Welfare
Department of Medical Health & Family Welfare
Government of Uttar Pradesh

Toxicology is the study of the adverse effects of drugs and chemicals on biological systems. It is understood as that branch of science which deals with poisons, and a poison can be defined as any substance that causes a harmful effect when administered, either by accident or design, to a living organism.

Toxicology does embrace the study of deleterious effects of substance exposure not only to the human body but also to the environment and all other organisms existing in the environment. Whereas, Forensic toxicology, is the use of toxicology and other disciplines such as analytical chemistry, pharmacology and clinical chemistry to cases and issues where those adverse effects have administrative or medicolegal consequences, and where the results are likely to be used in court.

This Module on Toxicology is a thoroughly modern science, based on published and widely accepted scientific methods and practices, for both analysis of drugs in biological materials, and interpretation of those results. Many of the methods it employs have been derived from innovations in clinical medicine and academic laboratories throughout the world.

The application of this knowledge of drug presence through forensic toxicology in tissues is to meet the varied needs of the law. The interpretation of effects of drugs and their duration of action for the purpose of a medico-legal process is best referred to as forensic pharmacology, although there is overlap between these two scientific disciplines.

I put my sincere thank to Dr. Ghyasuddin. Khan, Additional Director and State Medico Legal Expert and his accomplice Ms. Anurima Singh, Forensic Expert for authoring this Module as part of ongoing Continuing Medical Education endeavor of SIHFW. I would also like to thank Dr. Manish Singh, Assistant Professor, Training for his immense effort in drafting and editing this Module.

(Dr. Rajaganapathy. R)


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GENERAL TOXICOLOGY

INTRODUCTION

Toxicology is defined as a branch of medical science, which deals with poison in relation to their sources, properties, action, the symptoms which they produce, fatal dose, toxicity, diagnosis, treatment and autopsy features.

Toxicology

The word toxicology is derived from Greek and means "an arrow" which indicates its primitive use for smearing tips of arrows for slaying purpose.

The Poisons Act, 1919
- regulates the import, possession and sale of poisons.
- amended in 1958.

The Drugs and Cosmetics Act, 1940
- regulate the import, manufacture, distribution and sale of drugs

The Drug controls Act, 1950
- provides control of sale, supply and distribution of drugs

The Drugs and Magic Remedies (Objectionable Advertisement) Act, 1954
- bans the advertisement which offend decency or morality and, to prevent self medication and treatment, which causes harmful effects.

The Medicinal and Toilet Preparations (Excise Duties) Act, 1956
- provides for levy and collection of duties of excise on medicinal and toilet preparations containing alcohol and narcotic drugs

The Narcotic Drugs and Psychotropic Substances (NDPS) Act. 1985
- consolidates and amend the law relating to narcotic drugs
- makes stringent provisions for regulation of NDPS
- provide for forfeiture of property derived from narcotic drug trafficking
- implements the provisions of International Concention on NDPS

The Drugs and Cosmetics Rules 1945

- Schedule C – biological and other products
- Schedule E – list of poisonous substances under Ayurvedic, Siddha and Unani system
- Schedule F – vaccines and sera
- Schedule G – hormonal preparations, antihistamines, anti-cancer drugs
- Schedule H – barbiturates, amphetamines, reserpine, ergot and sulphonamides
- Schedule J – drugs claimed to prevent or cure aliments such as appendicitis, blindness, cancer, cataract, epilepsy, hydrocele etc. which must not be advertised or imported
- Schedule L – list of prescription drugs (which includesdrugs from Schedule H also).

Warning

- Schedule H and L drugs are required to be labeled with the words "SCHEDULE H DRUG" and "SCHEDULE L DRUG". Warning – to be sold by retail on the prescription of a Registered Medical Practitioner only.

Sec 85 IPC

- Criminal act done under involuntary intoxication

Sec 86 IPC

- criminal act done under voluntary intoxication

Sec 272 IPC

- adulteration of food or drink intended for sale.

Sec 273 IPC

- sale of noxious food or drink.

Sec 274 IPC

- adulteration of drugs.

Sec 275 IPC

- sale of adulterated drugs.

Sec 276 IPC

- sale of drug as a different drug or prescription.

Sec 277 IPC

- punishment for fouling water of public spring or reservoir.

Sec 278 IPC

- punishment for voluntarily making atmosphere noxious to health.

Sec 284 IPC

- negligent conduct with respect to poisonous substance.

Sec 299 IPC

- deals with culpable homicide.

Sec 300 IPC

- deals with murder.

Sec 304-A IPC

- causing death by negligence.

Sec 320 IPC

- deals with grievous hurt.

Sec 324 IPC

- voluntarily causing hurt by dangerous weapons or means.

Sec 326 IPC

- voluntarily causing grievous hurt by dangerous weapons or means.

Sec 328 IPC

- causing hurt by means of poison etc. with intent to commit an offense.

Sec 336 IPC

- act endangering life or personal safety of others

Sec 337 IPC

- causing hurt by act endangering life or personal safety of others.

Sec 338 IPC

- causing grievous hurt by act endangering life or personal safety of others.

General Toxicology: Nature of Poisoning

Homicide	should be colorless, odorless and tasteless
	easily available
	resemble natural disease
	symptoms should appear late
	should not produce vomiting
	fatal perios should be less
	no antidote available against it
	should be highly toxic
	should disappear from the body after death

Suicide	should produce an easy death
	easily available
	easily route of administration
	fatal period should be quick
Accidental	mistaken ingestion
	inhalation of vapors accidentally
	incorrect preparation of medicines
	excessive self medication
	drug addiction
	infected food

General Toxicology: Classification of Poisons

A. According to Nature of Poisons

- **Corrosive**
  - *Strong acids*
    - Mineral or inorganic acids, e.g. H2SO4, HCL
    - Organic acids, e.g. carbolic, acetic, oxalic acid
  - Strong alkalis, e.g. sodium hydroxide
  - Metals, e.g. mercuric chloride, ferric chloride.
- **Irritants**
  - *Inorganic*
    - Non-metal, e.g. phosphorus, iodine
    - Metal, e.g. arsenic, lead etc.
  - *Organic*
    - Animal, e.g. snake, scorpion
    - Plant or vegetable, e.g. castor, calatropis etc.
  - Mechanical, e.g. chopped hair, metal pieces etc.
- **Systemic**
  - *Cerebral poisons*
    - Somniferous
    - Inebriants
    - Stimulants
    - Deliriant
    - Depressant
    - Psychotropics
  - Spinal poisons, e.g. strychnine
  - Peripheral nerve poison, e.g. curare
  - Cardiac poison or cardio-toxic, e.g. aconite
  - Asphyxiants, e.g. carbon monoxide
  - Nephrotoxic, e.g. mercury
  - Hepatotoxic, e.g. phosphorus
- **Miscellaneous**
  - eg, Food Poisoning

B. According to procured effect

Hematologic System	• Anti-coagulants
Immunotoxic Agents	• Immunosuppressive drugs
Hepatotoxic Agents	• Hemolytic poisons • Ethanol • Paracetamol • Aflatoxin • Toxaphene • Iron • Chlorpromazine
Nephrotoxic Agents	• Coccaine • Quinine • Gold compounds • NSAIDs • Penicillamine • *PCT necrosis causing* • Phenol • Cantharides • Cephalosporins • Corrosive sublimates • Cresol • Carbon tetrachloride • *DCT necrosis causing* • Cisplatin • Amphotericin • Glycols • *Cystalluria causing* • Acyclovir • Ethylene glycol • Sulfonamides • *General Nephrotoxic Agents* • NSAIDs • Ergot alkaloids • Potassium chlorate • Heavy metals • Oxalic Acids
Pulmonary Toxic Agents	• Chlorine • Titanium • Silica • Phosgene • Cholinergic drugs • Paraquat

CNS toxic agents	• *Cerebral stimulants*
	  • Amphetamine
	  • Caffiene
	  • Cyclic antidepressants
	  • methamphetamine
	  • methylphenidate
	  • modafinil
	• *Cerebral Depressants*
	  • Alcohol
	  • Anesthetics
	  • Hypnotics
	  • Opioids
	  • Sedatives
	• *Cerebral Deliriants*
	  • Belladona
	  • Cannabis
	  • Coccaine
	  • Datura
	  • Hyoscyamus
	• *Spinal*
	  • Gelsemium
	  • Nux vomica
	• *Peripheral Nerve poison*
	  • Conium
	  • Curare
	  • Agricultural poisons

CVS toxic agents	• Aconite • Oleander • Quinine • Tobacco • Antiarrhythmic agents • aminoglycerides • General anesthetic
Dermatotoxic agents	• Arsenic • Camphor • Corrosives • Furocoumarins • Industrial solvents
Affecting Reproductive system	• alkylating agents • Chlorophenoxy herbicides • Plasticizers
Affecting Endocrine system	• Acrylonitrile • Atenolol • Aluminum • Calcitonin • Thiouracil

General Toxicology: Types of Poisoning

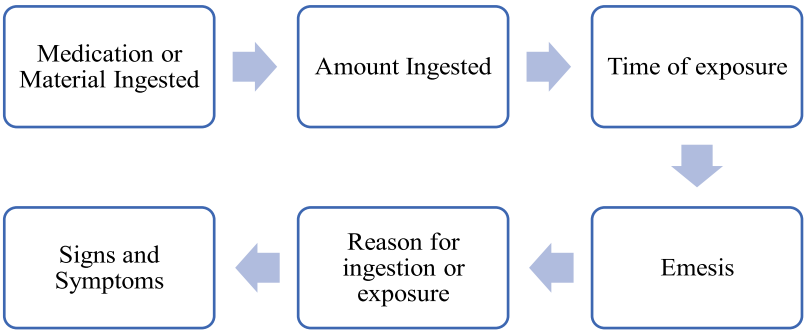
Fulminant	Acute	Subacute	Chronic
• produced by single massive dose	• caused by excessive single dose	• occurs gradually over some time • example of drug automatism	• caused by very minute dose taken over a long period of time

General Toxicology: Diagnosis of Poisoning

A. In the living

- Clinical History
- Clinical Signs
- Lab investigations

I. Clinical History



Suspicion of poisoning must be aroused following:

- Sudden onset of vomiting/diarrhea/pain in abdomen
- Onset of symptoms to many persons who had taken food together
- History of consumption of poison or suspicious material
    - Persistent vomiting and/or diarrhea with or without dehydration
    - Pain in abdomen
    - Altered sensorium/drowsiness/unconsciousness/coma
    - Breathlessness, constriction in chest, choking sensation
    - Convulsions
    - Palpitations, arrhythmias, chest pain
    - Hypothermia
    - Paralysis

II. Clinical Signs

General Examination

- Peculiar odour
- Taste
- Temperature-hypothermia/hyperthermia
- Ocular changes
- Oral manifestations

Poisons having typical odour	
Poisons	*Smell*
Cyanide	Bitter almond like
Phosphorus	Garlicky
Arsenic	Garlicky
Selenium	Garlicky
Thallium	Garlicky
Aluminum phosphide	Garlicky
Phenol	Phenolic
Organophosphorus	Kerosene like/garlicky
Conium	Mousy
Marijuana/Ganja	Burnt rope like
Hydrogen sulfide	Rotten egg like
Zinc phosphide	Fishy
Carbon monoxide	Coal gas
Nitrobenzene	Shoe polish
Chloroform	Acetone like
Camphor	Moth balls
Acetic Acid	Vinegar

Central Nervous System

- Grading of coma
- Patient may be drowsy, deliriant or in coma
- Convulsions
- Tremors
- Neuropathy
- Movement disorder
- Paralysis
- Headache
- Paresthesia
- Gait
- Delirium
- Encephalopathy
- Tingling
- Ataxia
- Psychosis

Poisons producing hypothermia
Opiates
Alcohol
Carbon monoxide
Barbiturate

Cardiovascular System

- Pulse-tachycardia/bradycardia

- Blood pressure: hypotension/hypertension
- Arrhythmias
- Circulatory collapse
- Vasoconstriction

Poisons producing hyperthermia
Strychnine
Cocaine
Datura
Curare

Taste of poisons	
Taste	*Poison*
Bitter	Datura, strychnine
Burning	Acids
Acrid	Calotropis
Sweet	Aconite
Sour	Oxalic acid
Metallic	Copper, Iodine
Sweet burning	Carbolic acid
Caustic	Alkali

Poisons producing ocular changes	
Ocular changes	*Poison*
Miosis	Opium Morphine Organophosphorus Barbiturates
Mydriasis	Alcohol Datura Carbon monoxide Cocaine
Nystagmus	Alcohol Thallium
Strabismus	Botulinus toxin Thallium
Hippus	Aconite
Diplopia	Opium Cannabis Alcohol
Photophobia	Mercury Quinine
Blurred vision	Alcohol Datura Aconite Nicotine

Poisons producing ocular changes	
Ocular changes	*Poison*
Optic neuritis/retrobulbar neuritis	Methanol Quinine Thallium
Conjunctivitis	Copper dust Acid fumes Marijuana
Deposition in lens	Mercury
Icterus/jaundice	Phosphorus Arsenic Bismuth Carbon tetrachloride Copper
Corneal edema	Chloroquine Irritant poisons Metal/acid fumes
Pitting of optic disc	Lead
Retinopathy	Chloroquine Phenothiazines
Fixation of pupils	Anesthetic agents Reserpine Rauwolfa alkaloids

Lacrimation	Ammonia Chili powder Irritant gases	Complete or partial blindness	Methyl alcohol Arsenic Chloroquine Ergot Lead Mercury Tobacco
Ophthalmoplegia	Thallium	Corneal stippling	Chloroquine
Altered colour perception	Carbon monoxide Cannabis LSD Digitalis	Retinal stippling	Lead Chloroquine Phenothiazines Tranquilizers
Corneal deposit	Amodiaquine	Papilledema	Lead Vitamin A toxicity
Xanthopsia	Aconite	Retinal hemorrhage	Carbon monoxide Lead
Ptosis	Thallium Gelsemium Botulinus toxin		

Contd..

Poisons producing oral changes

Glasgow coma scale

Eye opening	
a. Spontaneous	**4**
b. To speech	**3**
c. To pain	**2**
d. Nil	**1**
Best motor response	
a. Obeys	**6**
b. Localizes	**5**
c. Flexes (withdrawal)	**4**
d. Flexes abnormally (decorticate rigidity)	**3**
e. Extends (decerebrate rigidity)	**2**
f. Nil	**1**
Best verbal response	
a. Oriented	**5**
b. Confused conversation	**4**
c. Inappropriate words	**3**
d. Incomprehensible sounds	**2**
e. Nil	**1**

Oral change	Poison
Salivation (Sialorrhea)	Organophosphates Mineral acids Alcohol Aconite Croton Chili seeds Tobacco Copper Scorpion
Dryness of mouth	Datura Ephedrine Scopolamine Antihistaminics Antidepressants
Xerostomia	Narcotics Tricyclic antidepressants
Gingival hyperplasia	Phenytoin Sodium valporate
Stomatitis	Cyanide Calatropis Iodine
Parotitis	Iodine
Glossitis	Cyanide
Discoloration of teeth	Fluoride Tetracycline

Causes of coma
Alcohol Carbon monoxide
Opium
Organophosphates

Causes of paralysis
Arsenic Poison hemlock
Curare
Lead Manganese

Causes of tremors
Alcohol Mercury Manganese
Cocaine Arsenic

Causes of neuropathy/neuritis
Alcohol Lead
Organophosphate Methanol

Causes of headache
Alcohol Aniline
Cyanide
Tobacco Nitrite

Causes of paresthesia
Aconite Alcohol
Arsenic Cannabis Poison hemlock
Ergot Lead Thallium

Causes of ataxia
Alcohol Carbon monoxide
Lead
Lithium Narcotics

Causes of encephalopathy
Thallium
Arsenic Lead
Thallium

Causes of tachycardia
Carbon monoxide
Cannabis Amphetamine Atropine (Datura)
Poison hemlock Nitrites

Causes of psychosis
Carbon monoxide Cannabis
Amphetamine Atropine (Datura) Poison hemlock
Nitrites

Causes of delirium
Calotropis
Datura Cannabis Cocaine

Poisons causing various types of gait	
Gait	*Poison*
Staggering or rolling	Alcohol Barbiturate CNS depressants Phenothiazines
Stiff gait	Tranquilizers Reserpine

Causes of movement disorder	
Poison	*Disorder*
Strychnine	Rigidity Opsithonus Trismus
Carbon monoxide	Parkinsonsism
Organophosphates	Fasciculation

Causes of bradycardia
Organophosphates
Aconite Barium Digitalis
Neostigmine Pilocarpine

Causes of hypotension and shock
Aconite
Arsenic Nitrites Organophosphates
Snake bite Iron

Toxic causes of arrhythmia/cardiac irregularities
Carbamates
Aconite Oleander
Digitalis Zinc phosphide Lithium

Toxic causes of hypertension
Amphetamine Zinc phosphide
Ephedrine Chlorthiazides

Causes of dyspnea
Carbon monoxide
Strychnine Phosphine
Arsine

Toxic causes of pulmonary edema
Organophosphates
Chlorine Digitalis
Snake bite
Opium

Causes of respiratory disease
Alcohol Opium
Organophosphates Snake bite
Aconite
Barbiturates

Causes of circulatory collapse
Aconite Barbiturate Carbon monoxide
Cocaine Corrosive poison
Lead
Mercury Nicotine
Nitrites

Causes of vasoconstriction and/or gangrene
Ergot
Lead Tobacco
Cocaine Amphetamine

Respiratory System

- Dyspnoea
- Pulmonary edema
- Respiratory distress
- Cough
- Laryngospasm
- Bronchitis
- Emphysema

Toxic causes of cough
Acid fumes Metal fumes
Formalin
Chlorine Sulfur dioxide
Copper Ammonia

Toxic causes of emphysema
Tobacco smoke
Silica exposure

Toxic causes of laryngospasm
Irritant poisons Metal/acid fumes
Strychnine Thiopental Ammonia

Toxic causes of bronchitis
Chromium dust Nitrogen oxide Osmium
Phosgene
Tobacco smoke

Color of vomitus in differentpoisons	
Color of vomitus	*Poison*
Blue	Iodine
Green	Paris green Cannabis Copper sulfate
Brown	Acid Alkali Zinc phosphide
Blood (hematemesis)	Chronic alcoholism Manganese

Gastrointestinal System

- Vomiting
- Diarrhea
- Constipation
- Gastroenteritis
- Melena
- Abdominal pain
- Abdominal distension
- Ileus
- Thirst
- Dysphagia
- Odynophagia
- Pancreatitis

Poisons producing diarrhea
Arsenic
Boric acid
Cyanide Food poisoning
Iron

Poisons producing constipation
Ergot Calcium salts
Lead
Opium Arsenic

Poisons producing gastroenteritis
Arsenic
Thallium Croton

Poisons producing melena
Alcohols
Anticoagulants Corrosive
Iron

Poisons producing distension of abdomen
Anticholinergics
Caustics

Poisons producing abdominal pain
Caustics Cholinergic agents Cocaine
Iron
Salicylates

Poisons producing albuminuria
Arsenic
Mercury
Chromate Phenol Thallium

Poisons producing odynophagia
Camphor Corrosives
Hydrogen sulfide
Scorpion sting

Poisons producing hemoglobinuria
Acetic acid Arsine Copper
Nitrites
Snake bite

- Glycosuria
- Hematuria
- Porphyrinuria

Poisons producing glycosuria
Morphine Anesthetic agents

Poisons producing hematuria
Allopurinol

Poisons producing thirst
Arsenic Atropine
Chloral hydrate
Lead

Poisons producing ileus
Anticholinergics
Barium
Botulinus toxin Lead Thallium

Poisons producing dsyphagia
Camphor Corrosives

Cause of toxic pancreatitis
Copper Zinc
Organophosphate
Methanol

Genitourinary System

- Colour of urine
- Albuminuria
- Hemoglobinuria

- Oliguria
- Polyuria
- Dysuria

Arsenic
Mercury
Phenols

Poisons producing oliguria
Phenol
Arsenic
Chromate
Carbon tetrachloride

Poisons producing polyuria
Alcohol
Mercury
Nitrites
Bismuth
Digitalis

Poisons producing dysuria
Arsenic
Anticholinergics
Mushrooms

Poisons producing myopathy
Sea snake
Pentazocine
Meperdine
Heroin
Alcohol
Iodine

Poisons producing porphyrinuria
Lead
Mercury
Benzene
Carbon tetrachloride

Poisons producing myalgia
Copper
Arsenic
Lead
Sea snake

Poisons producing fasciculation
Lead
Organophosphate
Strychnine
Mercury

Poisons producing rhabdomyolysis
Bee stings
Barbiturates
Cocaine
Heroin
Paraquat
Snake bite

Poisons producing anemia
Chronic alcohol
Arsenic
Lead
Opium

Musculoskeletal System

- Myopathy
- Myalgia
- Rhabdomyolysis
- Fasciculation
- Smooth muscledepressant/stimulant

Smooth muscleDepressants/Stimulant	
Feature	*Poison*
Depressant	Barbiturates Nitrites Papaverine
Stimulant	Barium salts

Aniline
Benzene
Cadmium
Lead
Naphthalene
Zinc
Iron
Acetic acid

Poisons producing leukopenia
Aniline
Arsenic
Antimony
Lead
Chloromphenicol
Sulfonamides
Promazine

Poisons producing leucocytosis
Snake poisoning
Pilocarpine
Titanium tetrachloride
Thallium

Poisons producing stippling
Lead
Antimony
Bismuth
Barbiturate

Poisons producing hemolysis
Sea snake
Arsine
Castor

Blood Manifestations

- Anemia
- Blood dyscrasia
- Thrombocytopenia
- Leukocytosis
- Leukopenia
- Pancytopenia
- Polycythemia
- Stippling
- Bone marrow depression
- Hemolysis
- Methemoglobin formation
- Sulfhemoglobinemia

Poisons producing pancytopenia
Chloromphenicol
Sulfonamides
Erythromycin

Poisons producing polycythemia
Arsenic
Carbon monoxide
Aniline
Cobalt

Poisons producing bone marrow depressions
Beryllium
Cadmium
Fluoride
Phosphorus
Selenium

Copper sulfate Nitrofurantion
Lead

Poisons producing thrombocytopenia
Promazine
Sulfonamide Chloramphenicol

Poisons producing methemoglobin formation
Aniline
Nitrobenzene Nitrites
Methylene blue
Toluidine Copper

Poisons producing sulfhemoglobinemia
Poisons producing methemoglobin

Dermal Manifestation

Dermal features in poisoning	
Feature	*Poison*
Color	Pink – carbon monoxide Cherry red – cyanide Cyanosis – organophosphate, strychnine
Dry hot	Datura
Sweating/Moist	Organophosphate Arsenic Opium Pilocarpine
Flushing	Alcohol Arsenic Cyanide Datura
Blisters/ bullae	Barbiturate Carbon monoxide Viper snake bite Marking nut juice Methaqualone Meprobamate Tricyclic antidepressants Mustard

Dermal features in poisoning	
	Aniline derivatives
Pruritis	Ergot Chloroquine
Exfoliation	Arsenic Chloroquine
Hirsutism	Barbiturate
Corrosion	Acid/alkali Chromic acid Iodine Mercuric chloride Phenol
Edema	Arsenic Mercury Oxalate Phenol

	Calatropis Plumbago		
Petechiae, purpura or hemorrhagic lesion	PhosphorusArsenic Benzene Dicumarol Heparin	Pallor	Arsenic Barbiturate Cocaine Lead
Dermatitis	Arsenic	Ulcers	Acids/alkali Chromate Fluoride Iodine
Skin lesion	Acne – bromide, thallium Pigmentation – arsenic Erythema – iodide Papule – antimony dust Pustule – croton oil Rash – thallium, mercury	Urticaria	Bromide Phenobarbitone Sulfonamide Thiouracil Iodine
Contd..		Hair loss/ alopecia	Thallium Arsenic

Causes of vertigo
Alcohol
Cannabis Carbon monoxide Cyanide
Ergot Tobacco

Toxic causes of deafness
Methyl alcohol
Ergot
Quinine Tobacco
Streptomycin

Toxic causes of fever
Arsenic
Datura Barbiturates Cocaine
Metal fumes

Poison acting on enzyme system
Phosphorus Cyanide
Organophosphate
Mercury Lead

Ear Manifestations

- Rhinorrhea
- Deafness
- Vertigo

Poisons producing rhinorrhea
Iodine
Arsenic

Causes of tinnitus
Furosemide Indomethacin
Quinine
Aminoglycosides

- Tinnitus

Others

- Poisons causing fever
- Poisons acting on enzyme system
- Excretion of poison

Excretion of posion	
Excretion	*Poison*
Bile	Narcotic drugs Cocaine Paracetamol
CSF	Alcohol
Vitreous humor	Alcohol
Fatty tissue	Pesticides

B. In the dead

Autopsy Findings

Laboratory examintions

I. Autopsy Examination

External Examination

- Evidence of soiling of clothes
- Presence of bottle/container/label in pocket
- Presence of suicide note

Color of postmortem lividity	
Color	*Poison*
Cherry red	Cyanide
Pink	Carbon monoxide
Blackish	Opium
Bluish green	Hydrogen sulphide
Dark brown	Phosphorus
Chocolate brown	Potassium chlorate
Reddish brown	Nitrites

Poisons causing early appearance of rigor mortis
Strychnine
Hydrocyanic acid

- Vitriolage
- Trickling of poison/stigmata of poison over skin
- Presence of injuries
- Smell from body
- Rigor mortis: in some condition rigor may appear earlyor in some delayed
- Postmortem lividity: poisons may impart peculiar colour
- Some poisons resist decomposition or some may hasten

Internal Examination

Poison resisting decomposition
Arsenic
Datura
Alcohol
Formalin

- Cranial cavity: Presence of odour/cerebral edema/hemorrhages.
- Chest cavity: Note for presence of pleural effusion, pericardialeffusion,state of lungs and heart, hemorrhages,pulmonary edema

Causes of cerebral edema
Alcohol

Organophosphate
Aluminium phosphide

➢ Abdominal cavity: Stomach contains gastric contents of various colours. Mucosa may show erosions, ulcers, hemorrhagic gastritis or perforation. Gastric mucosa may be stained with the colour of poison.

Perforation of stomach
Sulfuric acid
Hydrochloric acid

Color of gastric mucosa	
Color	*Poison*
Blackish	Sulfuric acid Nitric acid
Yellow	Tobacco
Red velvety	Arsenic
Bluish green	Copper sulfate
Mucosa brown	Iodine
Bleached, sodden	Alkali

Hepatic necrosis
Arsenic
Carbon tetrachloride
Chloral hydrate Chloroform
Chromium salts

➢ Examine the state of intestine, liver, spleen, kidneys, pancreas, bladder, esophagus, lip, and oral cavity.

Cloudy degeneration of liver	
Bismuth	
Mercury	
Soft	Sulfuric acid
Bleached, sodden	Alkali
Leather like	Phenol
Scalded	Oxalic acid

Chronic liver degeneration and cirrhosis
Alcohol
Carbon tetrachloride Gold Manganese

II. Laboratory examinations

Qualitative Assays

- Thin layer chromatography is a simple and inexpensive techniquefor qualitative estimation of poison.

Quantitative Assays

- Gas chromatography
- High performance liquid chromatography
- Mass spectrometry
- Radio-immuno-assay
- Atomic absorption spectrophotometry
- Neutron activation analysis.

MANAGEMENT OF POISONING

DUTIES OF MEDICAL PRACTITIONER IN A CASE OF POISONING

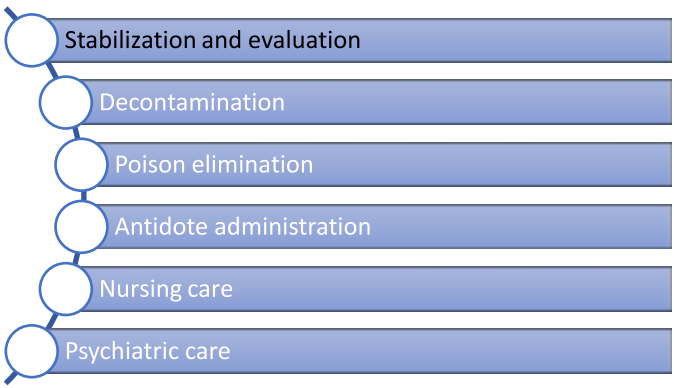
The duties of medical practitioner are both legal and professional.

- In all cases of poisoning, the doctor must record the preliminary particulars, viz., full name with address, age, sex, occupation, date and time, brought by whom, history, etc. When poisoning is suspected, the doctor's first duty is to guard his patient's interest. He should at once treat him after finding out the nature of the poison so that appropriate and timely treatment is instituted. If the nature of the poison is not known, treatment is instituted on general lines.
- In all cases of suspected or confirmed poisonings the attending doctor should record all the findings in a medico legal case report and inform the nearest police.
- If the homicidal poisoning is suspected by the doctor, the Police officer should be informed (S. 39 CrPC). If the doctor fails to report the matter to the police, he will be held culpable under section 176 I.P.C.
- It may be worth mentioning here that Section 43 of IPC stretches the ambit of the expression 'legally bound to do' to a considerable extent. Three categories have been mentioned, viz., (i) everything that is an offence, (ii) everything that is prohibited by law and (iii) everything that furnishes grounds for civil action.
- The attending physician should collect, preserve and seal the evidence related to the case of poisoning such as the gastric lavage fluid, vomitus, faeces and urine etc. for onward transmission to the Forensic Science laboratory for chemical analysis. If the doctor deliberately

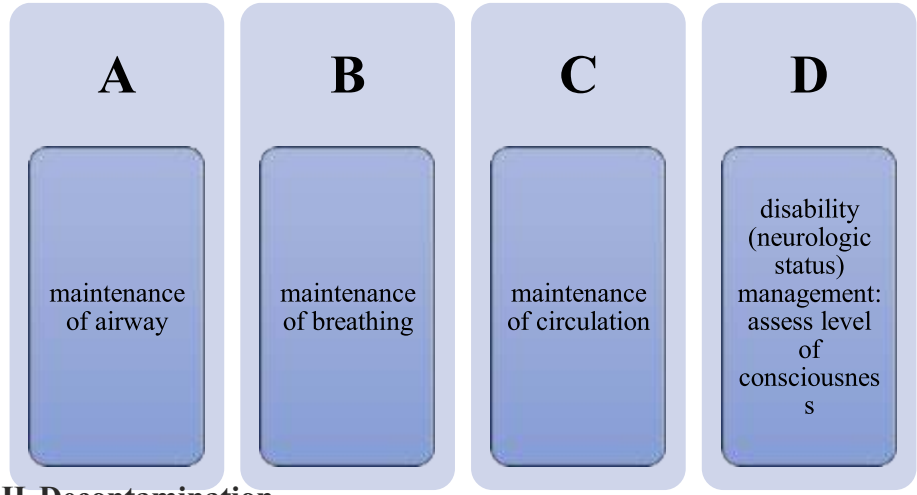
fails to do so he is liable to be punished under section 201 I.P.C.

- In all the cases of poisoning whether suicidal or homicidal, it is the duty of the attending physician to divulge to the police whatever information he has. Withholding the information or providing wrong information makes the doctor liable to be punished under section 202 and 193 I.P.C.
- In case of poisoned patient on the verge of dying it is the duty of the attending doctor to record dying declaration if the magistrate is not available at that time. Even when the magistrate is recording the statement of such a patient, the doctor should examine the patient in regards to his consciousness and whether there is clear faculty of thought and judgment or not (compos mentis).
- In cases that are brought dead to the hospital or the patient has died during the course of treatment, the doctor should not issue death certificate but instead send the body for autopsy examination.
- In cases of food poisoning, the doctor should collect the contaminated food and sent it to the forensic science lab for chemical analysis. It is the duty of the doctor to report such cases to public health officials.

Management of Poisoning



I. Stabilization and evaluation



II. Decontamination

In cases of inhaled poisons, the individual should be shifted to fresh air and artificial ventilation should be started.

In cases of injected poisons from bite or an injection, a ligature should be tied to prevent it from further reaching the circulation. The ligature is loosened for one minute after every ten minutes to prevent gangrene formation.

In contact poisons affecting the skin, mucous membrane, any wound or introduced in the uterus or vagina they should be irrigated with water and neutralized by application of suitable chemical.

The poisons are most commonly ingested orally. Depending on the time lapsed after ingestion; much of the poison lying unabsorbed in the stomach should be removed. In all ingested poisons the guidelines to treatment are:

1. The stomach should be emptied by gastric lavage or emesis but emetics

are avoided in corrosive intake.

2. Appropriate antidote should be given to neutralize the poisonous compound even despite gastric lavage and emesis as some poisons are secreted again in stomach after having been absorbed.

3. Aid elimination of poison by the intestines and kidneys of what has been absorbed.

4. Symptomatic treatment.

5. Egg whites are useful in most poisonings and tannin is antidotal to all alkaloids.

In ingested poisons, the methods of decontamination are:

(i) Gastric lavage

(ii) Emesis

(iii) Catharsis

(iv) Administration of activated charcoal

(v) Whole bowel irrigation.

(i) Stomach Wash (Gastric Lavage)

Gastric lavage is indicated in patients who present within three hours of ingestion of poison. The stomach wash can also be carried in the presence of gastric secretions, delayed gastric emptying or in case of ingestion of sustained release medications. Gastric lavage beyond 6-12 hours is recommended in ingestion of salicylates, tricylics, carbamazepine and barbiturates.

Gastric Lavage Tube: For gastric lavage, a soft rubber tube with a funnel at its one end known as Ewald or Boas tube is most commonly used. In adults an ordinary, soft, non-collapsible tube with 36-40 French size

having one cm diameter and 1½ mts length is used. In children with 22-28 French size (Ryle's tube) diameters should be used. The tube should have an attached glass funnel at one end and the other end should be rounded with lateral openings. There is a mark at a distance of 50cm from its rounded end. A suction bulb is placed at the mid of to tube to pump out stomach contents.

Procedure for Stomach Wash: Before performing stomach wash, the patient should be lying in a left lateral position or in a prone position with head hanging over the edge of the bed and the face is positioned downwards so the mouth is at a lower level than the larynx and chances of aspiration of fluid are eliminated.

The gastric lavage tube is gently passed in to the stomach through the mouth by lubricating with glycerin or Vaseline jelly up to a distance of 50cm in adults and 25cm in children. The position of the tube in stomach can be checked by stethoscope or putting the upper end in a cut of water, if the lower end in the trachea, air bubbles will come out. For carrying out stomach wash, initially 250- 300mL of warm (35oC) saline or plain water is passed through the funnel held high up. In children, instead of water 10-15mL/kg body weight of warm saline is used as in them there are chances of inducing hyponatremia and water intoxication. The stomach contents can be siphoned by the use of suction bulb.

The first sample of stomach wash should be preserved for chemical analysis. Then stomach is carried out with chemical agents specific to the poisons. In certain specific poisonings, instead of plain water or normal saline, other solutions can be used that are specific for particular poisons. These solutions are:

1. Potassium permanganate (1:5000 or 1:10,000) is used as gastric lavage fluid in various oxidizable poisons e.g., alkaloids, salicylates.

2. Sodium bicarbonate (5%).
3. Tannic acid (4%).
4. Sodium thiosulfate (25%) can be used in cyanides and
5. Calcium gluconate is used for oxalates.
6. 1:2 Castor oil and warm water solution is to be used for carbolic acid and phenolic group of poisons.
7. Desferrioxamine (2 gm in 1 liter of water) is used for iron poisoning.

In cases where potassium permanganate (a powerful oxidising agent) is used as gastric lavage fluid, the gastric lavage is continued till the colour of lavage fluid is colourless, odourless and no particulate matter is visible. At this time, a small quantity of fluid containing specific antidote or 1 gm/kg body wt. of the suspension of activated charcoal and/or an ionic cathartic is left in the stomach.

Complications of Gastric Lavage:

1. Laryngeal spasm.
2. Aspiration pneumonitis.
3. Perforation of stomach or oesophagus.
4. Sinus bradycardia and ST elevation on ECG.

Contraindication of Gastric Lavage: The contraindication for stomach wash are absolute and relative.

Absolute contraindication:

1. Corrosive poisoning except Carbolic acid as there is danger of perforation.
2. Convalescent poisons.
3. Comatose patient as there is risk of aspiration

4. Volatile poisons due to the risk of inhalation.
5. If the patient is hypothermic.

Relative contraindication:

1. If the patient is suffering from alimentary tract diseases like oesophageal varices.
2. Comatose patients.
3. Ingestion of alkali.
4. Advanced pregnancy.
5. Any haemorrhagic diathesis.
6. Any history of recent surgical operation.

(ii) Emesis

The emesis should be avoided as there is danger of aspiration of stomach contents in an unconscious patient. The easiest way to induce vomiting is by tickling the fauces. Also vomiting can be produced by the use of emetics of which ipecac is the most effective whereas mustard powder and warm saline can give rise to complications. Also Apomorphine and zinc sulphate are no longer used. These emetics are:

i. Warm saline water comprising of 2tsf salt in 200mL of water.

ii. Mustard powder 15 gms in 200mL of water

iii. Zinc sulphate 1-2gms in 200mL of water

iv. Apomorphine 3-6mg I.M. is the most potent and immediately acting emetic as it produces copious vomiting within 3-4 minutes. Apomorphine causes severe narcosis that is why it should not be used in comatose patients. If it is used, Naloxone hydrochloride, 5-10 mg i.m. should be administered that will counteract narcosis.

v. Ipecacuanha powder 1-2gm or 30mL (15mL in children) of ipecac syrup causes vomiting with satisfactory results in a poisoned patient. It is derived from the root of Cephalis ipecacuanha and C. acuminata. The active principles are cephaline, emetine and traces of psychotropine and it causes activation of peripheral sensory receptors in the GIT and also stimulates the chemoreceptor trigger zone and vomiting center in the medulla thus causing vomiting.

Contraindication of emesis: The contraindication of emesis are:

Absolute Contraindication:

1. Pregnancy.
2. Heart disease.
3. Haemorrhagic diathesis.
4. Cardiotoxic poison ingestion.
5. In infants and old patients.

Relative Contraindication:

1. During convulsions.
2. Ingestion of convalescent poison such as strychnine.
3. Ingestion of strong acids and alkalis due to the chances of perforation.
4. Ingestion of kerosene oil as there are chances of aspiration pneumonitis.
5. Comatose or unconscious patients.
6. Foreign body ingestion.
7. Impaired gag reflex.
8. Poison that causes emesis.
9. Ingestion of petroleum distillates or drugs causing altered mental

status. **Complications of Emesis:**

1. Features of cardio-toxicity such as bradycardia, atrial fibrillation and myocarditis.
2. Aspiration pneumonitis.
3. Oesophageal tears may be caused due to protracted vomiting.

(iii) Catharsis

Cathartic Salts The most effective cathartic is sorbitol in adose of 1–2 gm/kg body weight. Alone, cathartics do not prevent absorption of the ingestant and should not be used as a method of gut decontamination. Their primary use is to prevent constipation following charcoal administration.

(iv) Administration of activated charcoal

Adsorbents like activated animal charcoal that has the capacity to adsorb poisons(e.g. alkaloids) in the pores so that the poison can not come in contact with the wall of the stomach and is thus prevented from being absorbed.

(v) Whole bowel irrigation

It is performed by administer in a bowel cleansing solution containing electrolytes and polyethyl-eneglycol orally or by gastric tube at a rate of up to 0.5 L/hin children and 2.0 L/h in adults until rectal effluent is clear. The patient must be in sitting position. It may be appropriate for those who have ingested foreign bodies, packets of illicit drugs, slow-release or enteric-coated medications and agents that are poorly adsorbed by charcoal, e.g. heavy metals. It is contraindicated in patients with bowel obstruction.

III. Poison Elimination

- Hemodialysis
- Hemoperfusion
- Peritoneal dialysis
- Plasma perfusion

When more then 6-8 hours have elapsed after ingestion of the poison, the absorbed poison should be eliminated by excretory channels that are kidneys. In barbiturate and salicylates poisoning, this is the only treatment. Diuretics like chlorothizide, mannitol or furosemide are commonly used drugs. Fluid balance is to be maintained by IV fluid infusion. Peritoneal dialysis and exchange transfusion is done in small children in poisonings due to barbiturates, salicylates and iron. Hemodialysis is employed for removing barbiturates, salicylates, bromides, boric acid and thio-cyanates. The methods employed for the purpose are:

Catharsis

Cathartics reduce the transit time of the poisonous substance in the G.I.T but their efficacy in reducing the mortality or morbidity is not established. The cathartics are:

Saline cathartics: These cathartics alter the physico-chemical forces

within the intestinal lumen leading to osmotic retention of fluid which activates motility reflexes and enhances expulsion.

The dose of recommended cathartics is:

Magnesium citrate—4mL/kg.

Magnesium sulphate—30gm (250mg/kg in children).

Sodium sulphate—30.gm (250mg/kg in children) in copious amount of water.

The high dosage magnesium cathartics can result in hyper magnesemia.

Saccharide cathartics: Sorbitol (D-glucitol), 50 mL of 70% solution is the cathartic of choice in adults because of better efficacy than saline cathartics but in children there is risk of hyper natremia.

IV. Antidote administration

Recommended emergency antidotes
Activated charcoal
Amyl nitrate **Antivenin** **Calcium chloride**
Calcium gluconate gel **Desferrioxamine**
Digoxin immune Fab **Ethanol**
Folic acid **Fomepizole** **Flumazenil**
Glucagons **Leucovorin**
Methylene blue 1% **A-Actyl cysteine**
Naloxone **Physostigmine** **Polyethylene glycol electrolyte**
Pralidoxine **Sodium bicarbonate**
Sodium nitrite 3% **Sodium thiosulfate**
Succimer **Thiamine hydrochloride** **Vitamin K1**

Recommended antidotes for poisons	
Antidote	*Poison*
Acetylcysteine	Paracetamol
Amyl nitrite	Cyanide
Atropine	Organophosphate
Desferrioxamine	Iron
Ethanol	Methanol
Flumazenil	Benzodiazepines
Glucose	Insulin
Naloxone	Opiates
d-penicillamine	Copper
Physostigmine	Central anticholinergics

According to the mode of action antidotes are classified as:

1. Physical or Mechanical or Non-Specific Antidotes
2. Chemical Antidote
3. Physiological or Pharmacological Antidote
4. Universal Antidote
5. Chelating Agents

1. **Mechanical or Physical Antidotes**

The mechanical antidotes counteract the effect of poison mechanically preventing their absorption without inactivating the damaging action of the poisons. These are of the following types

Adsorbent like activated charcoal: Activated charcoal is a fine, black, odourless and tasteless type of amorphous carbon prepared by destructive distillation of materials such as burning wood, coconut shell, bone, sucrose, or rice starch that have much higher surface area than charcoal followed by treatment with an activating agent such as steam, carbon dioxide etc. The large surface area of activated charcoal confers a great adsorptive capacity to this material. Each gram of activated charcoal works out to a surface area of 1000 m2 . Activated charcoal strongly adsorbs aromatic substances such as acetaminophen, salicylates, barbiturates and tricyclic antidepressants thus reducing their absorption from the gastrointestinal tract. Most inorganic substances are poorly absorbed by activated charcoal. The dose is 1gm/kg body weight (50-100 gm in adults and 10-30 gm in children) after making a suspension in 4-8 times the water. 4-8 gms of activated charcoal acts mechanically by absorbing and also retains in its pores organic poisons and mineral poisons to a less degree. The side effects are vomiting, diarrhoea, constipation, pulmonary aspiration and Intestinal obstruction. The contraindication of the use of activated

charcoal are ileus, small bowel obstruction and when there is history of caustic or petroleum distillate ingestion.

Demulcents: Demulcents produce protective coating over the mucous membrane of stomach to protect it from the action of poison. These are milk, egg white, starch, milk of magnesia and aluminium hydroxide gel.

Bulky foods like Bananas, boiled rice or potatoes allow smaller amount of poison to be available to the stomach mucosa for absorption as they are admixed with the poison itself. The bulky foods are commonly used in ingestion of glass powder as these particles are embedded in them and prevent damage to the stomach mucosa. Diluents such as water, milk or other similar drinks that dilute the poison and delay their absorption.

2. **Chemical Antidotes**

The chemical antidotes disintegrate and inactivate poisons by undergoing chemical reactions along with the poisons and forming harmless or insoluble compounds. These are:

i. *Weak non-carbonate alkalis:* In corrosive acid poisonings, weak non-carbonate alkalis act as neutralizers. Strong alkalis are avoided as they can cause further damage the stomach. Non-carbonate alkalis are preferred as carbonate alkalis react with acids to produce carbon dioxide gas that inflates the stomach and may get ruptured.

ii. *Weak vegetable acids*: In corrosive alkali poisoning, weak vegetable acids like citric acid, and acetic acid (vinegar, lemon juice) may be used. For arsenic poisoning, freshly prepared solution of ferric oxide can be used as it forms non-absorbable ferric arsenate.

iii. *Albumen:* It is used for mercury poisoning as it precipitates Mercuric chloride.

iv. *Copper sulphate* is antidote for phosphorus

v. *Potassium permanganate* is an oxidizing agent use in poisoning with oxidizable substances like cyanides, phosphorus, atropine and other alkaloids. A dilute 1:5,000 or 1:10,000 solution of potassium permanganate is used in any poisonings such as aluminium phosphide, opium derivatives, insecticides, nicotine, cyanides, hydrocyanic acid atropine and strychnine etc.

vi. *Tincture iodine or Lugol's iodine* in a solution of 15 drops in half a glass of water precipitates lead, mercury, silver, alkaloids and strychnine.

vii. *Tannic acid (4%) or strong tea or 1 tsf of tannic acid* dissolved in water is used to precipitate metals like lead, mercury, nickel, zinc, copper, aluminium, cobalt and silver; strychnine, nicotine, cocaine etc.

3. **Physiological Antidote**

The physiological antidote acts on the tissues and various systems of the body and produce signs and symptoms opposite to the signs and symptoms produced by the poison. They are basically of use when some of the poison has already been absorbed in to the circulation. They antagonize the effects produced by the poisonous substance. These are like Atropine for organo phosphorus compounds, physostigmine, and neostigmine for Dhatura and barbiturate for strychnine.

4. Universal Antidote

Universal antidote comprises of:

Composition of Universal Antidote		
Components	*Quantity*	*Mechanism*
Charcoal	2 parts	Adsorbs poisons
Magnesium oxide	1 part	Neutralizes acids
Tannic acid	1 part	Precipitates

15 gms of this powdered mixture should be added to half a glass of warm water before consumption. The use of universal antidote is obsolete now-a-days but this can definitely used as a first-aid measure at homes.

5. **Chelating Agents**

Chelating agents inactivate a metallic ion with the formation of an inner ring structure in the molecule, when the metallic ion becomes the member of the ring. The chelating agents will form no-toxic stable soluble compounds with calcium and other heavy metals like arsenic, lead, mercury, copper, zinc, nickel, cobalt, manganese etc.

British Anti-lewisite (B.A.L)- Dimercaptopropanol

B.A.L. was originally used as an antidote for Lewisite, a vesicant containing arsenic that was used as war gas. This compound is used in heavy metal poisoning especially arsenic, mercury, lead, antimony, gold and thallium and also to some extent against copper, bismuth etc.

Mechanism of action: Heavy metal ions have a great affinity for sulphydryl (SH) radicals in the cells and tissue enzymes and they combine with them by displacing hydrogen, thereby depriving the body of certain tissue enzymes whose activity depend upon the SH groups. The thiol (SH) group of B.A.L. will combine with the heavy metals in the system and will dislodge them from their combination with the sulphydryl radicals in the tissue enzymes and thus sparing the tissue from its toxic effects. Dimercaprol forms a rather stable compound with the heavy metal and gets excreted out of the body mainly in urine without causing any damage to the liver, kidneys etc.

Dose: of B.A.L is 3-4mg/kg body weight that is to be administered deep intramuscularly within first 4 hours of poisoning. The ampoule of B.A.L (100mg/mL) contains 2ml of 10% compound in Arachis or peanut oil with 20% benzyl benzoate solution. The injection must be given 4 hourly for first 2 days and then thrice daily for 10 days.

Contraindications: Administration of Dimercaprol is absolutely contraindicated in cadmium poisoning (forms a nephrotoxic compound with cadmium) and any pre-existing liver disease. Also it is relatively contraindicated in any kidney disease.

Side effects: The side effects of administration of Dimercaprol appear when it is administered in a dose more than 3.5mg/kg body weight.

1. Anorexia, nausea and vomiting.
2. Excessive salivation or lachrymation.
3. Generalized aches and pains.
4. Hyperthermia.
5. Feeling of constriction of chest.
6. Burning of eyes and throat.
7. Fall of blood pressure.

E.D.T.A (Ethylene Diamine Tetra-acetic Acid)

E.D.T.A forms readily soluble, practically nonionized and nontoxic compound with heavy metals. It is a useful antidote for those heavy metals that have an affinity for calcium.

Mechanism of action: When lead, zinc, manganese, cadmium, iron and copper form compounds with edetate, calcium cannot displace them. Calcium disodium EDTA chelates extracellular lead in soft tissues as well as in bones. In practice, edentate is given in the form of calcium

disodium versenate to prevent rapid removal of calcium from the body. In the presence of lead, this chelating agent readily exchanges calcium for lead and thus detoxification and excretion of lead is done. The chelating agent renders lead water soluble, nontoxic, non-ionized and non-metabolized to get excreted intact in the urine. Edetate does not get metabolized in the body like Dimercaprol and is poorly absorbed by the gut. Calcium disodium versenate is the best known chelating agent against inorganic lead intoxication but not so effective in tetraethyl lead poisoning. It is used principally against lead intoxication but can also be used in copper, zinc and nickel poisoning. It is less effective in manganese, iron, cadmium and radio-active elements poisoning. It is superior to both BAL in the treatment of arsenic and mercury.

Dose: 5 mL ampoule of 20% calcium edetate is dissolved in normal saline or 5% saline dextrose (250-500mL) solution. It is administered by slow I.V. drip. The concentration should not exceed 3% and the drip should not take less than two hours to complete. The usual dosage is 50-70mgm/ kg per day and in adults 1gm can be given I.V. Twice daily for five days. This should be repeated again after a gap of three days. E.D.T.A. should not be given orally as lead will get chelated in the G.I.T. and more of it will be absorbed.

Side effects: 1. Thrombophlebitis from administration of strong solution.

2. Lower-nephron nephrosis.
3. Hypersensitivity to the agent.
4. Fever, headache, generalized malaise and fatigability.
5. Nausea, vertigo and vomiting.
6. Hypotension.

Contraindication: The only contraindication is raised intracranial

pressure when fluids are to be restricted.

Penicillamine (Cuprimine)

Penicillamine is the product of hydrolysis of penicillin. It acts as a chelating agent because of possessing stable SH groups. It is advantageous to use this compound against poisoning due to lead, copper, mercury and zinc preparations, as it can be used orally and continuously for a long time without producing any major toxicity. It is also useful in treating Wilson's disease (hepatolenticular degeneration) resulting from disorders of copper metabolism and cystinuria.

Dose: It is given 0.5 gm orally half an hour before meals four times a day for a period of 8-10 days. Pyridoxine 25-50mgm/day can be given to counteract symptoms of pyridoxine deficiency.

Side effects: The side effects of Penicillamine therapy are rare and are more pronounced when the patient is suffering from copper storage disease, cystinuria or scleroderma for a long period.

1. Hypersensitivity reactions in the form of skin rashes and nephrotoxicity
2. Optic neuritis resulting from pyridoxine deficiency.
3. Leucopenia, thrombocytopenia and agranulocytosis.

Desferrioxamine Mesylate or Deferoxamine

Desferrioxamine is a water soluble compound that has a great affinity for ferric ion. This acts as chelating agent against iron intoxication especially in case of acute poisoning through its role in accelerating removal of iron from the body in case of haemochromatosis cannot be challenged. It removes iron from ferritin, hemosiderin, a little from transferring but not from haemoglobin and cytochromes.

Dose: It can be given orally, intramuscularly as well as intravenously:

- Orally: 8-10gms dissolved in 80-100mL of distilled water
- Intramuscularly: 1gm initially to be followed by 0.5gms twice or thrice daily
- Intravenously: 1-2gm in 5% of 500 mL of dextrose saline solution; not more than 15mg/kg body wt per hour or 80mgm/kg in 24 hours should be administered.

V. Symptomatic Treatment

The patient should be treated for the symptoms accompanied with good nursing care under constant supervision of the physician. Respiratory tract infection is commonly encountered more common in older patients who have been unconscious for hours and in whom gastric lavage was done. In such cases, routine antibiotic prophylaxis should be given. Dehydration, anuria, convulsions, circulatory collapse hepatic and renal failure should be taken care of. Fluid balance to be maintained by I.V. fluid infusion and I.V. mannitol should be administered to combat renal failure. BP and pulse rate should be maintained continuously. The airways should be protected and artificial respiration with oxygen inhalation to be given.

CORROSIVE POISONS

Corrosives are the poison that fixes, destroys and causes erosion of the surface coming in its contact. Some organic acids like oxalic and carbolic acid act as corrosives in concentrated form as dicarbonates of sodium and potassium. Certain metallic salts e.g. sodium chloride, potassium cyanide, ferric chloride, chromates and bichromatesof alkalis also act as corrosives.

Showing corrosive substances	
Compound	*Examples*
Inorganic acids	Sulfuric acid, hydrochloric acid etc
Organic acids	Acetic acid, carbolic acid etc
Alkali	Sodium hydroxide, potassiumhydroxide etc
Metal salts	Ferric chloride, zinc chloride,chromate etc
Non-metal compound	Iodine, potassium permanganate,hydrogen peroxide etc

Mode of Action

The mode of action of inorganic or mineral acids like sulphuric acid, hydrochloric acid and the nitric acid are:

1. Causes only local action but no remote effects on the system.
2. In concentrated form, they cause corrosion and destruction of the tissue
3. Extracts the water from the tissues.
4. Fixes, destroys and erodes the tissues.
5. Converts Haemoglobin in to Haematin.
6. Causes coagulation necrosis by precipitation of proteins.
7. The mucosa of the Oesophagus is relatively resistant to the acids.

8. Mucous membrane of stomach especially pyloric region is very susceptible and in this region necrosis commonly occurs.
9. Complications develops from 3 weeks to 3months of ingestion of the substance.
10. Corrosives act as irritant and when well diluted act as stimulant.

Pathophysiology

Following phases have been identified after ingestion of corrosive agent:

1. *Inflammatory stage:* It occurs during the first 4 days. edema and erythema develops first followed by thrombosis of vessels and tissue necrosis.
2. *Granulation stage:* It starts at about day 4 and ends approximately7 days after ingestion. Fibroplasia results in the formation of granulation tissue with the laying down of collagen over the denuded areas of mucosal sloughing.
3. *Perforation:* Most often occur between day 7 and 21.During this period the tissues are week and the risk of perforation is high.
4. *Cicatrization stage:* Starts at 3 weeks and may persist for years. Dense fibrous tissue formation occurs at variable rates. Overproduction of scar tissue results in stricture formation and obstruction.

Ingestion:

- Pain in mouth, throat and abdomen
- Dribbling of saliva
- Eructation
- Retching
- Vomiting

- Hematemesis
- Dysphagia
- Dysarthria
- Dyspnea and dysphonia due to regurgitation or fumes.

Management

- Dilution of acid by milk or water
- Demulcent – starch, egg white, milk
- Supportive measures
- Contraindication.
    - Gastric lavage
    - Emesis
    - Neutralization with alkali as it may cause exothermic reaction and increases the risk of perforation
    - Carbonated alkali – may react with acid and produces carbon dioxide gas that may distend the stomach and increases risk of perforation.

General Principles of Treatment

1. The stomach wash should not be given as there are chances of perforation of stomach. However Levin tube can be used for stomach wash within half an hour of ingestion of poison. The tube should be passed softly and with due care avoiding the risk of perforation of stomach and oesophagus.
2. Emesis is to be avoided as there is risk of perforation of already thinned out stomach and oesophagus

3. Acids should be immediately diluted and neutralized by drinking plenty of water containing a tablespoonful of calcium oxide, magnesium oxide or aluminium hydroxide gel. If these are not available then Demulcents like vegetable oil, soap solution, milk, limewater or white of an egg should be

followed by Barley water and olive oil.

4. Bismuth subcarbonate 30gm should be given.
5. Morphine 15 mg i.m or i.v or Meperidine HCL50-150mg orally or i.v should be given for pain.
6. 10mL of 10% Calcium gluconate should be given intravenously.
7. Blood transfusion can be given if needed.
8. Tracheostomy can be performed if oedema of glottis is present.
9. Oxygen inhalation and artificial respiration can be given, if necessary.
10. Corticosteroids should be administered to prevent oesophageal strictures.
11. To prevent formation of strictures later on,½ inch mercury filled bougie should be passed daily.
12. In case of skin burns, wash with large quantities of water or apply paste of sodium bicarbonate.
13. Eye burns are treated symptomatically after irrigating with water for 10-15minutes.
14. Use of strong alkalis such as carbonates and bicarbonates of sodium and potassium should be avoided as they produce CO2 and can cause distension and perforation.

Complication of Inorganic Acid Poisoning

A) *Acute:*

1. Massive gastric hemorrhage
2. Bronchopneumonia
3. Perforation of stomach
4. Perforation peritonitis
5. Transient laryngeal edema
6. Infection/sepsis
7. Renal failure
8. Shock.

B) *Delayed (Chronic):*

1. Gastric outlet obstruction/pyloric stenosis
2. Malnutrition.

Causes of Death

I. Death occurring within few hours can be dueto shock or spasm or oedema of glottis

II. Within 24hrs, death results from perforation of stomach leading to peritonitis and shock.

III. Within first week the death may result from septic absorption

IV. After months or years due to exhaustion and malnutrition due to oesophageal or pyloric stricture or incurable dyspepsia due to destruction of coats of mucous membrane of the stomach.

Early Causes of Death	Delayed causes of Death
• Shock • Spasm or edema of larynx • Perforation peritonitis • Toxemia	• Aspiration pneumonia • Secondary infection • Renal failure • Malnutrition

Preservation of Viscera

In case of inorganic acid poisoning deaths, viscera should be preserved in rectified spirit.

Medicolegal Importance

- Accidental poisoning – common (mistaken for medicine, industrial, etc).
- May be thrown over face or body with malicious intention(vitriolage).
- Suicide – rare.

Sulfuric Acid H_2SO_4

Synonyms: Oil of Vitriol

Properties:

- Heavy, oily, colourless, odourless and non-fuming liquid

- Hygroscopic
- Carbonizes organic substances.
- Fatal dose: 5 to 10 ml
- Fatal period: 12 to 18 hours.

Diagnosis:

- Haemo concentration in red blood cells is noticed.
- Diffuse mottling of lung fields on X-ray when there is inhalation of acid vapour.
- Chemical tests
  - i. When sulphuric acid is mixed with barium nitrate or chloride, white precipitate of barium sulphate is produced.
  - ii. Strong sulphuric acid chars organic matter.

Autopsy Findings:

- Corrosion of chin, angle of mouth, lips, oral mucosa, tongue, throat.
- Corrosion over hands may be noted
- Teeth chalky white
- The corroded area of skin or mucous membrane appear brownish or blackish (due to chemical charring of the affected tissue)
- Perforation of stomach may be seen.

Nitric Acid

Synonyms: Aqua Fortis, Red Spirit of Nitre

Properties:

- Clear, colourless, fuming liquid
- Pungent odour
- With organic substances, it causes yellowish discolouration due to **xanthoproteic** reaction.
- Fatal dose: 10 to 15 ml
- Fatal period: 12 to 24 hours.

Autopsy Findings:

- Corrosion of skin, angle of mouth, lips, mucosa with yellowish discolouration
- Stomach wall is soft and friable, ulcerated.
- Perforation is less common.

Hydrochloric Acid

Synonyms: Muriatic Acid, Spirit of Salts

Properties:

- Colorless, odourless, volatile, fuming liquid
- May acquire yellowish tinge when exposed to air.

- Fatal dose: 15 to 20 ml
- Fatal period: 18 to 30 hours.
- Chemical tests:
  - On addition of silver nitrate to a solution of hydrochloric acid, a white precipitate of silver chloride is formed.
- Chronic Poisoning
  - Constant exposure to fumes produces chronic poisoning. The symptoms and signs of chronic poisoning are: (i) Coryza (ii) Conjunctivitis (iii) Corneal ulcer (iv) Pharyngitis (v) Bronchitis (vi) Inflammation of gums (vii) Loosening of teeth.

Autopsy Findings:

- The skin or mucous membrane shows corrosion. However, corrosion is less severe.
- The skin may be brownish discolored and parchment like
- Coagulation of the surface of the tongue and the mucosa of pharynx and esophagus is seen
- Stomach is soft, edematous, congested, and de-squamatedor may be ulcerated
- Perforations is less common
- Stomach contents – mixed altered blood with mucus
- Inflammation and edema of respiratory passage.

Vitriolage:

- Vitriolage means throwing of acid on the face or body of a person with a malicious intention to cause bodily harm or disfigurement or to cause blindness.
- The term is derived from the practice of throwing sulfuric acid (oil of vitriol). However, it is broadly used to denote injury caused by throwing any corrosive substance such as acid or alkali.

Acetic Acid

Synonyms: Ethanoic Acid, Ethylic Acid

Properties:

- Colourless, volatile liquid with pungent odour
- Pure acetic acid is an ice-like solid below 16°C, hence it often described as glacial acetic acid. Above this temperature, it is colourless liquid.
- The dilute form of acid is called as vinegar (vinegar is about 4-5% solution).
- Fatal dose: 50 to 100 ml (concentrated)
- Fatal period: about 48 hours.

Mechanism of Action:

- In concentrated form it acts as corrosive
- In dilute form it acts as an irritant
- Systemic absorption causes hemolysis, hemoglobinuria, renal failure, disseminated intra-vascular coagulation, metabolic acidosis and liver

dysfunction.

Autopsy Findings:

- Massive geographic liver necrosis
- Degeneration and swelling of renal tubular epithelium.

Carbolic Acid

Synonyms: Phenol, Hydroxy-Benzene

Properties:

- Colourless, prismatic, needle-like crystals that turns pink and liquefies when exposed to air.
- Has sweetish burning taste and phenol like smell
- Concentrated phenol is a dark brown liquid and contains impurities like cresol.
- Lysol is 50% solution of cresol in saponified vegetable oil. However, phenol is 8 times more toxic than Lysol
- Dettol is chlorinated phenol with turpineol
- Household phenol (sold as phenyle) contains five percentphenol in water.
- Derivatives of phenol
    1. Cresol
    2. Thymol
    3. Creosate (coal tar)

4. Menthol
5. Tannic acid
6. Napthol
7. Resorcinol.

- Uses:
  1. Antiseptic and disinfectant
  2. Manufacture of plastic
- Absorption, Metabolism and Excretion:
  - Phenol is absorbed from skin, gastric mucosa, per rectum, per vagina and respiratory tract
  - Phenol is converted into hydroquinone and pyrocatechol and excreted in urine. Traces are excreted by lungs, salivary glands, and skin.
- Fatal dose:
  - 2 gm crystals
  - 25 to 50 ml of household phenol
- Fatal period: 3 to 4 hours.

Mechanism of Action:

- Phenol has local as well as systemic action
- Locally it acts as corrosive agent and when absorbed, it causes CNS depression, metabolic acidosis and renal failure.
- Carbolic acid has great penetrating power and it coagulates protein.
- Phenols have a powerful antipyretic effect similar to that of

salicylates.

- Phenols and derivatives of phenols cause methemo-globinemia.

Clinical Features:

Local: When applied to skin or mucosa, it causes burning pain, numbness, tingling and anesthesia. It causes corrosion and produce white eschar (scar), which falls off in few days leaving brown stained area.

Systemic:

- GIT: Burning pain followed by tingling numbness and anesthesia. Nausea and vomiting.
- RS: Respiration is slow and labored.
- CNS: Headache, giddiness, unconsciousness, convulsions, coma.
- Oliguria and hepatic failure.
- Urine: May be colorless but on exposure to air turns green due to oxidation of phenol metabolites (hydroquinone and pyrocatechol). It is known as carboluria.
- The hydroquinone and pyrocatechol may cause pigmentationin the cornea and various cartilages, a condition known as oochronosis.

Management:

- Skin: Wash with undiluted polyethylene glycol.
- Oxygen/ventilatory support
- Intravenous fluids and vaso pressors to support blood pressure
- Ingestion: Cautious stomach wash with sodium or magnesium sulfate solution

- Lidocaine for ventricular arrhythmias
- Benzodiazepines for seizures
- Treat methemoglobinemia – if methemoglobinemia is> 30%, ingest Methylene blue (1-2 mg/kg). Exchange transfusion may be needed if methemoglobinemia

Autopsy Findings:

- Phenol smell
- Corrosion of skin, at angle of mouth, chin. Corrosions are initially white but turns brown in colour
- Splashing may be noted
- Tongue – white and swollen
- Mucosa of stomach is tough, white or gray, corrugated and arranged in longitudinal folds and looks leathery.
- Mucous membrane of mouth, throat, lips are sodden whitened or ash gray
- Urine on exposure to air turns green.

Medico legal Importance:

- Accidental poisoning.
- Suicidal ingestion.
- Homicide – not possible.

Oxalic Acid

Synonyms: Salts of Sorrel, Acid of Sugar

Properties*:*

- Colourless, transparent, odourless, prismatic crystals resembling the crystals of magnesium sulphate and zinc sulphate
- It has sour and slightly bitter acidic taste
- It is present in rhubarb leaves, beets and many other vegetables.
- Potassium oxalate, sodium oxalate and ammoniumoxalate are toxic salts of oxalic acid.

Uses:

- Bleaching and cleansing agent
- Ink remover
- Rust remover
- Metal polishing
- Cleaning brass and copper articles
- Fatal dose: 15 to 20 gm
- Fatal period: 1 to 2 hours

Mechanism of Action:

- *Local:* It acts as corrosive when used in concentrated form and act as irritants when used in dilute form
- *Systemic:* After absorption, oxalic acid combines with calcium ion and causes **hypocalcemia**. It also causes tubular necrosis and renal failure.

Clinical Features:

- *Local:* Corrosion of mucosa with underlying congestion. The corroded area is referred as "**scalded**" in appearance.
- *Systemic:*
  - Vomiting and diarrhea
  - Hypocalcemia (**tetany**)
  - Muscle irritability, tenderness, cramps
  - Convulsions
  - Accoucher's hand due to carpopedal spasm
  - Chavostek's sign positive. When tapping is done over facial nerve area, there is spasm of facial muscles.
  - Metabolic acidosis
  - Renal failure
  - Uremia.

Management:

- Local exposure: Wash the affected skin with copious water
- Gastric lavage with calcium gluconate or calcium lactate
- Calcium gluconate intravenously
- Symptomatic.

Autopsy Findings:

- Scalded mucosa of GIT
- Mucous membrane of mouth, tongue, pharynx, esophagus may be

bleached and has scalded appearance

- Kidneys show edema, congestion with oxalate crystals in renal tubules with necrosis of proximal convoluted tubule.

Medicolegal Importance:

- Accidental poisoning – common
- Suicidal ingestion – rare
- Homicide – not possible.

Corrosive Alkalis

Properties:

- Common corrosive alkalis are given in Table 34.3
- Ammonia is a colourless gas with pungent odour. It condenses to a liquid at –33.4°C. The chemical formula is NH_3.
- Ammonium hydroxide is a liquid containing about30 percent ammonia
- Other corrosive alkalis occur as white powder or colourless solution.

Mechanism of Action:

- In concentrated form, alkali acts as corrosive and in dilute form they act as irritant
- Strong alkali produces liquefaction necrosis and causes saponification of fats and dissolves proteins thus causing deep penetration in the

tissue resulting in extensive tissue destruction.

- Production of ulcers are more common
- Esophagus is more commonly affected than stomach resulting in stricture formation or perforation.
- Type of material ingested may result in varying degree and location of injury.
- Fatal Dose
  - Sodium carbonate – 30 gm
  - Potassium carbonate – 15 gm
  - Sodium hydroxide – 5 gm
  - Potassium hydroxide – 5 gm
  - Ammonia – 30 ml
- Fatal period: 24 hours.

Clinical Features:

- *Local:* Application causes chemical burns of the skin with

skin showing grayish, soapy, necrotic areas without charring.

Inhalation:

- Irritation of eyes and watering
- Cough, breathlessness
- Respiratory tract – edematous and inflamed
- Laryngeal edema or spasm may occur causing death.

Ingestion:

- Caustic taste and burning pain
- Abdominal pain
- Vomiting and vomitus is alkaline in reaction
- Diarrhea and tenesmus
- The lips, mucous membrane of oral cavity, and the tongue appears soft, swollen, bleached and bogy.
- The mucosa of GIT is swollen, soft, grayish or bleached and sloughs easily
- Esophagus is affected commonly than stomach and results in dysphagia, drooling and hematemesis.
- Alkali induced injury of esophagus is classified by Hawkins et al. It is determined at esophagoscopy.

Management:

- Local: Wash the affected area with copious water.
- Ingestion
    - Milk or water may be given to dilute the alkali
    - Contraindications:
- 1. Gastric lavage
- 2. Emesis
- 3. Neutralization with acid as it may cause exothermic reaction and increases the risk of perforation.
    - Assess the injury of esophagus by esophagoscopy

- Symptomatic.

Autopsy Findings:

- Ammonia like odour may be perceived
- Mucosa of mouth, tongue, esophagus and stomach is bleached and sodden with areas of necrosis
- Esophagus may show esophagitis or perforation
- Pulmonary edema
- Inhalation – laryngeal edema
- Skin application – chemical burns

Medicolegal Importance:

- Accidental poisoning – common (mistaken for medicine, industrial etc.)
- May be thrown over face or body with malicious intention(vitriolage)
- Suicide – rare.

INORGANIC NON-METALLIC IRRITANT POISONS

INORGANIC IRRITANTS: NON-METALLIC POISONS

Examples are:

- Phosphorus
- Iodine
- Chlorine
- Bromine
- Fluorine

PHOSPHORUS

Phosphorus is a non-metallic, hepatotoxic and protoplastic irritant type of poison. It exists in two forms; red phosphorus and white phosphorus. Of the two, red is not poisonous but white phosphorusis a deadly one.

White/yellow phosphorus: White phosphorus occurs in the form of white, waxy, translucent and pliably soft sticks. It is insoluble in water, somewhat soluble in alcohol and ether and readily soluble in carbon disulphide. On exposure to air, it slowly oxidizes or **'phosphorescences'** — Phosphorescenceis the condition of white phosphorus,when it emits white fumes of phosphorus trioxide, which is luminous in the dark giving strong garlic odour. At 34°C, it ignites in the air emitting greenish-white flame, hence it is to be preserved under water or kerosene oil. It should not be handled even by wet finger as even the body heat can cause ignition.

Red phosphorus: Red phosphorus is prepared by heating white

phosphorus at 240o-250oC in an atmosphere with nitrogen or carbon dioxide gases. It occurs ordinarily as violet red solid mass that isodourless, tasteless, is insoluble in carbon disulphide, and does not luminescence in the dark(Table 38.5).

Showing difference between yellowand red phosphorus

Features	*Yellow phosphorus*	*Red phosphorus*
Colour and appearance	White, waxy, crystalline translucent soft cylinders. On exposure to air becomes yellow	Violet-red, amorphous mass
Taste and odour	Garlicky odour and taste	Odourless and tasteless
Luminosity in dark	Luminous	Not luminous
Exposure to air	Phosphorescence	Not Phosphorescence
Toxicity	Highly toxic	Non-toxic

Uses of Yellow Phosphorus

1. It is used in the preparation of vermin pastes that contain arsenic, fluor, oil, sugar, some colouring agents mixed with 1-4% conc. Of yellow phosphorus
2. In manufacture of gunpowder, fireworks, and incendiary ammunitions and for creating smokescreens during warfare.
3. In various chemical and fertilizer industries.

Medicolegal Aspects

1. Phosphorus is not commonly used for homicidal purposes due to its

characteristic garlicky odour, taste, luminosity in the dark makes forits easy detection. But it acts as a god homicidal poison as phosphorus containing rat poison mixed with strong tea is not easily detectable. Besides, there is delay in onset of clinical features and post mortem findings simulate that caused by hepatotoxic drugs and diseases.

2. Vermin pastes, rat killers containing phosphorus are commonly used for suicidal purposes.
3. Accidental poisoning can occur in children as they may ingest rat-poison. The poisoning can also occur from fragments of projectiles containing yellow phosphorus, inhaling hydrogen phosphide gas produced in ship holds from cargo and inhaling the gas evolved from ferrosilicon used in steel industry.
4. Arson using phosphorus is done in villages when moist cow-dung and yellow phosphorus are mixed together and thrown over thatched cottage-roof that when dries in sun catches fire from phosphorescent phosphorus

Fatal dose: Adults—60-120 mg

Children—10-25 mg

Fatal period: Upto 24 hours (4-10 hours)

Mechanism of Action

(i) Locally it acts as an irritant (ii) On absorption, it remains in the blood in elemental form for a day or two, then gets oxidized into hypo phosphorus and phosphorus acid when it acts as hepato toxic, protoplasmic poison disturbing the normal cell metabolism by affecting cellular oxidation (iii) It produces widespread fatty infiltration with degeneration of different organs especially the liver and cells of cerebral cortex (iv) It also causes tissue destruction by

interfering with carbohydrate, fat and protein metabolism especially by deposition of fat in the liver at the cost of glycogen (v) Chronic absorption of phosphorus leads to bone formation in the epiphyseal cartilage and in the bone marrow and Haversian canal thus impairing blood circulation in bones. This leads to necrosis and sequestration of bones with or without spontaneous fractures.

Acute Phosphorus Poisoning

Signs and Symptoms

The signs and symptoms of acute phosphorus poisoning usually appear within a few minutes and sometimes may be delayed for up to 6 hours.

First stage: (i) Garlic like taste in mouth and smell in breath that is luminous in the dark (ii) Sense of warmth with burning pain in mouth and throatradiating down to oesophagus, stomach and finally all over the abdomen (iii) Intense uncontrollable and non-quenchable thirst with frequent gastriceructations and patient drinks lots of water trying to quench his thirst (iv) Copious vomiting occurs that is frequent, profuse and persisting; has garlickyodour, luminous in the dark, bile stained and even blood tinged in later stages (v) Painful throat as a result of repeated retching and vomiting (vi)Diarrhoea though uncommon is in the form of dark, offensive, and phosphorescent motions usually preceded by colicky pain (vii) Abdomen is tender and distended with epigastric discomfort (viii)Hypoglycemia may result (ix) Cardiac and respiratory depression, cold clammy skin, hypothermia occurs (x) Delirium is followed by convulsion, collapse, coma and death occurring within 12-24hours of ingestion of poison (xi) Patient usually does not die in Ist stage but he enters IInd stage by 36-48 hours when signs and symptoms reduce in intensity and vitals improve

Second stage: This is a stage of apparent improvement of signs and symptoms that may last for 2-4days. There may be little pain in the abdomen with occasional vomiting and purging, malaise and headache.

Third stage: (i) Vomiting and diarrhoea reappear and is more intense and distressing and contains mucous and blood (ii) Abdomen distended with severe abdominal pain (iii) Jaundice sets in and deepens quickly (iv) Liver is enlarged, soft and tender (v) Skin becomes cold, respiration labouredand pulse is feeble (vi) Haemorrhages in the form of epistaxis, haematemesis, haematuria, malena and menorrhagia are commonly seen (vii) Petechialhaemorrhages over the mucous membranes (viii)Blood urea is usually raised (ix) Urine is scanty, high coloured, contains blood, ammonia, casts, albumen, free fat globules, excess of lactic acid, bile and some amino acids like leucine, tyrosine and cysteine (x) Headache, restlessness, in somniamay be seen (xi) Tinnitus and vertigo are usually present (xii) Impaired vision, cramps, muscle twitchings and even paralysis, delirium and frequent priapism may also develop (xiii) Features of increasing hepatic and renal insufficiency followed by hypoglycemia, quick weak and irregular pulse, fall of BP, pulmonary oedema, dyspnoea, cyanosis, oliguria, even anuria, subnormal temperature progressing to death (xiv) Yellow phosphorus produces usually second or third degree burns that are slowly healing and are surrounded by blisters on the skin.

Treatment

1. Stomach wash using 0.5% potassium permanganate solution that oxidizes phosphorus into harmless phosphoric acid and phosphates itself changing into manganesedioxide.
2. Stomach wash using 0,1% copper sulphate solution or 250mg of copper sulphate in a glass of water may be given by mouth every5

minutes until free emesis starts. Copper sulphate also acts as an antidote as it is reduced by phosphorus and is precipitated as copper phosphide forming a coating over the phosphorus particles thus rendering them inert.

3. Hydrogen peroxide 2% solution can also be used as gastric lavage fluid.
4. Activated charcoal also helps in adsorbing the poison.
5. Oils, fats, milk etc. should not be given to the patient as they help in dissolving phosphorus and promotes its absorption.
6. Liquid paraffin retards absorption of phosphorus and hastens its elimination but castor oil should not be used.
7. Bowels should be thoroughly washed using potassium permanganates and purgatives especially magnesium and sodium sulphate.
8. Glucose and alkaline drinks by mouth in plenty to protect liver.
9. High carbohydrate and low protein and fat diet should be given.
10. To combat shock and dehydration plenty of glucose saline (500 ml of 5%) fluids and 10ml of 10% calcium gluconate should be administered intravenously.
11. Symptomatic treatment such as non-narcoticanalgesics for pain, multivitamins especially injections of vitamin K should be given.
12. Locally, skin can be irrigated with 1% copper sulphate solution in water for at least 10minutes.

Autopsy Findings

The post mortem findings will depend on the period of survival:

Death within first 24 hours: (i) Slight icteric tinge of the skin (ii) Mucous membranes of pharynx, oesophagus, stomach and intestines shows

signs of inflammation, redness, ulceration and corrosion(iii) Stomach is contracted and mucosa is inflamed, softened, yellowish green with garlicky odour that luminesce in dark

Death after 24-48 hours: (i) Skin will look jaundiced (ii) Petechial haemorrhages under the skin and over serous and sub mucous surfaces of the lungs, brain, lepto meninges, uterus and kidney(iii) Garlicky odour from the body (iv) Mucosa of stomach and intestine is yellow or yellowish green with evidence of inflammation, softening, corrosion and haemorrhagic. This is also luminous in the dark (v) Liver is usually enlarged though it may be normal or contracted also. It is lemon yellow with doughy, soft and greasy consistency. A moderate pressure will allow the pressing finger to sink deep down in to the liver substance, which will be easily friable. Small haemorrhagic spots are seen on the glisson's capsule and in liver substance. Evidence of fatty degeneration, cellular necrosis, fibrosis, cellular infiltration is there. Evidence of accumulation of fat in Kupffer's cell is the earliest manifestation of necrobiosis the liver undergoes in phosphorus poisoning (vi) Heart is soft, flabby and dilated with evidence of fatty degeneration and subendo cardial haemorrhages (vii)Kidneys may be filled with cellular debris, fatty casts, albumen etc. and are enlarged, soft, greasy, yellow with petechial haemorrhages and fatty degeneration.Although phosphorus is readily oxidized in air, it may de detected in un oxidised form in dead body several days after death, even when the body is in an advanced state of decomposition. This may be explained by the fact that reducing gases that are formed during decomposition protects phosphorus from oxidation. Phosphorus occursin combination with food articles, in tissue and body fluids mainly as phosphates. Hence, detection of phosphate has little medico legal importance; but when detected in elementary form in the body, it is taken to be sufficient to have produced phosphorus

poisoning as it does not occur in free form in nature.

Chronic Phosphorus Poisoning

Chronic phosphorus poisoning usually occurs due to inhalation of fumes of white phosphorus amongst those employed in manufacture of fireworks and some ammunitions where phosphorusis being used, inhalation of phosphorated hydrogenin preparation of acetylene gas from carbide and also from escape of the gas from ferrosilicon results in poisoning. It is also seen in workers in match box and sticks factory.

Signs and symptoms: The symptoms and signs usually develop after working for weeks, months or even years in these factories: (i) Nausea, vomiting, garlicky smelling eructation's and purging (ii) Generalized wasting, weakness, lassitude, emaciation and joint pain (iii) Abdominal discomfort and pain (iv) Anaemia, jaundice, shallow complexion (v) Trache it is and bronchitis (vi) ***Phossy jaw***—It is osteomyelitis and periostitis of jaw due to chronic phosphorus poisoning associated with necrosis of the alveolar part of jaw bone along with the sloughing of gums and loosening or falling of teeth. At first, there is history of toothache, swelling of jaw, loosening of teeth, sloughing of gums, necrosis and sequestration of mandible with multiple foul-smelling pus discharging sinuses. Phosphorus mainly attacks the lower jaw through carious or decayed teeth or through inter spaced between the missing teeth adjoining the raw surface.

Treatment

1. Prophylactic measures such as clean lines sand ventilation of factories; oral hygiene of workers regularly that is washing mouths with soda bicarb solution and extraction or filling up of carious tooth should be followed.
2. If necrosis of mandible occurs surgical intervention is undertaken.

IODINE

Iodine occurs in the form of bluish black, soft, scaly crystals that have a metallic sheen and unpleasant taste. It is freely soluble in water, the solubility increasing in presence of iodide. Idiosyncrasyis more common than bromide. It is very commonly used household antiseptic and potassium iodide is largely used as medicine.

Mechanism of Action

It acts as corrosive and coagulates cellular protein causing necrosis. The iodine vapours are irritantto respiratory passages. Iodine is converted to iodide in the body. The normal iodide content of the blood is 2-5 mg/100 ml. The prognosis is good if the patient survives for 48 hours after ingestion of the poison.

Fatal dose→2-4 gm (in solid form) and Two drachms of tincture iodine.

***Signs and symptoms*:** (i) Burning pain in mouth radiates to throat, oesophagus, stomach and abdomen(ii) Increased thirst, nausea, vomiting, salivation and purging (iii) The lips and angles of mouth are stained brown; the vomitus and faeces are stained dark brown or blue mixed with blood smelling of iodine (iv) Urine is scanty or suppressed and reddish brown containing albumen; painful micturition (v) Headache, muscle cramps, giddiness(vi) Cold clammy skin and fall of BP (vii)Respiratory depression, quick weak pulse (viii)Delirium and collapse (ix) Consciousness is retained till the end (x) Iodides may cause enlargement of salivary glands and lymph nodes (xi)Application of iodine to skin causes weeping eruptions, with raised temperature in sensitive individuals (xii) Injection of compound of iodine may cause sudden fatal collapse due to idiosyncrasy(xiii) Inhalation of iodine vapours results in glottic oedema and death from asphyxia.

Chronic Iodine Poisoning (Iodism)

It results from chronic use of potassium iodide in large doses as medicine. The clinical features are in the form of frontal headache, running nose, sneezing, watering from eyes, conjunctivitis, bronchitis, parotitis, excessive salivation, nausea, vomiting and diarrhoea associated with oedema of face and eyelids that clears up once the medication is discontinued.

Treatment

(i) Gastric lavage using 5% sodium thiosulphate solution or water containing soluble starch and albumen (ii) Emetics should be given (iii) Demulcentsas barley water, egg albumen, milk, alkaline drinks and castor oils should be given (iv) Shock and dehydration to be treated with 5% dextrosesaline drip i.v with mephentin and other analepticsto maintain BP (v) Glucocorticoids and antihistaminics in repeated doses help in allaying oedema. Tracheo stomy to be performed to relieve glottico edema (vi) 5% Sodium thiosulphate 100-150mLorally helps to reduce free iodine to non-toxiciodides (vii) fluid and electrolyte balance shouldbe maintained (viii) in chronic poisoning discontinue the drug and give large doses of Sodium bicarbonate or sodium chloride with fluids to has ten recovery.

Autopsy Findings

(i) The face and eyes are swollen and there is evidence of glottic oedema (ii) Mucosa of gastrointestinal tract is stained yellow or brown, is congested, inflamed and excoriated at places. Also the stomach contents are turned blue due tointer-reaction of iodine with starchy food (iii) Lungs are congested and oedematous (iv) Heart and liver shows fatty change (v) Kidneys may show glomerular and tubular necrosis (vi) Brain is usually congested and oedematous

Medicolegal Aspects

1. Accidental poisoning is common in children from accidental drinking of tincture iodine, betadaineetc. or when an alcoholic solution when excessively used for external application.

2. Suicidal and homicidal poisoning is rare.

CHLORINE

Chlorine is a greenish yellow gas having an unpleasant irritating odour even when diluted. It is largely used in industry as bleaching agent and disinfectant

Fatal dose→Exposure to air containing 1/1000chlorine for 5 minutes is fatal by causing acutepulmonary oedema.

Fatal period→Inhalation of pure chlorine gas results in death within 12-24 hours.

Signs and Symptoms

(i) Inhalation causes intense irritation of respiratory passages, throat and eyes causing dry harsh painful violent cough with yellowish expectoration, intense watering eyes and coryza (ii) Headache, nausea and vomiting, abdominal pain occur (iii)Tachypnoea, extreme dyspnoea, palpitation and pyrexia results (iv) In extreme cases, glotticoedema, asphyxia, intense cyanosis, tachycardia occurs (v) Unconsciousness soon supervene and death results from acute respiratory or cardiacfailure (vi) Chronic exposure to chlorine vapours shows features of anemia, cachexia, dyspepsia, chronic bronchitis and emphysema.

Treatment

1. Patient to be removed to fresh air immediately

2. Oxygen inhalation, artificial respiration and suction of frothy fluid from air passages
3. 5% dextrose saline drip with sodium bicarbonate to combat dehydration and shock
4. Treatment of pulmonary oedema, acidosis etc. should be ensued
5. As a prophylactic measure the respirator masks soaked in sodium bicarbonate solution and sodium hyposulphite and goggles for eyes should be used

Autopsy Findings

(i) The respiratory tract mucosa shows intense congestion (ii) Lungs are congested, oedematousexuding frothy, tenacious blood-stained fluid and showing areas of patchy haemorrhages and collapse (iii) Stomach and intestines are congested (iv) Heart is also enlarged (v) Odour of chlorine from the ventricles of brain that is congested (vi) All organs are congested.

Medicolegal Aspects

Poisoning is usually accidental by chlorine and its compounds especially bleaching powder in industry. Use of chlorine gas in Ist World war caused numerous causalities

BROMINE

Bromine is a dark reddish brown liquid that volatalises at ordinary temperatures giving out irritating fumes of unpleasant odour. Compounds of bromine displace chlorides from the plasma and cells; thereby will cause depression of central nervous system hence are not commonly used in medicine as sedatives. They are also used asanticonvulsant agents. Children may get more readily affected from bromide poisoning than adults. The poison gets rapidly eliminated in

the urine, saliva, sweat and milk.

Fatal dose→Uncertain; 20-30gms of sodium or potassium bromide can cause alarming symptoms causes alarming symptoms.

Fatal period→One ounce of undiluted bromine causes death in about 7 days.

Signs and Symptoms

(i) Intense burning sensation in mouth, throat, oesophagus and stomach (ii) Intense thirst and excessive salivation (iii) Nausea and vomiting, gaseous eructations, dysphagia and diarrhoea (iv)Inhalation of Bromine fumes causes acute bronchialcatarrh, irritating cough, running nose and watering from eyes and intense chest constriction(v) Oedema of glottis, pulmonary oedema develops with death from suffocation

Chronic Poisoning (Bromism)

Long continued use of bromides in large doses as medicines result in bromism, especially when the blood level reaches >50 mg%. persons with low sodium level are more prone to bromism apart from those who are sensitive to these preparations

Signs and Symptoms

(i) Bromide acne vulgaris' with popular or pustular skin eruptions on the face, shoulders and upper part of chest (ii) Headache, coryza, watering eyes(iii) Swelling of face and eyelids (iv) Indigestion with loss of appetite, constipation, furred tongue, foul breath (v) Tremors, muscular weakness, staggering gait, loss of memory, slurred speech(vi) Features of bromide psychosis such as confusion of ideas, delirium, delusions or hallucinations, loss of sexual ability with mental depression and drowsiness (vii) Stupor and coma may result

Treatment

(i) Patient to be removed to fresh air (ii) Stomach wash with plain water containing starch or albumen(iii) Emesis to be induced (iv) Artificial respiration and oxygen inhalation (v) Tracheostomyto relieve oedema of glottis (vi) Sodium chloride 1-2gm flavoured with fruit juice is given every 6 hours orally until bromide level falls below 50mg% (vii)5% Dextrose saline drip to be given i.v. that helps in excretion of bromides (viii) Diuretics like frusemide or chlorthiazide help in excretion (ix)Analeptics to treat shock and collapse.

Autopsy Findings

(i) Mucous membrane and skin bears dark brown parchment like stains (ii) Gastrointestinal tract is congested with inflammation (iii) There are features of pneumonia and pulmonary oedema (iv) Allorgans are congested.

Medicolegal Aspects

1. Accidental poisoning occur in those sensitive to bromides preparations. It was used in Ist World War when bromide vapours were used as asphyxiating and lacry mating agents.
2. Used commonly for homicidal purposes.

INORGANIC METALLIC IRRITANT POISONS

INORGANIC METALLIC IRRITANT POISONS

Even at present times, many metals and their salts causes morbidity and mortality. This chapter deals with poisoning of arsenic, mercury, lead, copper, zinc, iron, thallium andantimony compounds.

ARSENIC *(Sankhyal, Somalkar)*

Arsenic is a heavy metallic inorganic irritant poison. Metallicarsenic is not poisonous as it is insoluble in water and cannot be absorbed from the gastrointestinal tract. However arsenious oxide or arsenic trioxide (*sankhyal or somalkar*) is poisonous. Two organic arsenic non toxic variants, mostly present in foodregularly consumed by humans are *arsenobetaine* and *arsenocholine*. They are found in shell fish, cod, and haddock.

Showing inorganic compounds ofarsenic

Compound	*Common Name*	*Properties*
Arsenious oxide (Arsenic trioxide)	Sankhya Somakhar White arsenic	White crystalline powder
Arsenic disulphide	Manseel Red arsenic	Red powder
Arsenic trisulphide	Hartal Yellow arsenic Orpiment	Yellow powder
Sodium arsenates		White or grayish powder
Potassium arsenates	-	-
Arsenic acid	Arsenic pentoxide Arsenic anhydride	White crystalline powder
Arsenic trichloride	-	Colorless fuming liquid

Arsenic triodide	Arsenious iodide Areseniuretted hydrogen Arsenic hydride	Orange color crystals Colorless and inflammable gas, Garlicky odor
Sodium arsenite	-	White powder
Potassium arsenite	-	White powder
Copper arsenite	Scheele's green	Greenish crystalline powder
Copper acetoarsenite	Paris green	Greenish crystalline powder

Sources

Soil, well water, shellfish and arsenic compounds.

Absorption

Absorption is possible through all routes.

Action

Arsenic compounds act by inactivating the sulhydril enzymes, which in turn interfere with the cellular metabolism, in the liver, lungs, intestinal wall, and spleen. Arsenic can replace phosphorus in the bones where it may remain for years. It also gets deposited in the hairs.1 Epidemiologic studies of arsenic in *drinking water* suggest that arsenic can cause skin, lung, liver, kidney and bladder cancer 1 in 1000 cases.

Fatal dose 100 to 200 mg of arsenious oxide.

Fatal period 2 to 3 days.

Toxicity rating 5 for all arsenic salts, except arsenic trioxide, which has a toxicity rating of 6.

Showing difference between arsenicpoisoning and cholera		
Features	*Arsenic poisoning*	*Cholera*
Pain in throat	Before vomiting	After vomiting
Conjunctiva	Inflamed	Not inflamed
Vomitus	Contains mucus,bile and streaks ofblood	It is watery orwhey like
Purging	Follows vomiting	Usually precedesvomiting
Stools	Stools Rice watery inearly stages, laterbecomes bloody,discharged with straining and tenesmus	Rice water liquid, involuntary jet
Laboratory examination	1. Radio-opaque shadow on X-ray of abdomen in arsenic trioxide poisoning 2. Coproporphyrin in urine 3. Arsenic detected in chemical analysis	Vibrio cholera detected on microscopic examination

Signs and Symptoms

Arsenic poisoning clinically manifests in three forms:

- Acute fulminating type
- Subacute type (gastroenteritis type)
- Chronic type.

Acute Fulminating Type

Here symptoms appear within half an hour especially when heavy dose of arsenic is taken (3-5 grams). Acute fulminating type occurs due to

inhibition of sulphydryl enzyme system which is necessary for cellular metabolism and also due to its potentcapillary poisoning action. It causes marked dilatation of capillaries and myocardial failure resulting in fall of blood pressure, shock and death instantaneously.

Subacute Type (Gastroenteritis Type)

This type of poisoning occurs when small doses of arsenic are given at repeated intervals. It resembles case of cholera or food poisoning. The symptoms are first dyspepsia, cough and tinglingin the throat, followed by vomiting, purging with pain abdomen and tenesmus. Stool is at first rice water type, but later becomes bloody. However, the difference between arsenic poison and cholera may be enumerated as follows:

- In arsenical poisoning, vomiting precedes purging (stools are rice water initially and later turn blood stained), there is pain in throat, voice remains unaffected, conjunctiva gets inflamed and vomitus contains mucous, bile and streaks of blood. Arsenic can be detected on chemical examination.
- In cholera, purging precedes vomiting (stools are like rice water throughout and passed in continuous involuntary jet),there is no pain in the throat, voice becomes rough and whistling, conjunctiva is normal and vomitus is watery. Cholera vibrio can be detected on microscopic examination.

Chronic Type

Displaying differential diagnosisofraindrop pigmentation of arsenic
Rain drop pigmentation may be mistaken for 1. Addison's disease 2. Secondary syphilis

Occurs in persons engaged in smeltering or refining ore or longterm exposure to arsenic compounds. Chronic poisoning with arsenic presents with a sequence of five different set of manifestations.

- *Gastrointestinal* The victim presents with gradual weight loss, malnutrition, fatigue, loss of appetite, cirrhosis of liver, nausea, vomiting, etc.
- *Catarrhal changes* The victim presents with running nose, headache, conjunctivitis, bronchial catarrh, etc.
- *Raindrop pigmentation* It is known to produce *milk and roses complexion* initially, followed by *patchy brown pigmentation* of the skin (especially *face*), which resembles the *raindrops*.
- It might also show hyper keratosis of the skin of the palm and soles, which is prone to change into basal cell carcinomaat a later stage. The scalp may also show alopecia (baldness).
- *Meese's lines* The victim's nail manifests with whitish lines1 to 2 mm breadth across the nail of the finger and to esrepresenting the deposition of the poison as a result of high sulphhydril content of the keratin.

Arsenical neuritis The victim presents with polyneuritis, opticneuritis, anesthesias, paresthesias, atrophy of extensors resulting in wrist and foot drop, etc.

Showing hematological abnormalities in arsenic poisoning
Hematological abnormalities: Leukopenia Thrombocytopenia Mild eosinophilia Karyorrhexis – manifested by bizarre uclear forms
Megaloblastic anemia
Basophilic stippling

Diagnosis

Urinary level of > 100 mg/ 24 hours suggestive of arsenic toxicity. Blood level and hair levels are not reliable.

Treatment

- Haemodialys is is the line of choice in massive arsenic poisoning. Chelation therapy with BAL (British Ante Lewisiteor dimercaprol) is advised here to control the deleterious effects of arsenic redistribution.
- Perform gastric lavage with warm water or freshly preparedhydrated ferric oxide solution.
- Give butter and other greasy substances which acts as demulcents and prevents further absorption of the poison
- Specific antidotes are BAL– chelation therapy in the dose of 400-800 mg on the first day followed by 200-400 mg on the next two days and then tapering the dose slowly).DMSA (Di Mercaptosuccinic acid) or DMPS (Di Mercapto propane sulphonate) penicillamine, calcium disodium versnate, etc may also be useful.
- Symptomatic therapy.
- Injection: Vitamin B1 helps in peripheral neuritis.

Postmortem Findings

External Body will be dehydrated, skin is pigmented or rarely jaundiced, hands and feet cyanosed, with Mee's lines on the nails. Rigor mortis is observed to be unusually longer.

- ***Internal***

  - Stomach – Velvety red or brownish, patchy areas with small ulceration seen on the stomach mucosa. Gastric contents emit garlicky odour.

- *Heart* – Shows *sub endocardial haemorrhage*.
- Other viscera – may show fatty degeneration (liver, kidney and heart). Brain may show acute encephalitis with haemorrhagic spots.

Chemical Test

Reinsch's test: Put 1-2 strips of bright copper foils into suspected solution previously acidulated with HCl and then boil for 5-10 minutes. The copper foil becomes coated with steel grey or black deposits of arsenic if present. This foil when washedin water, alcohol and ether and then heated will show white deposits of arsenious oxide (octahedral crystal on microscopy).

Other advanced tests for detecting arsenic are Marsh's test and Gutzeit's test.

Medicolegal Importance

- In India, today most common source of accidental poisoning with arsenic is by consumption of well water. There is sufficient evidence that a sizeable proportion of several Asian countries are exposed to arsenic tainted water, especially the tube well water.
- According to Modi, arsenic was considered as an ideal homicidal poison in the past in India and west as it was cheap, easy to obtain, could be easily mixed and given with food without changing the smell and taste and the symptoms of poisoning would be similar to cholera or gastroenteritis. However, due to legal restriction of sale of arsenic and availability of sophisticated methods of detecting minute quantities of arsenic in blood and tissues from the dead bodies of victims of arsenic poisoning (e.g. *Marsh's test*, which can detect even the traces of arsenic -1/1000 mg dilution),criminal use of arsenic has become extinct now.

- Accidental poisoning instances do occur occasionally with those who consume arsenic for purposes like its aphrodisiac effects (quick remedy for impotence), eating arsenic arsenophagy, for better respiratory stamina among them ountaineers, etc.
- Arsenious acid, copper arsenite (Scheel's green) copperaceto arsenite (Paris green), liquor arsenicals(Fowler's solution which is 1 per cent arsenious oxide, were used as medicine for treating fever in the past). Potassium arsenite and sodium arsenite is used to make flypapers, rodenticides, fungicides and sheep dips. Arsenic sulphides used for making yellow pigment for art.

MERCURY *(Quick Silver, Liquid Metal, Para, Padarasa)*

Mercury is a liquid metal and is a metallic inorganic irritant poison. It is available in inorganic, organic and metallic forms. Metallic mercury is a heavy, silvery liquid and is non poisonous. But it volatilises at room temperature and inhalation of vapoursis toxic. Potential source of elemental mercury is at home, which includes mercury switches, mercury containing devices such asthermometers, thermostats, and barometers. Family members may also bring it home from laboratories, dental offices, and industrial sources, etc.

Absorption

Absorption is possible through all routes.

Action

Pure metallic form is nontoxic. However, the mercurial compounds can act by inactivating sulphhy dryl enzymes, which in turn interfere with cellular metabolism.

Toxic Compounds

Though the metallic form of mercury is non-toxic, its vapors as well as finely divided small particles of mercury can be toxic. There are several inorganic mercurial compounds, which are toxic.

Showing inorganic compounds ofmercury		
Compound	*Common Name*	*Properties*
Mercuric oxide	Sipichand	Brick-red crystalline powder
Mercuric Chloride	Perchloride of mercury Corrosive sublimate	Heavy colorless prismatic crystals
Merucric iodide	Red iodide	Scarlet red powder
Mercuric cyanide	-	White prismatic crystals
Mercuric nitrate	-	Deliquescent crystalline
Mercuric sulphide	Cinnabar Hirgul Ras sindoor Cheena sindoor Pigment vermillion	Red crystalline powder
Mercuric sulphate	-	White crystalline powder
Mercurous chloride	Calmol Raskapoor Subchloride of mercury	Fibrous, heavy, dirty white mass
Mercurous nitrate	-	Colorless crystalline powder

Fatal dose 100-400 mg of mercuric chloride.

Fatal period Few hours 1 to 2 weeks.

Toxicity rating 5 or 6 for most of the salts.

Signs and Symptoms

Mercurial poisoning manifests clinically in two forms. However, patterns and severity are dependent on the form of mercury and the route of exposure.

Acute Poisoning

Symptoms commence about half an hour after swallowing mercuric chloride—

- Initially there will be acrid, metallic taste, feeling of constriction of throat, hoarse voice and difficulty in breathing. Tongue and mouth gets corroded followed by burning sensation extending down the abdomen. Vomiting of greyish slimy material with bloody streaks is then followed by blood stained diarrhoea and tenesm us. Oral consumption can lead to glossitis, ulcerative gingivitis, and necrosis of the jaw.
- Nephrotoxi city leading to albuminuria, cylindruria, uraemia, acidosis, etc. Urine will be scanty and contains blood and albumin. Toxic mercury compounds are considered as *nephrotoxic poisons* and cause renal tubular and glomerular necrosis.
- Inhalation of fumes of mercury can lead to metallic taste, salivation, gingivitis, and loosening of teeth with foetid teeth.
- Strong concentration may also cause ataxia, paresis, delirium, etc.
- Locally, mercury salts have corrosive action.
- Blood peripheral smear can show *leucocytosis*, while *leucopenia* occurs with organic mercurial poisoning.

Chronic Poisoning

(Hydrargyrism / Mercurialism)

The synonym *Hydrargyrism*is after the Latin word *Hydragyrus*, which means mercury. Chronic poisoning occurs where the victim is exposed to mercury fumes in factories or excessivedose of mercurial compound are used for a prolonged period. Symptoms begin to appear at blood levels of 100 nano gramper cent of mercury. The victim manifests with:

- Excessive salivation (*ptyalism/sialorrhoea*), with swollen and painful salivary glands, metallic taste in the mouth, glossitis, ulcerative gingivitis, and necrosis of the jaw.
- A blue line on the gums called *Burtonian line* is a common clinical finding of chronic poisoning.
- Nausea, colicky pain, vomiting and diarrhoea are other gastro intestinal manifestations.
- Evidence of nephritis and uraemia may be seen.
- *Mercuria lentis* develops which is due to brownish deposit of mercury through the cornea on the anterior lens capsule and can be observed as a brown reflex on slit-lampexamination.
- *Mercurial tremors* can be detected in early stages with change in handwriting of the person as it first affects the muscles of the finger, followed by muscles of the tongue causing stammering and slurring speech, and finally affecting the muscles of the face, arms and legs. This was referred to as *Hatter's shake* in the past among the workers of *Hat industry*, where in mercury was used extensively in giving the peculiarkinking shape to the felt hats. Other drugs and poisons which produce tremos are—Alcohol, phenothiazines, caffeine *and*

theophylline, tricyclic antidepressants, carbon monoxide, and phosphorus.

- *Mercurial erethism* comprises personality change resulting in an abnormally high degree of irritability or sensitivity or excitability, shyness, amnesia, insomnia, delusions, hallucination, leading to insanity.

Treatment

- Perform gastric lavage with 5 per cent solution of sodium formaldehyde sulfoxylate. About 100 ml of the same maybe left in the stomach after the lavage.
- Administer demulcents like egg albumin.
- Administration of medicinal charcoal with magnesium sulphate is of great use.
- Specific antidotes are BAL (dimercaprol at a dose of 3-4mg/kg body weight. every four hourly), or penicillamine ata dose of 250 mg to 2 gm orally, or sodium formal dehydesulphoxylate as chemical antidote, etc.
- Symptomatic therapy.

Postmortem Changes

- ***External*** – Nothing specific.
- ***Internal*** – Mucous membrane of lips, mouth and pharynx show diffuse greyish white escharotic appearance. Stomach and intestine show severe irritation and corrosion with ulceration and softening. Intestines especially the caecum, colon and rectum are found to be inflamed, ulcerated or even gangrenous where the patient survives for some days. Kidney shows the findings of toxic nephritis. Liver shows fatty degeneration. Heart shows subendocardial haemorrhages and

fatty degeneration.

Medicolegal Importance

- Mercury is an industrial poison. It is used in industries, connected with manufacture of thermometers, barometers, mercury vapour lamps, firecrackers, explosives, paints, etc. *Hatter's shake* or *glass blower's shake* which are moderately course tremors interspersed by jerky movements found inthe workers of *glass blowing* and *hat industry* are some examples for chronic poisoning of industrial origin. Chronic mercury poisoning manifestations are prevalent among the gold miners and gold refining industry workers who are exposed to mercury used in the process.
- The medical use of mercury as diuretics, vaginal douching purposes, as a dental restorative material in dental clinical, etc. can also lead to accidental poisoning (by overdose)
- Incidences of suicide and homicide with mercury is though rare, accidental poisoning is quite frequent especially among the children, e.g. *Pharaoh's serpent (Diwali poison)*—is a black coloured tablet shaped fire cracker, which contains mercuric thiocyanate and on ignition yields a long black coloured tubular ash.

LEAD (SHISHA)

Lead is a metallic inorganic irritant poison. According to Health And Human Services Department USA, lead poisoning is the *most important environmental problem for the young children.* Blood levels once thought to be safe have been shown to be associated with IQ deficits, behaviour disorders, slowed growth, and impaired hearing.1 Studies in population blood-leadconcentrations have shown a fall by up to 80 per cent in the last twenty years, cases of lead poisoning continue to occur. Poisoning is more common from chronic

occupational exposure among lead smelters, battery manufacturers, painters, decorators, etc. Chronic exposure may also occur at home from paint, pottery and contaminated drinking water by lead pipes used for city water supply.

Absorption

Absorption is possible through all routes.

Action

Pure metallic forms are nontoxic is a steel-grey metal. However, lead compounds can act by producing spasms of the capillaries and arterioles or by fixation of the poison in the tissues such as brain, bones, etc. It can also combine with *sulphydryl enzymes* and interfere with its action. Lead can decrease synthesis of hemeleading to anaemia and can bring about hemolysis as well asrelease immature RBCs into circulation (reticulocytosis and basophilic stippling of RBCs). Lead can destroy nerve cells, myelin sheaths in CNS and also produce cerebral edema. It alsoexerts toxic effects on kidneys (nephritis) and reproductive system(infertility).

Showing inorganic compounds oflead

Compound	*Common Name*	*Properties*
Lead acetate	Sugar of lead Sugar of Saturn	White acicular crystals
Lead carbonate	White lead Safeeda	White powder
Lead nitrate	-	Crystalline powder
Lead Sulphate	-	White powder
Lead chromate	Chrome yellow	Bright yellow powder
Lead chlorate	-	White needle shaped crystals
Lead iodide	-	Bright yellow powder
Lead sulphide	Surma	Cubic crystals
Lead monoxide	Mudrasang Litharge	Brick-red scaly mass
Lead tetraoxide	Sindoor Metia Red lead	Scarlet crystalline powder
Lead tetra-ethyl	-	Heavy oily volatile liquid

Fatal dose depends on toxic compound (20 gm of leadacetate).

Fatal period 1 to 2 days.

Toxicity rating 3 or 4 for most of lead salts.

Signs and Symptoms

Lead poisoning clinically manifests in two forms: acute and chronic forms.

Acute Poisoning

This usually occurs with high dosage of lead acetate, starts with burning and dryness in the throat, salivation and intense thirst. Vomiting occurs within 24 hours with colicky pain and tender abdomen. Constipation is a common feature. Urine is scanty. Finally there may be peripheral circulatory collapse, headache, insomnia, paresthesia, depression, convulsions, exhaustion andcoma leading to death.

Subacute Poisoning

This type of poisoning occurs from repeated small doses of leadacetate. Blue line on the gums is seen with gastrointestinal symptoms. Urine is scanty and in deep red colour. In the later stages, nervous symptoms become prominent with numbness, cramps and flaccid paralysis of lower limbs. Death is rare but may be followed by convulsions and coma.

Treatment of Acute and Subacute Poisoning

- Emetics
- Stomach wash with 1 per cent magnesium or sodium sulphate solution
- 25 gm of magnesium sulphate orally with demulcent drinks
- Calcium gluconate 1 gm to relieve colic

- Intravenous fluids
- Chelating agents like EDTA, BAL and penicillamine are helpful.

Chronic poisoning (Plumbism, Saturnism) Chronic poisoning with lead compounds manifests with a set of symptoms, which may be enumerated as:

Facial pallor: Pallor seen especially around the mouth also known as *circum oral pallor* is due to the *vasospasm* of the capillaries and arterioles, around the mouth.

Anaemia: Hypochromic, microcytic anaemia with *reticulocytosis* and *punctate basophilia* with presence of marked *basophilic stipplings* in the RBCs. Platelet count decreases. Anaemia is probably due to decreased survival time of RBCs and inhibition of haem synthesis by interference with the incorporation of iron into protoporphyrin.

Burtonian line (lead line): It is a stippled blue line seen atthe junction of the gums usually nearer to a tooth caries, especially in the upper jaw. This is due to the deposition of lead sulphide formed by the action of the combination of lead with hydrogen sulphide which had evolved from the decomposed food debris in the caries tooth.

Lead colic and constipation: The victim will complain of severe colicky pain abdomen relieved by pressure and bowel irregularities. Abdominal muscles become tense and retracted.

Lead palsy: There is a typical paralysis affecting the extensor muscles of the fingers and wrist causing '*wrist drop*' and '*clawshaped hand*'. Similarly paralysis may extend to the extensor muscles of the foot leading to foot drop.

Lead encephalopathy: Mostly seen in infants presenting with severe ataxia, vomiting, lethargy, stupor, convulsion and coma. Cerebral

psychic affection may be present.

Cardiorenal manifestations: Elevated blood pressure and arteriosclerotic changes are observed. Urine contains albumin and abnormal quantity of lead, coproporphyrin III and deltaaminolaevulinic acid. Interstitial nephritis may occur.

General manifestations: such as weakness, anorexia, metallic taste in the mouth, dyspepsia, foul breath, etc.

Laboratory Diagnosis of Chronic Lead Poisoning

- Urine lead levels of more than 0.08 mg per litre collected in 24 hours
- Blood lead level more than 0.8 mg per litre
- Increased coproporphyrin level in urine
- Increased urine and plasma delta-amino laevulinic acid
- X-ray evidence of *increased density* or *radio opaque bands*or *lines* at the metaphyseal ends of long bones in children. This is are also referred to as *lead lines*.
- Presence of lead as *radio opaque material* on X-ray stomach and intestines may be seen in children particularly with history of *pica* (meaning abnormal craving for non-nutritivesubstances).

Treatment

- Potassium or sodium iodide for eliminating lead through the kidney.
- Large dose of sodium bicarbonate: 20 to 30 gm per day in divided doses increases the output of lead owing to thetransformation of the insoluble tribasic lead phosphate in to the soluble dibasic lead phosphate through the liberated carbonic acid.
- Calcium gluconate or calcium chloride to relieve colic

- Saline purgatives like magnesium sulphate or sodium sulphate to remove lead from the bowel
- Calcium disodium versanate as deleading agent

Postmortem Changes

- ***Acute Poisoning***
  - ***External:*** Nothing specific.
  - ***Internal:*** Stomach-gastric mucosa is congested, eroded and patchy in appearance with greyish white deposits. Large intestine may show black colored faecal matter. Evidence of renal tubular degeneration.
- ***Chronic Poisoning***
  - *Blue line* on the gums. Muscles are flaccid and show fatty degeneration. Intestines are contracted and thickened. Liver and kidneys are hard and contracted. Heart is hypertrophied. Renal tubular necrosis is usually noticed.

Medicolegal Importance

- Lead is an *industrial poison* presenting as an *occupational hazard*. It is commonly used in industries concerning manufacturing of battery cell, paints, crayon, hair dyes, toys, etc. Accidental poisoning by contamination of the drinking water occurs in places where *lead pipes* are used. Drinking fruit juices or water stored in improperly glazed ceramic wares can result in lead poisoning.
- Lead is *rarely* preferred to commit suicide or homicide.
- In cases of long standing gun shot bullet/s lodged and retained in the body, have reported of chronic lead poisoning due to absorption of lead particles from these bullets. Gunshot wounds in adults and

children has been reported of causing anorexia, abdominal pain, vomiting, anaemia, encephalopathy, seizers, etc. The surface area of retained lead particles, location of retained lead particles (especially synovial fluid), length of time for which one is exposed to lead and type of activation (uncoated bullets-yielding greater surface area of lead for dissolution) are all factors that may lead to lead poisoning.

- Long standing use of cosmetics containing lead salts (*surma, sindhur, vermillion,* etc) can result in chronic lead poisoning.

COPPER *(Thambe, Blue Vitriol)*

Copper, an inorganic metallic irritant, is not poisonous in metallic state, but some of its salts are poisonous, e.g. copper sulphate*(blue vitriol)* and copper subacetate *(virdigris)*.

Copper sulphate is a crystalline salt with blue colour and has a metallic taste. In small dose of 0.5 gm it acts as anemetic, but in large doses it acts as an irritant poison. Poisoningis usually accidental or suicidal. Homicidal use is rare because of its metallic taste and striking blue colour.

Copper Subacetate or Verdigris

Copper subacetate is a bluish green salt. It is formed by the action of vegetable acids, while cooking in copper cooking utensils, which have been not properly tin lined. Thus, accidental *verdigris* poisoning from contamination of food cooked in suchutensils, are often reported.

Signs and Symptoms

Acute Poisoning

- It is reported that renal failure and death may follow ingestion of as little as 1 gm of copper sulphate.18 However, fatal poisoning by

copper is very rare. Symptoms of poisoning commence within fifteen to thirty minutes after swallowing the poison. There is a metallic taste in the mouth. Salivation and thirst are present. The mucosa of the mouth is discolored blue. There is pain in the mouth, oesophagus and stomach. Vomiting and diarrhoea occur. Vomitus is blue or green incolour. Stool is brownish or bloody. Oligurea, haematuria and uraemia may develop in some. There may also be low urinary output with casts and albumin in urine. Jaundice occurs in severe cases due to centrilobular necrosis and biliarystasis. Later muscular spasms cramps, coma and circulatory collapse precede death.

- A rare syndrome of *intravenous copper intoxication* with symptoms of nausea, vomiting, abdominal pain, diarrhea, anxiety and depression due to copper released from coppertubing during *haemo dialysis* was noticed among patients under going haemo dialysis.
- ***Fatal dose*** Copper sulphate—30 gm *Verdigris*—15 gm
- ***Toxicity rating*** 4 for copper salts.
  - ***Treatment***
- *Stomach washes* with warm water. Egg albumin acts as anantidote by forming an insoluble and innocuous copper albuminate. Stomach wash with potassium ferrocyanide 1per cent solution in water also acts as an antidote by formingcupric ferrocyanide.
- *Calcium EDTA or BAL* is the recommended antidote.
- Maintain electrolyte and fluid balance.
  - ***Autopsy Finding***
- Skin may be yellow due jaundice.
- The mucosa of the mouth, oesophagus and stomach are discolored greenish-blue and may show areas of corrosion and congestion.

- Colon and rectum may show large ulceration or perforations.
- Liver may be enlarged and show fatty degeneration.
- Copper is one of those poisons that can be detected by its characteristic colour.
- The kidneys are congested and may show focal necrosis of *proximal tubules*.

Chronic Poisoning

Chronic poisoning is common among the industrial workers of copper and copper salts or its alloys owing to inhalation of copper dust or fumes. Copper welders may develop the *metal fumefever*. Chronic copper poisoning is also observed among those who consume contaminated food with verdigris obtained from dirty copper vessels for a long period. Airborne dusts of inorganic copper salts have been reported to produce low toxicity. *Hystiocytic granulomatous lung* and *liver disease* have been observed among individuals who had been exposed to copper sulphate spray for 2 to 15 years.

- ***Sign and Symptoms***

- The symptom complex of chronic poisoning is called by several names: *hemo chromatosis, bronzed diabetes* and *pigment cirrhosis.*
- It presents with green or purple line on the gums, coppery taste in the mouth, nausea, headache, colicky pain, vomiting and diarrhoea, and anaemia. Atrophy of the muscles maybe the other symptom observed.
- Skin is jaundiced.
- Urine and prespiration become green.

- ***Treatment***

- Remove the cause and prevent further exposure.

- Provide fresh air.
- Give massage and warm bath.
- Provide proper diet.
- Copper vessels if used for cooking, should be tinned and regularly kept scrupulously clean.

o ***Autopsy Finding***

- No findings externally in acute poisoning cases. The mucosa of mouth and tongue may show bluish or greenish blue tinge. Internally the same tinge is observed with mucous membran esof oesophagus and stomach. Stomach mucosa is congested desquamated and hemorrhagic. Upper part of small intestine may also show mild to moderate irritation. The chief findings are fatty degeneration of liver and degeneration of the epithelial cells of the kidneys.
- In chronic poisoning cases gums appear unhealthy with bluish lining. There is mucosal atrophy. Liver and kidneys show varying degree of degeneration. Poisoning due to Inhalation of vapours chronically can present with findings of chronic pneumonitis. Blood picture may show premature cells in the peripheral smear of the victim.

Medicolegal Importance

- Copper coins when swallowed may remain in the stomach or in the intestine for days without producing any poisoning symptoms. However, when alloyed with other metals and reduced to a fine powdery state, copper may act as poison. All copper salts are poisonous.
- The colour and the strong metallic taste prevent it from being used for homicidal purposes. However cases of using copper sulphate mixed with powdered glass, sweetmeat or some other food is known in India.

- Fair number of suicidal cases are reported in India as it isused in leather industry and for white washing.
- Accidental poisoning by contaminated food due to Verdigris is often reported.
- Copper sulphate is used often as a preservative or colouring agent to vegetables. It is added often to impart rich green colouration to tinned green peas, and mango pickles. Quantity added is usually small (<60 mg) and hence toxic effects are not produced. Conversion into harmless albuminate of copper in the stomach may be the other reason for resultant toxicity. However, constant consumptions of such food articles can lead to chronic poisoning manifestations.
- Copper is a normal and essential constituent of human body and is found in urine, feces, blood and other biological fluids and in liver (Normal serum level is 151.6 micrograms).

IRON

Iron is an inorganic metallic irritant. Mills and Curry have comprehensively surveyed the current status of iron intoxication. There has been an annual average of 22,000 reported exposures to medications containing iron over last 3years. Most exposures involve children less than 6 years of age who have ingested paediatric multivitamin preparations. Most of these patients remain asymptomatic or develop minimal toxicity. Concentrated iron supplements overdoses more of ten results in serious poisoning and can present to emergency department at any stage. However, if the patient doesn't develop any symptoms within 6 hours of ingestion, it is unlikely that iron toxicity will develop. Iron salts are used for treatment of prophylaxis from iron deficiency anaemia. There are several Indian iron preparations, containing different amounts of

elemental iron. Usually iron salts poisoning incidences are reported in children due to consumption of adult dose by mistake, or while giving intravenous injection. Ferrous sulphate and ferric chloride are some of the toxic compounds.

Action

The early features of iron poisoning are due to corrosive effects of iron, while later effects are largely due to the disruption of cellular process. Iron tablets may adhere to the stomach and duodenum causing irritation and in severe cases haemorrhagicnecrosis and perforation. Absorbed iron is rapidly cleared from extraellular spaces by uptake into parenchymal cells, particularly in the liver. It causes mitochondrial damage and cellular dysfunction resulting in metabolic acidosis and necrosis. Eventually widespread organ damage become apparent, hepatic failure with hypoglycaemia and coagulopathy may develop and this is often fatal.

Signs and Symptoms

The clinical course of iron poisoning occurs in four phases:

Phase 1*:* In the first few hours (from 30 minutes to several hours) after ingestion, there is vomiting, abdominal pain and haemorrhagic gastroenteritis with black or grey vomitus and stool with metallic odour. In severe cases gastrointestinal haemorrhage can result with circulatory collapse and coma may supervene.

Phase 2*:* In the second stage, 6-24 hours after ingestion patient shows improvement and the clinical symptoms abate and the patient either recovers or moves on to next phase. In severe cases this may not appear or a latent phase occurs and is deceptively reassuring.

Phase 3*:* Occurs in 12 to 48 hours after ingestion, which is characterized by severe lethargy, coma, convulsions, gastrointestinal haemorrhage,

shock, cardiovascular collapse, metabolic acidosis, hepatic failure with hepato cellular necrosis, jaundice, hypoglycaemia, coagulopathy, pulmonary oedema and renal failure.

Phase 4*:* This is a late phase of complication after 2-5 weeks with formation of gastric strictures and pyloric stenosis.

Diagnosis

- X-ray abdomen shows iron tablets
- Serum Iron level > 150 microgram per cent
- ***Fatal dose*** 20-40 gm of ferrous sulphate/> 150 mg of elemental iron.
- ***Fatal period*** Uncertain
- **Toxicity rating** 3.

Treatment

- Gastric lavage, with dilute (2% solution) of sodium bicarbonate.
- Demulcent drinks like milk or egg albumin is useful.
- Whole bowel irrigation in acute poisoning is found to be safe and effective method. However, there is no report on controlled studies confirming this.
- Electrolyte correction. Intravenous glucose.
- *Antidote:* De-feroxamine (De-sferrioxamine) is the specific antidote. A solution of 2 gm in 1 litre of water can be used for gastric lavage; followed by 2 gm in 10 ml sterile water should be left in stomach. 2 gm of this is then given intramuscularly or by a slow IV infusion at the rate of 15mg/kg body weight per hour, to a maximum of 80 mg/kgin 24 hours.

Medicolegal Importance

- Till recently iron was believed to be nontoxic. However, itis well known today that iron salts in excess can produce poisoning and acute toxicity often results in children due to over dose. Iron poisoning is usually, accidental.
- Suicidal or homicidal ingestions by iron are rarely reported.

ZINC (JASAT)

Zinc is soft, bluish white, lustrous metal. The poisonous compounds are listed.

Showing inorganic compounds ofzinc		
Compound	*Common Name*	*Properties*
Zinc sulphate	White vitriol	Colorless crystalline salt
	Safed tutia	Resembles magnesium sulphate and oxalic acid
Zinc chloride	Butter of zinc	Colorless opaque mass
Zinc oxide	Jasat Bhasam White zinc	White colorless powder
Zinc phosphide	-	Steel gray powder with garlicky odor
Zinc stearate	-	White bulky powder

Clinical Features

- Metallic taste
- Salivation
- Vomiting
- Substernal pain
- Abdominal pain

- Diarrhea
- Convulsions
- Shock
- Metal fume fever
- Pneumonitis
- Acute pancreatitis with raised serum amylase level
- Hepatic damage
- Renal failure
- Coagulation failure.

Fatal Dose

- Zinc phosphide – 5 gm
- Zinc sulphate – 10 to 20 gm
- Zinc chloride – 5 gm

Fatal Period: *Variable*

Management

- Gastric lavage
- Chelation
- Supportive.

Autopsy Findings

- Acute hemorrhagic gastritis, esophagitis
- Pancreatitis
- Liver necrosis
- Acute tubular necrosis

- Pulmonary edema

Medicolegal Importance

- Accidental poisoning – common
- Suicide/homicide – rare
- Metal fume fever.

ANTIMONY

Poisonous antimony compounds are given in Table.

Showing inorganic compounds of antimony

Compound	*Common Name*
Antimony tartarum	Tartar emetic
Antimony trioxide	Antimonious oxide
Antimony trichloride	Butter of antimony
Antimony Trisulphide	Black antimony Surma

Clinical Features

- Vomiting
- Abdominal pain
- Diarrhea
- Hematemesis
- Dermatitis

- Renal failure and oliguria
- Hepatic failure

Management

- Gastric lavage
- Chelation with BAL
- Hemodialysis
- Supportive

Fatal Dose

- Antimony tartarum – 90 to 180 mg
- Antimony trichloride – 8 to 12 ml

Fatal period: 24 hours.

Medicolegal Importance

- Accidental poisoning – common
- Suicide/homicide – rare

METAL FUME FEVER

This is a syndrome caused by inhalation of fumes offollowing metals

1. Antimony
2. Cobalt
3. Cadmium
4. Chromium
5. Copper
6. Iron

7. Lead
8. Manganese
9. Magnesium
10. Mercury
11. Nickel
12. Selenium
13. Zinc

- Persons involved in welding, galvanizing, smelting, metal refining, electroplating, alloy making etc. are affected
- The syndrome resembles flu-like illness and begin safter four to six hours after exposure to metal fumes.
- It is characterized by fever, chills, myalgia, cough, dyspnea, fatigue, metallic taste, salivation, sweating and cyanosis.

ORGANIC IRRITANT PLANT POISONS

ORGANIC IRRITANT POISONS

An extended communication on all the Indian poisonous plants in a particular region is though beyond the scope of this section, some important plants commonly involved in poisonings, follows logically. It would be however relevant to mention here that a great deal of ignorance about these poisonous plants is a fact even among many of the clinicians routinely dealing with poisoning cases. The problem is made more intense by the fact that there is no accurate information available in India, since very few cases are reported or published in literature. It is also agreed that many of the experimental works are performed on laboratory animals and discussed in veterinary literature. The applicability of such studies in human beings is an open challenge. It is also true that many of plant poisoning cases the treatment is practically same, i.e. symptomatic measures and supportive therapy. Rarely these cases have any antidote therapy. There is virtually chaos in the areas of plant identification and nomenclature.

These are organic irritants derived from poisonous plants. Phyto toxicology is term used to denote the study of plants that produce or evoke specific deleterious effects on human. The organic irritants are classified as:

1. Gastrointestinal irritants:
   - e.g. castor, abrus, capsicum

2. Cardiotoxic poisons:
   - e.g. aconite, oleander, tobacco, etc.

3. Neurotoxic poisons:

- Datura, cannabis, opium

4. Hepatotoxic poisons:

- e.g. neem, akee (Blighia sapida)

5. Dermal irritants:

- e.g. mango, St. John's wort.

Poisonous Parts of the Plants

All parts of plant are poisonous,

- Nerium odorum
- Cerbera thevetia
- Calatropis

Leaves

- Tobacco
- Conium maculatum (hemlock)
- Curare
- Digitalis

Fruits,

- Capsicum annum
- Strychnous nux vomica
- Colocynth

Seed,

- Abrus
- Castor
- Croton
- Datura
- Semicarpus anacardium

Stem/bark,

- Cinchona bark
- Plumbago rosea

Root,

- Aconite
- Plumbago
- Colocynth

Toxic substances in plant may present in form of:

- Toxalbumin (phytotoxin)
- Alkaloids
- Glycosides
- Resins
- Irritant juices
- Acrid oils
- Amino acid
- Plant acids

Toxic Substance And ActivePrinciples In Plants

- **Toxalbumin**
  - Abrin
  - Ricin
  - Crotin
- **Alkaloids**
  - Atropine
  - Strychnine
  - Aconitine
  - Nicotine
  - Curarine
  - Ergotamine
- **Glycosides**
  - Nerin
  - Thevetin
  - Cerebrin
  - Digoxin
  - Calotropin
- **Resins**
  - Cannabinol
- **Juice/oils**
  - Semicarpol
- **Plant acids**
  - Oxalic acid
  - Malic acid

ABRUS PRECATORIUS

Distribution—Grows all over India

Common name—Jequirity bean, rosary pea, Buddhistrosary bead, rosary bead, Indian bead, Indian liquorice, Seminolebead, prayer head, crab's eye, weather plant, lucky bean, *ojode pajaro, gulagunchi, rati,* etc.

Family—Leguminoceae.

Plant characteristics—It is a slender vine and climber, with compound leaves having 10-15 pairs of narrow leaves, smallpinkish flowers with seedpods which split open when ripeexposing 4-6 seeds within. These seeds are bright red in color with black spot in one pole and weigh about 105 mg.

Toxic part of the plant—*Whole* plant is poisonous.

However, seeds are more often used.

Toxic Principles

- N-methyltryptophan
- Glycyrrhizin (lypolytic enzyme—the active principle of licorice)
- Abrin (Toxalbumin*)
- Abrine (amino acid)
- Abralin (glucoside)
- Abric acid

Signs and Symptoms

Signs and symptoms manifest only if the seed is masticated and swallowed. It can act both locally as well as remotely.

- *Locally*—can lead to dermatitis, conjunctivitis, rhinitis, asthma, etc. Oral ingestion can produce severe gastroenteritis, hemorrhagic gastritis with severe pain, copious vomiting, and diarrhoea that may

become bloody, severe thirst and circulatory collapse. *Death* is reported to be due to persistent gastroenteritis.

➢ *Remotely* – when implanted as 'suis' or the seed extract is injected parenterally, the person can develop cardiacmanifestations like a *viperine snakebite*, with the site of injection turning oedematous and haemorrhagic. Victim (animal/human)then turns drowsy, unable to move, goes into coma, followed by convulsions and death. According to Seth, Lal et al abrin can lead to development of cardiac arrhythmias, convulsions and cerebral oedema.

Usual Fatal Dose—60-120 mg of abrin (1-2 crushed seeds)

Fatal Period—3-5 days

Toxicity Rating—5 to 6 *(Super toxic)*

Treatment

All cases who report within 4 hours of ingestion should be treated by usual method of decontamination (lavage, charcoal, and cathartics). The presence of spontaneous diarrhea may obviate the need for cathartics. However following have also been found to be effective:

➢ Oral poisoning cases give: Acid hydrochloric pepsin mixture and 10 per cent sodium bicarbonate IV.

➢ Local injected cases: Dissect out the *Suis*.

➢ Symptomatic measures as required.

Postmortem Findings

➢ Findings show inflammatory changes and congestion of gastro intestinal tract.

➢ When injected, local signs of inflammation are seen.

Medicolegal Importance

It is a commonly used cattle poison in Indian villages by injecting the

seed extract into the animal in the form of certainfine needle-shaped structures known as *Sui* (meaning *needle* in *Hindi*). These are prepared by mixing the seed extract with opium, datura, and spirit/water and then blended into paste, shaped into fine needles and dried in the sun and used to kill cattle by driving it deep into the animal body by blowing through a hollow bamboo pipe.

Suis have been also used criminally and reported of *homicides* often in the Indian villages. It is kept in between the fingers of hand and slapped on the face of a victim, driving it deep into the skin, which releases toxic principle abrin and brings about its action.

Seeds are often used in rosary beeds, necklace, etc. in rural India.

Seeds reported of accidental poisoning among children, on account of their attractive colour and ingested orally, mistaken for a peppermint or just out of curiosity.

Indian goldsmiths sometimes use seeds as a measure to weigh gold or precious stones.

Seed are reported to have being used as birth control pills in the past in rural India.

RICINUS COMMUNIS *(Castor Oil Plant)*

Distribution—Grows all over India, especially in waste lands.

Common name—Castor, arandi, mole bean.

Family—Euphorbiaceae.

Plant Characteristics—It is a large shrub with greenish-redleaves. Fruits are borne in clusters and are soft-spined greenish/brownish capsules with seeds. Seeds are oval/round in shape and are of two types:3 larger in size, red in colour with brown blotches (yields 40% oil) and second variety small in size, grey in colour with glossy bright, polished, brown mottling

(yields37% oil) (Figs 33.6A to C).

Toxic part—Seeds, especially the seed oil (*castor oil*) extract, which is pale yellow in colour and with faint odour and an acrid taste. Left over *cake* after the extraction of oil is also highly toxic.

Active principle—*The oil extract of the seeds has an acid calledricinoleic acid* and the left over cake has the *toxalbumin* called *ricin*. Ricin is one of the most toxic *parenteral* substances in theplant kingdom. It contains two polypeptide chains held together by a single disulphide bond. Both these chains can bind with cell surface facilitating toxin entry into the cell and then disrupt the protein synthesis. Since the cell binding and protein disruption needs some time, its toxic effects are usually delayed but are wide spread.1 Ricin is more poisonous than cobra venom an disclassified as *super toxic poison*.

Signs and Symptoms

➢ *Locally*—It can lead to dermatitis, conjunctivitis, rhinitis, asthma, etc. Castor bean dust is highly allergenic and may cause *anaphylaxis*.

➢ *Orally*—Seeds are effective orally only if *masticated* and swallowed. It produces burning pain in the throat, followed by nausea, vomiting, colicky pain in the abdomen and bloody purging. Both can ultimately lead to dehydration, muscular cramps, etc.

➢ *Parenterally* — It can produce same manifestation as on oralingestion, but occurs more rapidly than oral route.

Fatal dose—1 mg/kg body weight or 6 mg of ricin (about 8-10 seeds).

Fatal period—Several days.

Toxicity rating—Ricin 6 (Super toxic), castor oil 2 (Slightly toxic).

Treatment

Every patient should receive the usual measures to prevent absorption (syrup ipecac, charcoal, cathartics) after taking usual precautions.

Recommended treatment for asymptomatic patient who has chewed one or more seeds include emergency department evaluation, gastric decontamination, administration of activated charcoal, observation for 4 to 6 hours, and discharge with instruction to return if symptoms appear. However, all symptomatic patents need hospitalisation for treatment with IV fluids, supportive care, and monitoring for hypoglycaemia, haemolysis and complications of hypovolaemia. Most patient respond well to IV fluid therapy and recover without any permanent sequelae. Enumerated below is the line of management:

- Prevention of absorption by giving syrup ipecac in ducingemesis/ activated charcoal/ cathartics.
- Gastric decontamination by stomach washes with water.
- Give plenty of demulcents.
- Rehydrate the victim by intravenous fluid and maintain electrolyte balance.
- Blood transfusion may be needed in some cases.
- Other symptomatic measures as needed.

Postmortem Findings

Inflammatory changes and congestion of gastrointestinal tract. Liver, kidney and pancreas are considered as primary target organs clinically, may show inflammatory changes and congestion on autopsy.

- *Microscopy*: Microscopic examination, of stomach contents in a victim, revealing a prismatic appearance of the outer cells coat of the castor seeds (and also in *croton, abrus, and jatropa seeds*), has helped in identification of the poisoning.

Medicolegal Importance

Castor oil obtained from smaller variety seeds, is usually usedin medicine purposes as a *purgative*. Oil which is obtainedfrom bigger variety of seeds is used largely for illuminationpurposes or as an industrial lubricant.

Poisoning is usually accidental.

CROTON TIGLIUM

Distribution—Croton plant grows all over India, especially in wastelands. Grown in many varieties for their brightly coloured foliage; it is widely cultivated as a houseplant.

Common name—Croton, *Jamalgota, Naepala.*3

Family—Euphorbiaceae.

Plant Characteristics—It is an evergreen tree with smoothash-colored bark. The leaves of the tree are ovate-lanceolate.Flowers are small, and oblong. Fruits are three lobed containing oval, dark brown seeds, with brownish black colour and longitudinal striations. Seeds though resemble castor seeds; the longitudinal striations

mark the difference from castor seeds, which has mottling.

Showing difference between castorand croton seeds		
Features	*Castor seed*	*Croton seed*
Appearance	Grayish brown, mottled, glossy	Dark brown,non-glossy, notmottled
Shape	Flattened-oval in shape	Oval
Cross sectionat tip	Lumen is almost circular	The lumen is slit like
		with radiating creases

Toxic part—Seed and oil extracted from the seeds is extremely toxic. Seed oil is commented to have tumour promoting *phorboldiesters*.

Active principles—There are two active principles:

- Crotin (toxalbumin)
- Crotonoside (glycoside)

Signs and Symptoms

Resembles *Ricinus communis* (castor) poisoning manifestation

Fatal dose—1 to 2 ml of oil or 4 to 6 crushed seeds

Fatal period—4 to 6 hours to 3-6 days

Toxicity rating—5 (croton oil)

Treatment

Same as for *Ricinus communis* (castor).

Postmortem Findings

Findings show inflammatory changes and congestion of gastro intestinal tract.

Medicolegal Importance

- Mistaken for castor oil or castor seed, resulting in *accidental*poisoning.
- Cases of deliberate homicide are also reported with Crotonoil. Madras Chemical examiners report mentions one such case in 1936.
- Rarely the croton oil is used as an *abortifacient* in rural India.

CALOTROPIS

Distribution—Grows all over India, especially in wasteland and deserts.

Common name—Madar has two species.

- *Calotropis gigantea, which* is a purple flowered plant.
- *Calotropis procera, which* is a white flowered plant

Family—Asclepiadaceae.

Plant characteristics—It is a tall shrub with yellowish-whitebark, and oblong thick leaves and purplish or white flowers. When the stem, branches and leaves are cut, crushedor incised, it yields milky white latex, which is an acrid juice called *madar juice*.

Toxic part—Stem, branches, leaves and the milky white latex*(madar juice)*.

Active principles—They are four:

- Uscharin
- Calotoxin
- Calotropin
- Gigantin

Signs and Symptoms

- *Locally*—It can give rise to lesions resembling bruises on skin(called *fabricated injuries*), which at times can lead to pustule formation and vescication. Juice when instilled into the eyes or coming in contact with eyes can result in severe *conjunctivitis*.

- *Orally*—Bitter in taste. Produces burning pain in the throat, salivation, nausea, vomiting, etc. followed by diarrhea, painabdomen, mydriasis, tetanic convulsions, delirium, collapse and death.

Fatal dose—Uncertain.

Fatal period—12 hours.

Treatment

- Gastric lavage with warm water or potassium permanganate(KMnO4)

- Give demulcent drinks
- Symptomatic measures
- Washing with soap and water can treat skin lesions.
- Cases of conjunctivitis can be managed by saline irrigations.

Postmortem Findings

Froth at nostrils, stomatitis and inflammatory changes of gastrointestinal tract with ulceration, stomach may show perforation. All viscera including brain usually shows congestion.

Medicolegal Importance

- Parts of the plant or the juice extract are used as folk medicinein rural India.
- Accidental poisoning is common due to quackery.
- The criminal use of the juice includes criminal abortion, infanticide, cattle poisoning, and creating conjunctivitis and *artificial bruises*.

SEMICARPUS ANACARDIUM

Distribution—Grows all over India.

Common name—Marking nut, Bhilwan, Bibva, Bhela, and Oriental Cashew.

Family—Anacardiaceae.

Plant Characteristics—It is a small tree of East Indian origin. Its flower is dull/greenish yellow in colour. Fruit is black, heart shaped with hard rind within which is a thick fleshy pericarp which yields brown oily resinous fluid. This turns black on exposure to air. This fluid is often used as 'marking ink',on linen and cotton clothes by the washer men (*Dhobis*).

Active principles Two active principles are isolated in the fluid

extracted from the pericarp:

- Semicarpol (monohydroxy phenol compound).
- Bhilawanol (alkaloid).

Signs and Symptoms

- *Locally*—On skin produces bruise like lesions which are actually raised blackish blisters or vesicular ecchymatouserruptions, which are itchy and scratching of which can cause similar lesions on the tips of fingers, on the nail beds, below the nail tips. These can lead to pain, fever and stranguria with excretion of brownish urine.

- *Orally*—Large dose can produce blisters in mouth and throat, with gastroenteritis. Can also produce dyspnoea, cyanosis, tachycardia, coma and death.

Fatal dose—Uncertain.

Fatal period—12 to 24 hours.

Treatment

- For skin lesions, wash with water and apply liniments.
- For oral ingestion cases perform gastric lavage and give demulcents
- Symptomatic measures as needed.

Postmortem Findings

Inflammation of gastrointestinal tract and congestion of viscera and skin showing black vesicles with acrid serum.

Displaying difference betweencontusions and marking nut lesion		
Features	*Contusion*	*Marking nut lesion*
Shape	Regular	Irregular
Margin	Diffused	Sharp and clear
Color change	Occurs	Does not occur
Itching	Absent	Present
Extravasation of blood	Present	Absent
Blisters	Absent	Present
Nail beds	Not significant	Similar lesions due to itching
Caused by	Rupture of capillaries	Chemical changes to skin

Medicolegal Importance

➢ Common criminal use of the juice is by malingerers' (those who pretend *injury* or *illness* with some hidden motives) to fabricate wounds (usually a *bruise*) by external application over the skin. However, unlike the true bruise the lesion produced by the fluid will result in blister formation with irritation/itching. A dilute solution of the fluid is also at times instilled in the eyes by malingerers' to induce abrupt *conjunctivitis* or *ophthalmia* as conjunctivae turn red due to local irritation effect.

➢ Accidental poisoning is also common when used as *amedicine* by *quacks*.

➢ Criminally it has been used to pour over geniatalia or introduced into the vagina for infidelity.

➢ Juice if often used as an *Illegal abortifacient* to induce *criminalabortion* by its local application through the *abortion stick* with a cloth piece soaked in it, wrapped over its tip and thrust into the uterus.

➢ Modi has reported cases of homicide and infanticide by marking nut juice:

➢ The fruit extract poured on the body (especially *genitalia*)of the prisoners for extortion of truth or confession of guilt.

➢ Occasionally used to commit *vitriolage*.

CAPSICUM ANNUM

Common name—Chillies, *Lal mirchi, Red pepper, cayenne pepper.*

Family—Solanaceae.

Plant characteristics – It is a small herb bearing some what long, tapering fruits, which become red when ripe, and possess a pungent odor and taste. The fruit (chilly) contains a number of small, flat, yellowish seeds which bear a superficial resemblance to datura seeds.

Toxic part—Fruit and the seeds

Active principles—Capsicin (crystalline) and capsaicin are both acrid, volatile, alkaloid substances.

Signs and Symptom

➢ *Locally*—It can produce irritation resulting in burning and redness of skin; and burning, redness, and lacrimation of the eyes.

➢ *Orally*—Large quantity can produces burning and fiery hot sensation in the mouth, salivation, excessive perspiration, abdominal pain, vomiting and diarrhea. Urine may also turn dark.

Usual Fatal Dose—Can cause serious toxicity.

Fatal Period—Fatality unlikely.

Treatment

➢ *In case of oral ingestion do the following:*

– Stomach wash with warm water

– Blunt scraping of the tongue

– Sucking of ice

– Sips of ice-cold water.

➢ *In case of local skin contamination,*

– Wash the area with copious amount of water.

– According to Jones et al, affected skin may be kept immersed in *vinegar* (5% acetic acid).

Medicolegal Importance

➢ Other than irritation fatality due to the chilly is never reported. It constitutes one of the most common and popular condiments in Indian cooking to enhance flavour or enjoyment. Also they are used in preparation of variety of pickles and sauces. As a carminative or an appetizer in a dilute form it is often used as a household remedy. In modern medicine it has been also used as a countre irritant in the form of an ointment or as an adhesive plasters to relieve muscular sprain and such other conditions.

➢ Dermatitis and burning of hands and fingers known as '*Hunan hand*' is common among the pickle industry workers, who use their hands for handling chilly paste or powder for prolonged period.

➢ The chilly in the form of powder or paste has also been reported to have been used for the purpose of felonious act of extorting truth, by thrusting into the urethra, vagina, rectum, rubbing over breasts in females.

➢ *Datura* seeds may be consumed *mistaken* for chilly seeds, resulting in grave datura poisoning.

➢ Criminals often use chilly powder to facilitate act of felony, robbery, rape, etc. by putting the victim in sudden agonized or helpless condition by throwing it into their eyes prior to the criminal act.

EUCALYPTUS GLOBUS

Distribution—Grows in South India, especially in hills of Nilgiris, Tamil Nadu.

Common name—Eucalyptus, blue gum.

Plant characteristics—It is a tall tree with smooth bark, long curved leaves, and large flowers. Eucalyptus oil obtained by steam distillation of the extract derived from the leaves.

Active principles—Eucalyptol (cineole).

Signs and Symptoms

- Burning pain in the mouth, nausea, vomiting, diarrhea, abdominal pain.
- Bronchospasm, tachypnoea, chemical pneumonitis, respiratory depression.
- Headache, vertigo, drowsiness, slurred speech, ataxia, convulsions, and coma.
- Breath and urine may smells of eucalyptus oil.

Usual fatal dose—5 to 10 ml can cause serious toxicity.

Fatal period—Fatality is unlikely.

Treatment

- Stomach wash
- Symptomatic measures as needed.

Postmortem Findings – Nothing specific

Medicolegal Importance

- Eucalyptus oil is a *house holds remedy* 15 for common ailments like common cold and pain.
- Most of the poisoning cases are due to the accidental consumption, by mistake or due to over dose. However, death due to eucalyptus oil is rare.

AZADIRACHTA INDICA

Distribution—Grows all over India. It is grown for its medicinal use.

Common name—Neem

Family—Meliaceae

Plant Characteristics—It is a tree. Seed grown in the treeyields a yellowish oil (*margosa oil*), which has a disagreeableodour and bitter taste.

Active principles—The oil contains active principles enumerated below:

- Azadirachtin
- Meliantriol
- Salanmin
- Nimbin
- Nimbidin
- Unrefined oil may also contain aflatoxins, which are injurious to health.

Signs and Symptoms

Hepatotoxicity characterized by vomiting, dehydration, drowsiness, encephalopathy and metabolic acidosis.

Usual fatal dose—Can cause serious toxicity.

Fatal period—Fatality unlikely.

Treatment

- Rehydration
- Treat cerebral oedema
- Correct the metabolic, acidosis
- Symptomatic measures.

Postmortem Findings—Not reported

Medicolegal Importance

- For centuries neem leaves has been in use in ayurvedic medicine. Neem is a medicinal plant whose oils, seeds, and leaves contain organic antibiotics, organic pesticides, and organic fungicides. Neem tree products are used in natural cosmetics and organic skin-care products. Margosa oil is used in treating skin diseases, cough, common cold, helminthiasis, etc.
- Resin tapped from the bark provides a gum commonly usedas glue.
- Neem timber has been shown to be decay and insect resistant.
- The Neem tree's wide strong branches produces excellent firewood
- Daily, millions of people brush their teeth with Neem twigs. Dentists confirm that this practice guards them against periodontal disease.
- A paste made from the leaves has been found to successfully treat skin lesions. Also small portions of leaves mixed with regular feed seem to affect intestinal parasites in livestock.
- The neem tree can produce up to 50 kg of olive-like fruit per year from which an antiseptic soap can be made.
- The seed has an active ingredient which acts as a pesticide, insecticide and even a fungicide.

COLCHICUM AUTUMNALE

Common Name—Autumn crocus, meadow saffron, naked ladies

- ***Distribution:*** Eurasia, Africa.
- ***Family:*** Liliaceae
- ***Genus:*** Colchicum
- ***Species:*** Autumnale

Plant Description

Perennial herb (C*ategory:* Bulbs) Height: 15-30 cm, with basal, slender leaves; and long, tubular, 6-parted, flowers which are pink, violet/ lavender or white in colour.

- ***Poisonous part***—All parts of plant are highly poisonous, and may be fatal if eaten.
- ***Active principle***—Alkaloid colchicines and demecolcin.
- ***Mode of poisoning*** — Oral ingestion.

Signs and Symptoms

- *Gastrointestinal system*—Presents with vomiting, diarrhoea, abdominal pain, cramping and hepatic dysfunction.
- *Cardiovascular system*—It can bring about increased blood pressure. Rarely, it can produce disseminated intravascularcoagulation and bone marrow failure.
- *Respiratory system*—Rarely it can produce respiratory failure.
- *Urinary system*—It may also cause signs and symptoms of renal dysfunction.
- *Hairs*—It can produce alopecia.

Usual fatal dose—Can cause serious toxicity.

Fatal period—Fatality unlikely.

Treatment—In oral poisoning cases, perform gastric lavage. Treat with antihy pertensives for increased blood pressure. Renal failure is to be treated by dialysis.

Postmortem Findings – Nothing specific

Medicolegal Importance- Not reported

ERGOT

Common name—Mother of rye

Characteristics

Ergot is an alkaloid. It is the sclerotium (mycelium) of a fungus *Claviceps purpura*, which grows on many cereals like *rye*, barley, wheat, oat, etc. fungus gradually replaces the whole grain toa dark purple mass, which on drying yields ergot.

Active principles—are three and all are *ecbolics*, which can contract gravid human uterus in late pregnancy and they are:

- *Ergotamine*
- *Ergotoxin*
- *Ergometrine*
- They also contract arterioles which can lead to gangrene of part supplied.

Signs and Symptoms

Acute poisoning—Very rare.

GI. Tract: Irritation of throat, dryness, severe thirst, nausea and vomiting, diarrhea, pain in abdomen, tingling in hands and feet, cramps in muscles (all due to smooth muscle contraction),dizziness, feeling of coldness, etc. It might also present with symptoms of hypoglycemia, anuria, abortion and hemorrhage in a pregnant woman. Death is usually slow after a week of poisoning.

Chronic poisoning – is called *Ergotism* and is quite common.

It appears in two forms:

- *Convulsive form*—presents with painful toxic contraction of voluntary muscles followed by drowsiness, headache, giddiness, madness, etc. Victim may complain of feeling of itching/numbness and ant

crawling sensation under the skin.

➢ *Gangrenous form*—Begins as pustules and swelling of limbs and feet, followed by intense hot feeling, severe pain, numbness, etc. and results ultimately into gangrenous changes (resemble Raynaud's disease). Recovery is possible, if ergot is withheld.

Fatal dose and period—both uncertain.

Toxicity rating—4 to 5.

Treatment

- ➢ Stomach wash with tannic acid and magnesium sulfate
- ➢ Amyl nitrate inhalation
- ➢ Sodium nicotinate 140 mg intravenously
- ➢ Withdrawal of ergot contaminated substance in chronic cases
- ➢ Symptomatic measures as needed.

Medicolegal Importance

➢ Ergotamine may be abused by patients with migraine headaches. If it is used for long period, the development of a rebound headache constitutes a major clinical problem which is alleviated only by continued use of the drug.

- ➢ Used as an abortifacient, inducing *criminal* abortion.
- ➢ Accidental poisoning, usually by food contamination
- ➢ Used as medicine.

ORGANIC IRRITANT ANIMAL POISONS

ORGANIC IRRITANTS: ANIMAL BITES AND STINGS

Examples of animal irritants include snake, scorpion, bee, wasp, ant, spiders, centipedes, etc. This chapter deals with commonly encountered animal bites and stings. Snake have venomous bites that inject venom through specialized oral structure called as fangs whereas scorpions, bees and wasp shave stings. The stings of these animals are painful and attimes prove fatal.

Organic animal irritant poisons include effects of bites/stings of poisonous snakes and insects. Envenomation that can occur with these bites and stings lead to toxic condition which at times be serious enough to cause even the death of the victim. The following discussion orbits around the more common of the setoxicological syndrome complexes.

SNAKES AND SNAKE BITES

Snakes are ectothermic (*cold-blooded*) limbless vertebrates of the Class *Reptilia*, which also include lizards, crocodiles and alligators, tortoises, turtles, etc. There are at least 3,000 species of snakes, but only 400 are poisonous, which means most of them is non-venomous. Some snakes have evolved specialized glands, which produce venom, mostly derived from salivary glands. Venom may have several functions for the snake such as rapid *immobilisation* and *predigestion* of prey. According to Burton, *Cobra venom* is a potential source of medicines also, including *anti cancer drugs* and *painkillers.*

Epidemiology of Snake Bite

It is estimated that the true incidence of snake bite/envenomationcould exceed 5 million per year. About 100,000 of these develop severe sequelae. Around 30-40 thousand people die every yeardue to snake bite all over the world. Around 10,000-15,000deaths are reported in India

annually. The global disparity in the epidemiological data reflects variations in health reporting accuracy as well as the diversity of economic and ecological conditions. Hospital records fall far short of the actual number owing to dependence on traditional healers and practitioners of witchcraft, etc. It has been reported that in most developing countries, up to 80 per cent of individuals bitten by snakes first consult traditional practitioners before visiting a medical centre. Owing to the delay, several victims die during transit to the hospital.

Feature of Snakes in General

Body is elongated and covered with horny epidermal scales, which are shed/ moulted off several times a year.

Snakes usually move on the tips of their ribs

Eyelids are fused so appear to be absent

There are no visible external ears, and hence there is a great controversy on whether snakes can hear sound

Skull bones are movably articulated

Tongue is forked at the tip and serves as a sense organ and can be protruded out even when the mouth is closed through a gap in the upper jaw.

Jacobson's organ—The snakes have nostrils and olfactory(smell) organs connected with the smell centre of the brains, they also have an organ called *Jacobson's organ*. It is a cavity in the roof of the mouth with olfactory cells in which the snake inserts the tips of the forked tongue. The snake wave sits tongue, searching for scent molecules, which it then transfers to this organ, where it is analyzed and the information is transferred to the brain.

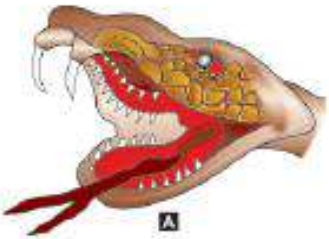
They have got a paired copulatory organ and the cloacalaperture is transverse

Epidermal scales—Scales on the head are plate like andare called *shields*, on the back are small arranged in midline almost quadrangular and are called *vertebrals*, on the sides of the trunk are called *costals*, on ventral aspect (belly) are transversely elongated and are called *ventral shield*, on lowersurface of ventrum of tail are in single/double rows and are called *subcaudals*.

Classification of Snakes

- Venomous/poisonous snakes
- Non-venomous/non-poisonous snakes.

difference between poisonous and non-poisonous snakes		
Features	*Poisonous snake*	*Non-poisonous snake*
Physical features	Stout, dull color	Slender, brightly colored
Tail	Rounded or flattened or abruptly tapering tail	Gradually tapering
Belly scales	Broad and complete	Small and do not extend across the entire width
Teeth	At least one pair of teeth in the upper jaw	All teeth are uniformly small in size, no fangs



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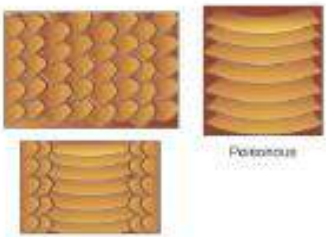

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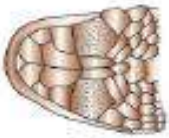

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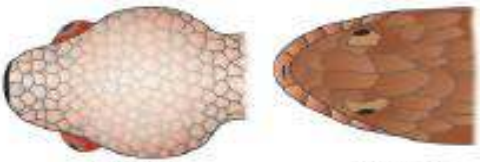


C

Important features of poisonous and non-poisonous snakes shown in the picture above:

(A) Poison apparatus and fangs (canalised and grooved),

(B) Ventral shields (belly scales) and vertebrals,

(C) Head scales,

(D) Cobra (note the hood and spectacle mark)

(E) Cobra – Third supralabial touching eye and nasal shield,

(F) Krait — four infralabials (note the large fourth one),

(G) Krait — enlarged vertebrals on the back, and

(H) Pit viper — a pit between eye and nostril.

VENOMOUS SNAKES

Based on their morphological characteristics including arrangement of scales, dentition, osteology, myology, sensoryorgans, etc. snakes are categorized into several families. Venomous species are usually confined to *5 families - Colubridae, Elapidae, Viperidae, Hydrophiidae,* and *Atractaspidae.*

Colubridae

Only a few of these can cause significant injury to humans. There are also several species of Colubridae that have modified salivary glands producing toxins, but *without* true fangs. They rely instead on the trauma caused by other teeth to provide an entry track for their "venom". A few of these may cause some effects in humans, though are not expected to be lethal.

Elapidae

Include "*cobra*" type snakes, common in Asia and Africa. They have small to moderate sized fangs at the front of the mouth, possibly the "*true fangs*". Cobra venoms are quite toxic and they are a major cause of human

envenoming morbidity and mortality. Examples are *cobras, kraits, coral snake s*and *mambas.*

Viperidae

Vipers constitute this family. All species have *well-developed,longer fangs* on hinged maxillae, allowing *rotation*(elevation) when biting, than in any other groups of venomous snakes. Venom glands are also typically *larger* than in other groups. They have heat-sensing pit organs at the front of the head, giving some degree of infrared or heat sensitive "vision".Vipers are a major cause of snakebite in the Americas, Africa, Europe and Asia. Examples are *vipers, pit vipers,* and *rattlesnakes.*

Hydrophiidae

These are *sea snakes* and are closely related to the *cobras*, with similar fang structure, but live most or all of their lives in anaquatic, usually *marine* environment. They are a significant cause of envenoming amongst fisherman in the Indian and Pacific oceans. Example: *Stoke's sea snake.*

Atractaspidae

These are side fanged *viper like* snakes confined to Africa and the Middle East. They have unusual fang structure and venoms, which contain *endothelin* like compounds called *sarafotoxins*, causing potent smooth muscle contraction.

The Major Families and Speciesof Venomous Snakes in India

There are about 52 poisonous species of snakes in India and they belong to *three* families:

Elapidae, which includes *common cobra, king cobra and krait Viperidae,* which includes Russell's viper, pit viper and sawscaled viper and *Hydrophidae (the sea snakes).*

However, among these, majority of bites and consequent mortality in India is attributable to only five species and they include King Cobra

(*Ophiophagus hannah*), Common Cobra (*NajaNaja*), Russell's Viper *(Daboia rusellii*), Krait (*Bungarus caeruleus*) and Saw-scaled Viper (*Echiscarinatae*).

Features of common poisonous snakes

Common cobra

- Zoological name: Najanaja
- Common names: Common cobra, nag
- Features:
  - Common cobras are usually brown or black in color
  - Head is covered with shields. The third supra-labial shield touches the eye and nose
  - A small wedge shaped scale called as cuneate is present between 4th and 5th infra-labials
  - Pupils are round
  - Hood is present. Dorsal aspect of hood may have monocellate (monocele) or binocellate (spectacle) mark. Ventral surface of hood have two dark spots
  - Fangs are short, grooved and situated anteriorly
  - Tail is cylindrical. Caudal scales (scales on undersurface of tail) are divided and double
  - Venom — neuro toxic.

Common krait

- Zoological name: Bungarus caeruleus
- Common name: Indian krait, common krait, Maniyar, Kawadya
- Features:
  - Usually steel blue or black in color with single or paired white

bands on back. The bands are more distinct towards the tail

- Pupils are round
- Large hexagonal scale presents over back
- The 4th infra-labial scale is the largest scale of other infra-labial scales
- The subcaudal (ventral scales distal to vent) are undivided and entire
- Fangs are short, grooved and situated anteriorly
- Venom — neuro toxic.

Banded Krait

- Zoological name: Bungarus fasciatus
- Common name: Banded krait
- Features:
  - Inverted "V" shaped mark on head
  - Broad black and yellow glistening bands encircle the body. On cross-section, the bands are triangular in shape
  - As per habitat, the snake is shy in nature often seen basking near water bodies usually in morning hours
  - Venom — neuro toxic.

Saw scaled viper

- Zoological name: Echiscarinatus
- Common name: Carpet viper, phoorsa, afai
- Features:
  - Aggressive snake
  - Viviparous
  - Usually brown in color and grows up to 1.5 to 2 feet

- Head triangular with small scale. White “arrow mark” or “spear mark” may present on head
- Pupils are vertical
- Wavy white line (zig-zag pattern) may present on each flank
- Diamond shaped markings over back
- Belly scales are broad and cover entire width
- The scales of viper are serrated, saw like thus name saw scale viper
- Fangs are long, curved, hollow, channelised and hinged
- Venom — vasculo toxic and hemo toxic.
- (Can also be remembered as 5 V's; V= viper, V=viviparous, V=vertical pupil, V=v shaped head (triangular), V=vasculotoxic venom).

Russell's viper

- Zoological name: Viperarusselli
- Common name: Kander, ghonas
- Features:

- Head is large, flat and triangular with small scales. White V shaped mark present on head
- Pupils are vertical
- Large nostrils
- Body is stout and fatty with brown or yellowish color.
- Body scales are semi-elliptical
- Three rows of chained dark spots present on back
- Tail is narrow and short. Scales are divided into two rows
- Fang are long, curved, hollow, channelised and hinged
- When disturbed, makes a loud and hissing sound
- Venom — Vasculotoxic and hemotoxic.

Sea snakes

- Sea snakes are usually bluish, grayish or greenish in color. They have prominent nostrils and are situated on the top of snout
- Body is flat and belly scales are not broad
- Tail is flattened and paddle shaped
- Venom — myotoxic.

POISON APPARATUS

It is a modified salivary (Parotid) gland consisting of gland, duct and fangs.

Gland—It lies just below and behind the eyes, one on either side and is somewhat almond shaped.

Duct—This arises from the gland to carry the poisonous, venom from gland to the fangs.

Fangs—These are two in number, one on either side of upper jaw (could be more than 2 and kept in reserve), are hollow hypodermic needle like. It could be grooved as in cobra, kraitor could be tubular as in vipers.

SNAKE VENOM

Snake venom is the poisonous secretion (saliva), ejected from the poison apparatus (modified parotid gland) of a poisonous snake, during the act of biting. Snake venoms are usually clear and amber coloured fluid when fresh. On drying, its potency will be the same as that of liquid state. Snake venoms are poisonous only when injected by a needle or by bite of a snake. However, the venom is non-poisonous when taken orally.

Toxic Principles in Snake Venom

Venoms of the different species of poisonous snakes are usually a mixture of toxic substances or toxins, enzymes and toxalbumins in varying proportion.

Types of Snake Venoms

Basically snake venoms are of three types, namely *neurotoxic, haemotoxic* and *myotoxic* venoms.

Neurotoxic Venom

- *Origin*—Common in Elapidae snakes, e.g. krait, cobra, etc.
- *Action*—Acts like Curare, mainly on the *motor nerve cells* and results in *muscular paralysis*, the muscles are affected in following order:
  - Firstly—Muscles of the mouth
  - Secondly—Muscles of the throat
  - Finally—Muscles of respiration
- *Symptoms at bite site*—Local manifestations are least with neurotoxic venom snake bite.
- *Other symptoms*—Convulsions may be seen with Cobra venom (Krait venom produces only paralysis).

Haemotoxic Venom

- *Origin*—Common in Viperidae snakes, e.g. *Pit viper* (*Crotalidae*); Pit-less viper (*Russell's viper, Saw scaled viper*/*Phoorsa/Echis/ Echis Carinata*), and Bamboo snake (*Common green pit viper*).
- *Action*—Acts by cytolysis of endothelium of blood vessels, lysis of red cells and other tissue cells and coagulation disorders. All these can lead to:
  - Severe swelling with oozing of blood and spreading cellulitis at bite site. Blood from such patients fails to clot even on adding thrombin, because of very low level of fibrin.
  - Necrosis of renal tubules, and
  - Functional disturbances like convulsions, due to intracerebral haemorrhage.

Myotoxic Venom

- *Origin*—Common in hydrophidae or sea snakes
- *Action*—Produces generalized muscular pain, followed by:
  - Myoglobinuria within 3 to 5 hours.
  - Death usually occurs due to respiratory failure.

NONVENOMOUS SNAKES

Important Features

- They have no poison apparatus
- They possess 4 longitudinal rows of teeth in upper jaw and 2 rows in lower jaw
- Tail is not compressed
- Ventral shields are small/moderately large
- Head scales are usually larger and without any special features
- Fangs are short and solid
- These are not nocturnal
- The bite marks show more than two teeth markings. Among the poisonous species, snakes belonging to family *Elapidae* and *Viperidae* would be dealt with in more detail, as they are responsible for the most of the snakebite fatality in India and other parts of the globe.

VENOMOUS SNAKEBITE (OPHITOXAEMIA/ENVENOMATION)

Incidence

Snakebites are usually accidental. Rarely it can also be homicidal or suicidal.

Entry of Venom on Snake Bite

Venom is inoculated into the body. Snakes like cobra can inject/emit the venom by spitting.

Degree of Toxicity

Degree of toxicity depends on three factors:

- Toxic principle in the venom
- Quantity of venom injected
- Type of fang (see Fig. 33.12)
  - Channeled (viperine) fangs—complete transfer of venom
  - Grooved (elapid) fangs—less transfer of venom.

Signs and Symptomatology

Instantaneous death due to shock due to *fear* of snakebite is observed more often than the actual toxicity of the venom. Signs and symptoms of snakebite depend on the type of venom and are discussed under local effects (at the bite site) and its systemic effects.

1. Neurotoxic Venomous Snake Bite

- ***Local action***—Severe burning at bite site, rapid edema and inflammatory changes followed by oozing of serum.
- ***Systemic action***—Found within 15-30 minutes or 2 hours of

biting. Flow chart below presents the neuro toxic effects of colubrine snakebite. Giddiness, weakness, lethargy, muscle weakness etc, are followed by:

A spreading paralytic symptom
↓
Coma
↓
Bulbar or central paralysis
↓
Respiratory paralysis
↓
Death

However, recovery is *complete* from paralysis if patient survives, e.g. cobra bite.

2. Haemotoxic Venomous Snake Bite

- ***Local actions***—Severe pain at bite site, followed by swelling, ecchymosis, cellulitis and severe haemorrhage.
- ***Systemic action***—It is due to hemolytic effect on heart and blood vessels resulting in cardiovascular collapse and death. If the patient survives suppuration, sloughing with infection at the site of bite, haemorrhage from the mucosa of rectum, other natural orifice, etc. and gangrene of the parts involved can occur.

3. Myotoxic Venomous Snake Bite

- ***Local actions***—Minimal swelling and pain

➢ ***Systemic action***—Myalgia, muscle stiffness, myoglobinuria, renal tubular necrosis.

TREATMENT OF SNAKE BITE

Principle

- To allay the anxiety and fear
- Prevention of the spread of venom (first aid measures)
- Antivenom treatment
- General measures

Laboratory tests in snake bite cases
Blood - Complete blood count - Blood group and cross-matching - Liver function test - Kidney function test - Coagulation studies
Urine - For blood - For myoglobin ECG
Arterial blood gas analysis Chest X-rays

1. Allaying of the Anxiety and Fear

Convince and reassure the patient that all snakes are not poisonous, and even if poisonous need not be fully charged with poison, even if fully charged, the quantity that it has injected at the bite site, need not be lethal to kill.

2. Prevention of the Spread of Venom

The spread of venom is usually by lymphatic. Following first aid may be useful.

First Aid

- Reassurance of the victim.
- Do not tamper with the bite wound, except wipe with a damp cloth to remove the venom lying on the skin surface.
- *Immobilisation* of the bitten limb.
- Transport the patient to a medical facility immediately.
- Identify the snake if possible but not necessary. Better to take along the dead snake for identification. Be sure it is dead. Severed snake heads, both fresh and preserved, have inflicted severe and even fatal bites.
- Avoid potentially harmful traditional first aid measures such as cauterisation, incision, excision, or amputation of bite site; suction by mouth, vacuum pump, or syringe; combined incision and suction by 'Venomex' apparatus; injection or instillation of compounds such as potassium permanganate, phenol (carbolic soap), and trypsin; application of ice (cryotherapy) or electric shocks; herbal, folk, and ayurvedic remedies such as emetic plant products and parts of snake; multiple incisions and tattooing; insufflations of oily substances into the trachea; and application of irritants in conjunctivae, etc.
- Do not apply tourniquets, ligatures, or constricting bands unless

the snake is a neurotoxic envenomating, *i.e. snakes of species: elapids—Indian Cobras, Kraits; Australian elapids (genera: Acanthopis, Micropechis, Oxyuranus, Pseudechis, and Pseudonaja) and sea snakes,* However avoid this for long periods because local area will develop depletion of fresh blood. Similarly opening of the tourniquet will cause the rush of blood to the site and hence the rapid spread of venom, therefore, the anti venom treatment should be applied before releasing the tourniquet.

Dangers of Tourniquets, Compression Bandages and Other Occlusive Methods

- Ischaemia and gangrene
- Damage to superficial peripheral nerves, especially the lateral poplitial (common peronial) nerve at the neck of fibula.
- Increased fibrinolytic activity in the occluded limb.
- Congestion, swelling, and increased bleeding from the occluded limb.
- Shock on releasing a tight tourniquet.
- Intensification of local effects of venom in the occluded limb. A firmly applied crepe bandages exerting a compression of approximately 55 mm Hg may be used after bites by neuro toxic elapids and sea snakes and may left in place for several hours. Tourniquets tight enough to obliterate the arterial pulse are painful and must be released for about one minute after an hour. If reapplied they can finally be removed after two hours in a hospital or dispensary after an intravenous infusion of anti venom is begun and drugs and resuscitation equipment is ready for immediate use.

3. Antivenom Treatment

Do not use anti venom treatment routinely and indiscriminately because of reasons:

- All commercial anti venoms carry a risk of potentially serous serum reaction.
- Anti venom is not always necessary; many patients are bitten by non venomous snakes, and a large portion of those bitten by venomous snakes are not envenomated.
- Anti venoms have a range of specific and para specific neutralizing activity and are useless for venoms outside that range. Specific anti venoms are not available for treatment of envenomation by some species (e.g. *Bungarus candidus* in Southeast Asia)
- Anti venom is expensive, always in short supply, and has a limited shelf life.

Indications for Anti venom

Adverse Reaction to antivenin
Anaphylaxis
Delayed type of hypersensitivity reactions

A. Systemic envenomation

- Haemostatic disturbances; spontaneous systemic bleeding (*e.g. gums, epistaxis*), coagulopathy (*e.g. incoagulable blood, prolonged clotting time,* thrombocytopenia, etc.)
- Cardiovascular abnormalities: shock, hypo tension, abnormal electrocardiogram, arrhythmia, cardiac failure, pulmonary oedema.
- Neuro toxicity.
- Generalized rhabdomyolysis.
- Impaired consciousness of any cause.
- in patients with definite signs of *local envenomation*, the following

indicate significant systemic envenomation:

- Neutrophil leucocytosis
- Elevated creatine phoshokinase and amino transferases,
- Haemo concentration, uraemia, hyper creatininaemia, oliguria, hypoxaemia, acidosis and vomiting.

B. Severe local envenoming

Local swelling involving more than half of the bitten limb, or associated with extensive blistering or bruising, especially in patients bitten by species whose venoms are known to cause local neurosis (*e.g. viperidae, cobras*). Bites on digits carry a high risk of necrosis.

Contraindications to Antivenom

Adverse Reaction to antivenin
Anaphylaxis
Delayed type of hypersensitivity reactions

There is no absolute contraindication to anti venom in patients with life-threatening systemic envenomation. However, patients with an atopic history (asthma, hay fever, vernal conjunctivitis, eczema, and food and drug allergies) and those who had reaction to equine antiserum on previous occasions have an increased risk of severe reactions.

In case of pretreatment with subcutaneous adrenaline and intravenous antihistamine and corticosteroids may prevent ordiminish the reaction.

Rapid desensitisation is not recommended.

Timing of Anti venom

Give as soon as signs of systemic or severe local envenomationare evidenced. Average time between bite and death:

- *Cobras* – 8 hours (12 minutes to 120 minutes)
- *Bungarus caeruleus* – 18 hours (3 to 63 hours)
- *Viperarusselli*– 3 days (5 minutes to 264 hours)
- *Echiscarinatus*– 5 days (25 hours to 41 days)

It is almost never too late to try antivenom treatment: it has been effective up to 2 days after sea snake bites and 10 days or more after *Echiscarinatus*bites.

Anti venom Specificity

Optimal treatment consists of *monospecific / monovalentantivenom.* If no dead snake is brought in for identification, polyspecific*/polyvalent anti venom* may be useful.

Administration

Preferably give anti venom intravenously: 5 ml/minute, or dilutein isotonic fluid, infused over 30 to 60 minutes. Dress venipuncture sites with a pressure bandage. Injection of anti venom into the fang marks is probably ineffective and painful.

Dosage

Children must be given the same dose of anti venom as adults.

Response to Anti venom:

Time to Possible Response

- Neurotoxicity – slowly
- Cardiovascular effects –(hypotension, bradycardia) – 10 to 20 minutes
- Stopping spontaneous systemic bleeding – 15 to 30 minutes
- Blood coagulability restored – 1 to 6 hours
- Repeat the initial dose of anti venom if severe cardio vascularor neurotoxic symptoms persist for more than 30 minutes and in coagulable

blood persists for more than 6 hours after first dose.

Antivenom Reactions

1. Early reactions: 10 to 60 minutes after starting intravenous anti venom, cough, tachycardia, itching (especially of scalp),urticaria, fever, palpitation, nausea, vomiting, headache, etc. may develop. Over 5 per cent with early reactions develop manifestations of severe systemic anaphylaxis: hypotension, bronchospasm, angioedema, etc. and a few may die.

Treatment: Adrenaline (epinephrine) subcutaneously: 0.5 to1.0 ml 0.1 per cent (1 in 1000) for adults; 0.01 mg/kg for children. In severe cases, give same dose by intra muscularinjections, or during cardiac resuscitation, by slow IV or even intracardiac injection. Follow with an antihistamine, e.g. chlopheniramine maleate – 10 mg (adults); 0.2 mg/kg(children).

2. Pyrogenic reactions: develop in 1 to 2 hours after treatment and include—chills, cutaneous vasoconstriction, goose flesh, shivering, drop in temperature, sweating, vomiting, diarrhoea, etc.

Treatment: Lie flat; reduce temperature by fanning, tepidsponging, hypothermia blankets, or antipyretic drugs such as acetaminophen (5 mg/kg by mouth, suppository, or vianasogastric tube).

3. Late reactions (serum sickness type): About 7 days after treatment (5 to 24 hours).

Treatment: Antihistamines may restrain a milder attack. Steroids may be useful in more severe cases.

Ancillary Treatment

Local Envenomation

➢ *Secondary infection:* Prevention with penicillin or erythromycin and booster dose of tetanus toxoid.

- Clean wound with antiseptic.
- Bullae can be aspirated to dryness with a fine sterile needle.
- Nurse limbs in most comfortable position.
- Examine the wound frequently for evidence of necrosis.

Polyspecific/Polyvalent Antivenom

- Haffkin's *Institute, Mumbai and Central Research Institute Kasauli, Himachal Pradesh, King's Institute, Chennai, Serum Institute, Pune* prepares this. It is available as a *lyophilized powder* in an ampoule, with potency for nearly 10 years.
- It can neutralize the venom of *cobra*, *common krait*, *Russel's viper* and *Echiscarinata*.
- It is effective when given within *four* hours of biting.
- It is to be dissolved in distilled water or normal saline before use.
- Use only if the solution prepared is *clear*, if it is opaque, it is considered as not potent.

Mode of Administration

Always perform *serum sensitivity test* (*test dose*) before giving it.

- ***The skin test procedure***—Inject 0.02 to 0.03 ml of the anti venin in 1:10 dilution, intra dermally. If there is a hypersensitivity reaction (urticarial wheal with erythema within 15 minutes) the patient should be *desensitized*.
- ***The desensitisation procedure***—Inject 0.1, 0.2 and 0.5 ml of the anti venin in 1:100 dilutions at an interval of 15 minutes. Subsequently a 1:10 dilution is given in the same manner, followed by undiluted antivenin. If no severe reaction occurs, the usual dose is given intravenously.
- ***According to another view*** —Skin testing is *not* necessary, but

adrenaline should be injected subcutaneously as pre medication with a dose of a systemic corticosteroid.

Dose Schedule

Inject 40 to 60 ml as *follows:*

- One third of total dose is given subcutaneously at bite site
- Next one-third is given intramuscularly, and
- Final one-third is given intravenously.
- This schedule can be repeated every sixth hour till the symptoms disappears.
- *According to another view*—antivenom serum is to be given intravenously at the rate of 15 drops per minute. After finishing the first dose, which is completed in an hour, one may increase the rate. If the patient's response is not favourable, further infusion may be given.

General guidelines of specific serumtreatment in the hospital
1. Installation of the intravenous drip infusion solution of physiological saline with or without vasopressor drugs untilhypotension is overcome.
2. Sensitivity test to horse serum. 3. Alleviation of pain with small amount of pethidine but not morphine.
4. Intravenous administration of polyvalent antivenom serum in adequate amount. 5. Infiltration of the site of bite with a small amount of the antiserum 6. Tetanus toxoid if required.
7. Monitoring of blood pressure, blood count, coagulation, and observation of oedema. 8. Blood transfusion, if anaemia develops. 9. Surgery if necrotic area appears. 10. For detailed treatment, the hospital doctors

may decide on the individual case to case basis.

Note: The specific treatment, may vary from patient to patient, and hence a medical doctor should be consulted in all cases of snakebites

Specific Antivenom Serum

- In India, *only polyvalent anti venin* is available, which is effective against common cobra, common krait, Russell's viper and sea-scaled viper.
- Is preferred when identity of biting snake is known
- Dosage and schedule of administration is same as for polyvalent antisnake venom serum.
- *Adrenaline* may be useful in paralytical cases.
- *Artificial respiration* is often required
- *Cortisone* 50 to 100 mg intramuscularly can combat the shock
- *Antihistamines* may also be helpful

General Measures

Symptomatic measures as required, but, avoid *alcohol* or *morphine*, for these can increase the rate of absorption of venom. However, pethidine may be used instead of morphine.

Postmortem Findings

Bite mark—Two in number, 1 cm deep for colubrine bite and2.5 cm deep for viperine bite. At the bite site there may be alittle oedema, cellulitis, bleeding, etc for viperine bites.

- For colubrine bite deaths, changes will be that of *asphyxia*.
- For viperine bite deaths, changes will be that of haemorrhage in lungs, pleura and pericardium. Kidneys will show renal tubular necrosis, desquamation and cloudy swelling.

Medicolegal Aspects

Snakebites are usually accidental, in warm and moist climates. Rarely bites could be suicidal as well (Cleopetra, Queen of Egypt, committed suicide by snake bite). However, homicidal cases though rare, where death is alleged to be from snake bite, the presence of snake venom has to bee stablished.

Cattle poisoning: To obtain the hide from cattle, the skin workers in India adopted a very peculiar method, wherein, a cobra and a ripe banana were placed in an earthen pot and the pot was warmed up to enrage the cobra which would then bite the banana. The cobra was then released; banana was then smeared on a rag and thrust into the rectum of the animal by a bamboo stick, leading to the death of the animal.

To identify the venom at the bite site the following steps may be considered:

– This is done by injecting the washings into the fowls or rabbits with specific antivenom sera injected previously

– Detection of cholinesterase activity- *colubrine* snake biteis confirmed

– Detection of thromboplastin activity- *viperine* snake biteis confirmed.

INSECTS

Cantharides *(Spanish Fly, Blister Beetle, Lytta)*

It is a winged insect, which has a body of: length 2 cm and breadth 0.6 cm and greenish black colour with shiny wings of same colour. The insect as such or the powder of dried body has the toxic (active) principle.

- ***Active principle:*** Cantharidin.
- ***Route of absorption:*** Skin and all other muscosa.

➢ **Signs and Symptoms**

➢ Within 2 to 3 hours coming in contact with the skin, the poison produces burning pain, red-ness and vesication. Taken orally, symptoms manifest within 30 minutes to 2 hours.

➢ ***Fatal dose***—15 to 30 mg of cantharidin 1.5 gm of powdered cantharides.

➢ ***Toxicity rating***—For cantharidin

➢ ***Treatment*** Stomach wash, demulcents, symptomatic.

➢ **Postmortem Findings**

• Mouth, stomach and intestines may show inflammation and vesication. Particles of insect may be found in the stomach contents.

• Heart, lungs, kidneys are also found to be inflamed and haemorrhagic.

➢ **Medicolegal Importance**

• Used often as an aphrodisiac

• Poisoning is reported very rarely (accidental/homicidal)

• Used also as stimulant for scalp hair growth (hair oil).

CENTIPEDES

Centipedes belong to Myriapoda (under class Arthropods) and are organic animal irritants. They have a long segmented dark to brownish black colored body with a pairs of legs in each segment.

Signs and Symptoms

Usually centipedes can inflict painful bites with erythema, oedema, and local lymphangitis.

Treatment is by washing the bite site with soap and water and administering analgesics.

SCORPIONS

Scorpion is a poisonous insect (Arthropod) with a crab like body with eight legs and a segmented tail having a bulbous expansion and a sting in the last segment which has a clear, colourless *venom* (toxalbumin) having two components, a *hemolytic* and a *neurotoxic* fractions. Fatality is rare as dose in sting is not lethal.

Venom

Scorpion venom is clear, color less fluid and contains following toxic components

- Phospholipase
- Acetyl cholinesterase
- Hyaluronidase
- Serotonin
- Neurotoxin

Mechanism of action

- Scorpion venom delays the inactivation of sodium channels of autonomic nervous system resulting in autonomic storm
- a-Receptor stimulation plays an important role in the pathogenesis of pulmonary edema
- Scorpion venom is powerful arrhythmogenic agent.

Signs and Symptoms

- Haemolytic factors can mimic viperine snakebite. Diagnosis is by locating only one deep punctured wound (snake bite— fang marks are always two in such wound) with red surrounding area (inflammation), oedema, severe burning pain, etc.
- Neurotoxic factor can mimic strychnine poisoning. Victim presents with nausea, vomiting, restlessness, fever followed by

convulsions, paralysis, coma and death (due to respiratory paralysis).

Treatment

- Measures to reduce the rate of absorption of the venom by:
  - Tourniquet above the level of stinging
  - Ice packing, incision and suction (first aid)
  - Wash with solution of ammonia.
- Local anaesthetics can also be helpful to reduce the pain
- Intravenous administration of calcium gluconate may reduce swelling.
- Giving barbiturate can alleviate anxiety
- Atropine may be given to reduce the pulmonary edema (do not use morphine).

Autopsy findings

- Local part swollen, inflamed, sting may be present, surrounding tissue may show hemorrhagic infiltration
- The sting sites are usually at peripheral sites or part of body such as toes, fingers, palms, soles, etc. However, occasionally, other part may be involved such as back, shoulder, etc.
- Systemic examination reveals pulmonary edema, pulmonary hemorrhages, gastrointestinal tract hemorrhage, intracerebral hemorrhage, signs of consumption coagulopathy, myocarditis.

Medicolegal importance

- Accidental envenomation occurs and fatality is more in children.

BEES, WASPS, HORNETS, ANT

Venom of bees, wasps, hornets, ants, etc. is a complex mixture of biomedical compounds ranging from simple amines to complicated proteins or enzymes.

Action

Action is usually local. It may be rarely fatal if venom is histamine(especially when bites on neck, face, etc.). However, it can result in laryngeal edema leading to asphyxia and deat, when not treated immediately. When there are multiple stings, it can lead to severe systemic reactions, resulting in gastrointestinal disturbances, shock, unconsciousness and death.

Treatment

- Measures to reduce the rate of absorption of venom by:
  - Tourniquet above the level of stinging or incision and suction
  - Adrenaline is also useful.
- Apply tincture iodine/antihistaminic (to reduce inflammation)
- Adrenocorticotrophic hormone (ACTH) 25 mg in 1000 ml normal saline given as an intravenous drip can help in prevention of severe allergic reactions
- Calcium gluconate given intravenously can reduce edema/rash.

MECHANICAL IRRITANTS

Mechanical irritants, *per se*, are not poisons and do not causetoxic effects but cause local irritation at the site of application.For example, glass powder can cause irritation of gastrointestinalmucosa if ingested. These agents are consideredas "unwholesome drugs" or other drugs of the section 328of IPC.

Examples are:

- Powdered glass/glass particles
- Diamond powder
- Needles/metal pins
- Chopped animal hairs
- Vegetable hairs
- Stone pieces
- Nails

Clinical Features

➢ Powdered glass, diamond powder, needles, etc. maycause pain in abdomen, nausea and vomiting, may injuretissue and causes bleeding. If bleeding is considerableand acute, death may occur due to hemorrhagic shock.If bleeding is gradual and concealed, e.g.malena mayinduce anemia, weakness, general debility, etc.

➢ Similarly death may occur if the agents cause perforationof stomach or intestine

➢ Pieces of chopped hairs cause nausea, vomiting and irritation.GIT mucosa may be inflamed.

Fatal dose and fatal period: Uncertain

Complication

- Bowel/esophagus perforation
- Mechanical intestinal obstruction
- GIT hemorrhage
- Perforation peritonitis.

Management

- Bulky food and then purgatives to pass the irritants in
- stools
- Ice pieces to reduce thirst
- Analgesics to relieve pain.

Autopsy Findings

- Erosions may be noted in mouth, pharynx, esophagus,
- stomach and intestine
- Fragments of glass, stone, hairs, etc. may be found in
- GIT adhered to mucosa
- Mucosa of GIT may be inflamed.

Medicolegal Importance

- Accidental ingestion may occur with jam, jelly or food, etc.
- Show-men may swallow glass particles while showingthe show
- These agents may be used with an evil intention to causeill health and death
- Occasionally used as cattle poisons
- These agents are considered as "unwholesome drugs"under section 328 of IPC
- Children having access to these substances may accidentallyingest them or may inhale in respiratory tractcausing respiratory obstruction.

PESTICIDES

Pesticides are the compounds used to kill pests. Pests mayinclude insect, rodent, fungi etc. Pesticides are classified as:

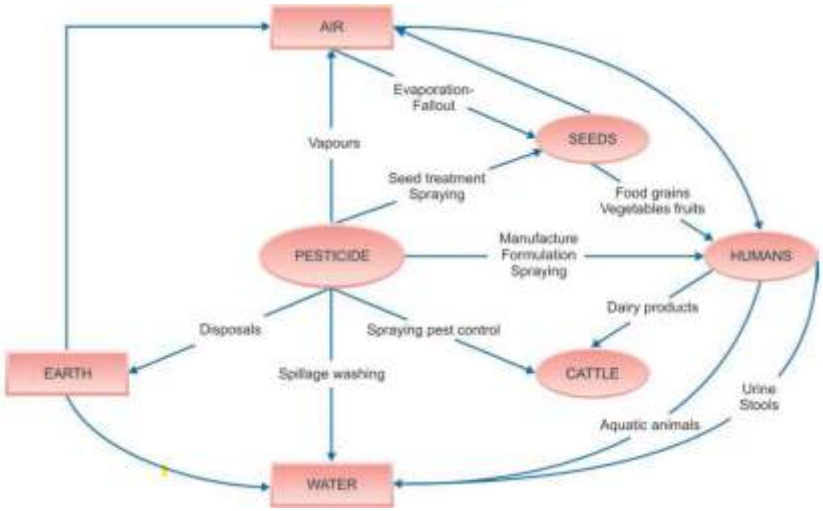
1. Insecticides: these are the compounds used to kill orrepel insects and related species. Examples are — organophosphates,carbamate, organochlorine, pyrethroids.

2. Rodenticide: these are the compound used to kill rodentlike rat, mice, mole etc. Examples are — zinc phosphide,barium carbonate, strychnine, warfarin etc.

3. Herbicide: these are the compounds used to kill weed.Examples are — acrolein, glyphosate, paraquat etc.

4. Fungicide: these are the compounds used to kill fungi andmoulds. Examples are — thiocarbamate, sodium azide.

5. Miscellaneous compounds include lead, copper, mercury,nicotine etc.



Pesticide cycle in environment

ORGANOPHOSPHORUS COMPOUNDS

Organophosphate poisoning is the most common poisoning inIndia followed by aluminium phosphide.Organophosphoruscompounds are available as dust powder or liquid.

Organophosphorus compounds are classified as:

1. Alkyl compounds — such as tetraethyl pyrophosphate(TEPP), hexa ethyl tetraphosphate (HETP), octa methylpyrophosphate (OMPA), malathion etc.

2. Aryl compounds — such as parathion, chlorothion, diazinon(Tik-20), paraoxon etc.

Absorption, Metabolism and Excretion

Organophosphorus compounds are absorbed by any route— skin, conjunctiva, inhalation, oral or by direct injection.

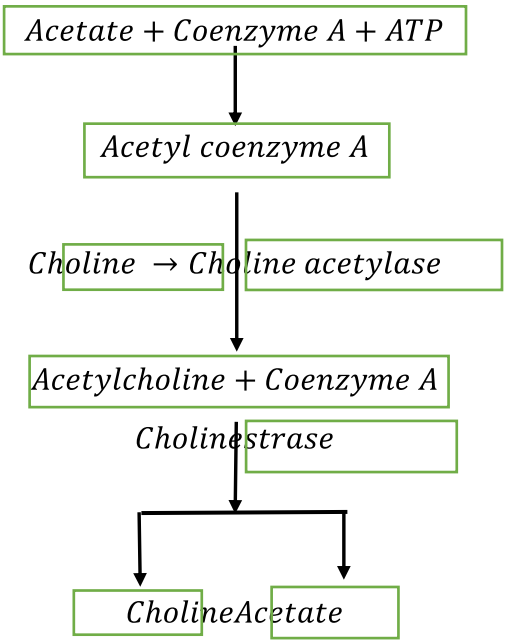
Some compounds such as parathion are stored in body fatand are released slowly in the circulation thus prolongingthe duration of toxic action.

Parathion is first metabolized to paraoxon, which is theactive toxic agent and then to paranitrophenol that isexcreted into urine. Malathion is metabolized in liver byesterases and part of this metabolized product is excretedin urine as phosphate.

Mechanism of Action

Organophosphorus compounds are inhibitors of acetylcholinesterase. Acetyl cholinesterase is required to hydrolyseacetylcholine to choline and acetic acid. As aresult, there is accumulation of acetylcholine with continuedstimulation of local receptors and eventual paralysis of nerveor muscle. Organophosphate intoxication leads tocharacteristic end-plate

abnormalities that reflect the degreeof AchE inhibition and increase in Ach concentration at theneuromuscular junction.



Fatal Dose

- Malathion about 60 gm4
- TEPP 100 mg
- OMPA 175 mg
- Parathion 175 mg
- HETP 350 mg
- Diazinon 1 gm

Fatal period

- 24 hours

Clinical Features

Organophosphate insecticide poisoning in human canproduce:

- Acute poisoning — due to acute peripheral and central cholinergic block
- An intermediate syndrome with weakness
- A delayed distal polyneuropathy

Acute Poisoning

A. Muscarinic effects

Due to muscarinic like action, following clinical features

are observed

- Bronchial tree — cough, increased secretions, bronchoconstriction, wheezing, dyspnea, pulmonary edema.
- Gastrointestinal — nausea, vomiting, abdominal cramp, diarrhea.
- Sweat glands — increased sweating.
- Salivary glands — increased salivation.
- Lacrimal glands — increased lacrimation.
  - **Chromodacryorrhea**(shedding of red tears) due to accumulation of porphyrin in the lachrymal glands is seen very rarely.
- Eyes — miosis, blurring of vision or dimness of vision. Miosis develops due to the inhibition of cholinesterase and marked parasympathomimetic stimulation of iris. However, dilatation of pupil in organophosphate intoxication have been recorded, therefore, it is essential not to rely only on pupillary size as diagnostic criteria for organophosphate compound poisoning.
- Heart — slow pulse, hypotension.
- Urinary bladder — frequency of micturation, urinary

incontinence.

{Muscarinic manifestation can be remembered with the mnemonic **"DUMBELS"** D = diarrhea, U = urination, M = miosis, B = Bronchospasm, bradycardia, E = emesis (vomiting), L = lacrimation, S = salivation}

B. Nicotinic effects

The nicotinic effects are as follows:

1. Striated muscles — easy fatigue, weakness, musculartwitching, fasciculation, cramps

2. Sympathetic ganglia — pallor, occasional elevationof blood pressure (hypertension), tachycardia

3. Increased adrenal medulla activity

(Nicotinic manifestation can be remembered with the mnemonic "**MATCH"** M = muscle weakness and fasciculation, A = adrenal medulla activity increase, T = tachycardia, C = cramps in muscle, H = hypertension).

C. CNS effects

The CNS effects are:

- Irritability
- Apprehension
- Restlessness
- Fine fibrillary tremors of hands, eyelids, face or tongue

- Muscular weakness
- Convulsions — the convulsions may be tonic (limbs stretched and rigid) or may be clonic (rapid repetitive movement). Clonic convulsions are more common.
- Mental confusion progressing to stupor to coma
- Depression of respiratory and circulatory centers.

D. Other features

- Toxic myocarditis had been reported.
- Pancreatitis may be noted. The parasympathetic stimulation of the pancreas with Ach, pilocarpine or vagal stimulation causes augmentation of the secretory flow
- and increased intraductal pressure. However, the exact cause of Pancreatitis is unknown.
- Organophosphorus compound produce metabolic acidosis by respiratory depression, bronchoconstriction, pulmonary edema, CNS depression and lactacidosis.

Causes of Death

- Respiratory failure
- Cerebral hypoxia
- Hyperthermia
- Hepatic failure
- Renal failure

Intermediate Syndrome

- Intermediate syndrome is a neurotoxic effect that appearsafter acute cholinergic crisis but before the expected onset of delayed neuropathy.
- The cardinal feature of the syndrome is muscular weakness, affecting predominantly the proximal limb muscles and the neck flexors. Cranial nerve palsies are common. (The intermediate syndrome predominantly affect muscles
- innervated by the cranial nerves — neck flexors, proximal muscles of the limb and the muscles of respiration).
- This syndrome carries a risk of death because of the associated respiratory depression.
- The muscle weakness had an acute onset, noticed within 24 to 96 hours after poisoning.

Difference between intermediatesyndrome and delayed polyneuropathy

Variable	*Intermediate syndrome*	*Delayed polyneuropathy*
Time of onset after poisoning	1 to 4 days	2 to 3 weeks
Site of weakness - Limb muscles - Neck muscles - Cranial nerves - Respiratory muscles	 Proximal Present Present Present	 Distal Absent Absent Absent
Electromyogram	Tetanic fade	Denervation
Recovery from time of onset	4 to 18 days	6 to 12 months
Organophosphate compound commonly involved	Fenthion, dimethoate, monocrotophos	Methamidophos, trichlorphos, leptophos

Delayed Polyneuropathy

Delayed polyneuropathy appears 2 to 3 weeks afterpoisoning

The delayed polyneuropathy is due to inhibition ofenzyme neurotoxic esterase with nerve demyelination.

In delayed polyneuropathy, the paralysis is usually limitedto the distal muscles of the limbs; cranial nerves andrespiratory muscles are spared.

The disorder is characterized by flaccid weakness andatrophy of distal limb muscles or spasticity and ataxia.

Diagnosis

1. Cholinesterase level

- Depression of RBC cholinesterase level more than 50 percent of normal indicates organophosphate poisoning. The decrease is due to binding by phosphate group of pesticide. It is better parameter than plasma cholinesterase.
- Depression of plasma (serum) cholinesterase activity more than 50 percent of normal indicates Organophosphate poisoning. This test is not specific as plasma cholinesterase activity is also depressed in cirrhosis of liver, neoplasia, malnutrition, septicemia due to burns, obstructive jaundice.

2. Colorimetric method

- 1 ml sample urine is taken and 1 ml of NBB {45% in acetone 4-(nitrobenzyl) pyridine} added and mixed for 30 seconds in vortex mixer. The mixture is heated at 100° C for 20 minutes. Organophosphate insecticideshows a characteristic purplish bluecolor thatcan be read using spectrophotometer.

3. P-nitrophenol test

4. Paper chromatography

5. Thin layer chromatography (TLC)

6. Gas chromatography (GC)

7. Gas chromatography-mass spectrometry (GC-MS)

8. High performance liquid chromatography (HPLC)

9. ECG may show right axis deviation, ST segment depressionand T wave inversion.

Management

Principles of treatment consist of:

- Stabilization of patient
- Decontamination
- Antidote administration
- Supportive measures
- Nursing care

Decontamination

- Skin — the affected part should be washed thoroughly with copious water
- Ocular — copious eye irrigation with normal saline or tap water
- Ingestion — gastric lavage and administration of activated charcoal.

Antidote Administration

- Atropine is competitive antagonist of acetylcholine and blocks muscarinic manifestations of Organo phosphate. It does not reverse peripheral muscular paralysis, which is nicotinic action. The atropine

should be given2 mg intravenous promptly with dose repeated every10 minutes till pupil dilates (up to 200 mg has been administered in a day).Some authorities recommend administration of atropine until bronchial and other secretions have dried. According to them pupil size and heart rate cannot be used as end-points.Continuedtreatment with maintenance doses may be required for1 to 2 weeks.

- Oximes are used as they helps to regenerate acetylcholine sterase at muscarinic, nicotinic and CNS sites. Pralidoxime (2-PAM) is given intravenously as 500-mg/20 ml infusion in a dose of 1 to 2 gm (children 20to 40 mg/kg).

Supportive Measures

- Oxygen administration, ventilator assistance
- Maintain vital parameters, hydration, urine output
- Convulsions should be controlled with judicious use of diazepam.

Avoid Giving

- Other acetylcholine sterase inhibitors such as Physostigmine, endorphonium
- Succinylcholine for rapid in tubation.

Autopsy Findings

- Insecticide like smell (sometimes garlicky or kerosene like)
- Froth at mouth and nostrils
- Cyanosis
- Constricted pupils
- Stomach contents have insecticide like smell. Muco sastained with

compound color, congested and eroded

- Congestion of organs
- Pulmonary edema
- Cerebral edema
- Features of toxic myocarditis had also been reported. Microscopic examination of heart reveals dilatation of pericardial blood vessels with hemorrhages in the surrounding tissues, interstitial edema of myocardium, inflammatory cells, hemosiderin-laden macrophages and fatty infiltration of the myocardium.

Medicolegal Importance

- Accidental poisoning may occur in farmers while spraying in the fields or opening the lid of the containers.
- Suicidal poisoning is common with this insecticide.
- Homicidal is rare as it is difficult to mask the smell of insecticide.

ORGANOCHLORINES

Organochlorine insecticides are chlorinated hydro carbons and are divided into four types as:

- DDT (dichlorodiphenyl-trichloroethane) and analogues
- Benzene hexachloride group — e.g. BHC, lindane
- Cyclodines and related compounds — e.g. endrin, aldrin, dieldrin, endosulfan, sobenzan
- Toxaphene and related compounds — e.g. toxaphene.

Availability

These compounds are available as

- Dusting powder
- Emulsion
- Granules
- Solutions.

Fatal dose

- DDT 15 to 30 gm
- Lindane 15-30 gm
- Aldrin, endrin, dieldrin 2 to 6 gm.

Absorption, Metabolism and Excretion

Organo chloride compounds are absorbed through skin, inhalation and through gastrointestinal tract. Most of the compounds are metabolized slowly in the body and remains in tissues, especially in fatty tissues for prolonged duration.

These compounds are metabolized in liver and are excreted in urine, feces and milk.

Mechanism of Action

- Organochlorines affect nerve impulse transmission by altering membrane Na+ and K+ flux, resulting in CNS hyperexcit ability.
- Organochlorines produces myocardial irritability thus predisposing to cardiac arrhythmias
- DDT and related compounds affect the sodium channel and

sodium conductance across the neuronal membrane, especially of axons

➢ Cyclodine and lindane appear to inhibit the GAB Amediated chloride channels in the CNS.

Clinical Features

➢ ***Acute poisoning***

- GIT — nausea, vomiting, diarrhea hyperaesthesia or paresthesia of mouth and face
- CNS — headache, vertigo, myoclonus, mydriasis, weakness, agitation, confusion, convulsions, coma.
- Respiratory system — cough, wheezing, if aspiration or inhalation occurs
- Renal failure
- Hepatitis
- Dermatitis.

➢ ***Chronic Poisoning***

- Exposure of these compounds for prolonged duration may result in cumulative toxicity characterized by anorexia, weight loss, weakness, tremors, opsoclonus, ataxia, pseudotumor cerebri, abnormal mental changes, oligospermia, thrombocytopenic purpura. Lindane and BHC have been linked to aplastic anemia.

Management

➢ Skin — the affected part should be washed thoroughly with copious water

➢ Ocular — copious eye irrigation with normal saline or tap water

- Ingestion — gastric lavage and administration of activated charcoal
- Oxygen administration, ventilator assistance
- Maintain vital parameters, hydration, urine output
- Convulsions should be controlled with judicious use of diazepam or lorazepam.
- Hyperthermia should be managed in usual way
- Arrhythmias can be managed with lidocaine.

Avoid Giving

- Epinephrine — may exacerbate ventricular arrhythmias
- Atropine
- Oil based fluid/food/cathartics

Autopsy Findings

- Insecticide like smell
- Froth at mouth and nostrils
- Cyanosis
- Congestion of organs
- Pulmonary edema
- Cerebral edema.

Medicolegal Importance

- Accidental poisoning may occur in farmers while spraying in the fields or opening the lid of the containers.
- Suicidal poisoning is also common with this insecticide.

- Homicidal is rare as it is difficult to mask the smell of insecticide.

CARBAMATE

Carbamates are popular insecticides and include aldicarb, propoxur (Baygon), carbaryl, carbofuran, methomyl, triallate, bendiocarb etc.

Mechanism of Action

Carbamate causes reversible inhibition of acetylcholine sterase due to which there is accumulation of acetylcholine at muscarinic and nicotinic receptors and in the CNS.

Fatal Dose

- Extremely toxic or highly toxic — carbaryl, carbfuran, methomyl, propoxur
- Moderately toxic or slightly toxic — aldicarb, triallate.

Clinical Features

Clinical features of Carbamate poisoning are same that of Organo phosphate poisoning with following difference:

- Carbamate causes reversible inhibition of acetylcholine sterase, therefore the signs and symptoms are less severe than Organo phosphate poisoning
- Carbamate toxicity is short lived and it hydrolyse spontaneously from the site
- It does not penetrate effectively in CNS so it produce little or no CNS toxicity.

Management

Decontamination

- Skin — the affected part should be washed thoroughly with copious water
- Ocular — copious eye irrigation with normal saline or tap water
- Ingestion — gastric lavage and administration of activated charcoal.

Antidote Administration

- Atropine is competitive antagonist of acetylcholine and blocks muscarinic manifestations. The atropine should be given 2 mg intravenous promptly with dose repeated every 10 minutes till signs of at ropinization are evident.
- Oximes are ineffective in Carbamate poisoning and are not recommended.

Supportive Measures

- Oxygen administration, ventilator assistance
- Maintain vital parameters, hydration, urine output.

Autopsy Findings

- Insecticide like smell
- Froth at mouth and nostrils
- Cyanosis
- Constricted pupils

- Congestion of organs
- Pulmonary edema
- Cerebral edema.

Medicolegal Importance

- Accidental poisoning may occur.
- Suicidal poisoning is also common with this insecticide.
- Homicidal is rare as it is difficult to mask the smell of insecticide.

PYRETHRUM, PYRETHRINS AND PYRETHROIDS

- Pyrethrum is extract of the chrysanthemum flower. Pyrethrum contains six active components labelled pyrethrins.
- Pyrethroids are synthetic derivatives of pyrethrins.
- These compounds are commonly used as insect and mosquito repellants.
- Examples — allethrin, D-allethrin, cypermethrin, permethrin etc.

Mechanism of Action

- Pyrethroids prolong the inactivation of the sodium channel by binding to it in the open state.
- These compounds quickly inactivate insects but mammals are relatively resistant to them. However, in most of the cases, toxicity with these agents occurs because of the allergic reactions to these compounds.

Fatal Dose

Pyrethrum 1 gm/kg

Clinical Features

- Dermal exposure causes erythema, dermatitis, blister formation
- Ocular exposure causes irritation, lacrimation
- Inhalation causes rhinorrhea, sore throat, wheezing, cough, dyspnea
- Ingestion causes nausea, vomiting, paresthesia, vertigo, fasciculation, hyperthermia, altered mental status, convulsions, pulmonary edema and coma.

Management

- ***Decontamination***
  - Skin — the affected part should be washed thoroughly with copious water
  - Ocular — copious eye irrigation with normal saline or tap water.

- ***Systemic Poisoning***
  - Ingestion — gastric lavage and administration of activated charcoal
  - Fatty substance should be avoided as they promotes the absorption through GIT
  - Allergic reaction should be treated with epinephrine and antihistamines
  - Broncho spasm should be treated with appropriate bronchodilators
  - Convulsions should be controlled with judicious use of diazepam
  - Oxygen administration, ventilator assistance.

Autopsy Findings

- Insect repellant like smell
- Froth at mouth and nostrils
- Cyanosis
- Congestion of organs
- Pulmonary edema
- Cerebral edema.

Medicolegal Importance

- Accidental poisoning may occur.
- Suicidal poisoning is rare.

PARAQUAT

These are popular herbicides. They are spread on unwanted weeds and other vegetations. Paraquat and diquat belongs to dipyridyl group.

Fatal Dose

4 mg/kg

Clinical Features

Paraquat causes corrosion to mucosa of mouth, esophagus and gastrointestinal tract. It causes pain in mouth and in abdomen. There is diarrhea, vomiting, dysphagia andaphonia. Patient develops hepatic failure and renal failure. There may be metabolic acidosis with hyperglycemia. Lungs show pulmonary edema and after 4 to 5 days may show pulmonary fibrosis with progressive respiratory failure.

Management

- Gastric lavage with activated charcoal28 may be beneficial if performed within one hour of ingestion of poison
- Pain should be controlled by giving ice-cold fluid and parental analgesics
- Hemo dialysis or Hemo perfusion is said to be beneficial
- Supportive measures include maintenance of airway, circulation, hydration, urine output and vital parameters.

Autopsy Findings

- Corrosion around lips, mouth, esophagus and erosion in GIT
- Lungs may be edematous or may appear stiffened with evidence of hemorrhage and pulmonary fibrosis
- Liver — signs of hepatic failure and kidneys show acute tubular necrosis
- Other organs congested.

Medicolegal Importance

- Accidental poisoning may occur.
- Suicidal poisoning may occur.

ALUMINIUM PHOSPHIDE

Aluminium phosphide is used as a grain preservative. In northern part of India, it is the leading cause for death due to poisoning.

- ***Availability***

  - Aluminium phosphide is available in grayish green tablets of3 gm each. Each tablet release 1 gm of phosphine.The tablets are available in market with various trade namessuch as — celphos, alphos, sulphas etc.

- ***Fatal Dose***

- 3 gm

➢ ***Fatal Period***

- 12 to 36 hours

Mechanism of Action

Aluminium phosphide liberates phosphine when it comes in contact with air and moisture. It reacts with acidic media(HCL) of stomach and release phosphine gas, which is rapidly absorbed from gastrointestinal tract by simple diffusion. Phosphine is a protoplasmic poison interfering with enzymesand protein synthesis. In animal studies, phosphine has been shown to cause non-competitive inhibition of cytochromeoxidase of myocardial mitochondria.

Clinical Features

- Metallic taste
- Vomiting
- Thirst
- Burning sensation
- Pain in abdomen
- Diarrhea
- Persistent hypotension with shock
- Dyspnea
- Cough
- Tachycardia
- Tachpnea
- Bleeding diathesis

- Restlessness
- Altered sensorium
- Coma
- Respiratory distress.

Diagnosis

- Garlicky smell
- Altered liver function tests with raised transaminase levels
- Increased PT and PTT
- ABG shows metabolic acidosis
- ECG — sinus tachycardia, ST depression in leads II and III.
- Silver nitrate test – the patient is asked to breathe through a piece of filter paper impregnated with 0.1 Nsilver nitrate solution for 5 to 10 minutes. If filter paper becomes black, it suggests presence of phosphine. The blackening is imparted because phosphine reduces silvernitrate to silver.

Management

- Secure airway, oxygen, ventilator assistance
- Manage shock with intravenous fluid and inotropic support
- Manage metabolic acidosis with sodium bicarbonate
- Magnesium sulfate administration remains controversial. It is said that administration of magnesium sulfate is beneficial for management of cardiac arrhythmias
- Gastric lavage is contraindicated since administration of water will release phosphine from the available aluminium phosphide in the

stomach.

Autopsy Findings

- Typical garlicky odor
- Congested organs
- Bright fluid blood
- Pleural effusion
- Pulmonary edema
- Toxic myocarditis
- Bleeding diathesis
- Gastric mucosa shows shedding
- Kidney shows acute tubular necrosis
- Liver shows fatty change, congestion, edema, in flammatory infiltrate in portal tract and centrizonal necrosis.

Medicolegal Importance

- Suicidal poisoning is common with this agent, especially in the northern part of India.
- Rarely accidental poisoning may occur to workers working in grain elevators, wear houses and grain freighter etc

Cardiac poisons act mainly on the heart, either directly or through the nerves. Though there may be several cardiac poisonousplants, three are important and they are (i) oleanders (Nerium and cerbera) (ii) aconite, and (iii) nicotine.

CARDIAC POISONS

Examples are:

- Tobacco
- Digitalis
- Oleander
- Quinine
- Aconite
- Hydrocyanic acid

TOBACCO

Tobacco (tambakhu) is prepared from cured leaves of N*icotina tabacum*.

Active Principles

- Nicotine
- Nornicotine
- Dried leaves contain 1 to 8 percent of nicotine. Nicotine is colorless, volatile, bitter and hygroscopic liquid.
- *Toxic part:* tobacco leaves.

Uses

- Smoking tobacco
- Chewing
- Snuff
- Insecticide

Mechanism of Action

Nicotine acts on nicotine receptors present in autonomic ganglia,

adrenal medulla, central nervous system, spinal cord, neuromuscular junction and chemoreceptors of carotid and aortic bodies.

Absorption, Metabolism and Excretion

Tobacco or nicotine is absorbed from skin, mucous membrane and lungs. It is metabolized in liver and excreted in urine. Small amounts are also excreted in milk.

Fatal Dose

- 40 to 60 mg of nicotine
- 15 to 30 gm of crude tobacco

Fatal period: 5 to 15 minutes

Clinical Features

- ***Acute Poisoning***
  - Nausea and vomiting
  - Diarrhea
  - Pain in abdomen
  - Salivation
  - Tachycardia followed by brady cardia
  - Anxiety
  - Headache
  - Blurred vision
  - Confusion
  - Fasciculation
  - Convulsions
  - Paralysis
  - Coma
- ***Chronic Poisoning***

- Also known as nicotine addiction
- Common among smokers, tobacco chewers
- Develops physical dependence, manifested by recurrent craving for tobacco, tolerance, cough, impaired memory and amblyopia.
- Tobacco withdrawal is manifested by change in mood, insomnia, restlessness, constipation, headache and anxiety.

Complications

- Carcinoma of lung, esophagus, mouth, larynx
- Smokers cough
- Bronchitis
- Thromboangiitis obliterans
- Tobacco amblyopia

Maternal Smoking Leads to:

- Increased risk of spontaneous abortion
- Fetal death
- Increased frequency of abruptio placentae or placentaprevia
- Low birth weight baby

Cause of Death

- Respiratory failure — due to paralysis of CNS or paralysis of the endings of the respiratory nerves (myoneural junctions).
- Early death — due to stimulation of vagal cardiac ganglia with stand still of heart.

Management

- Gastric lavage with activated charcoal
- Benzodiazepines for convulsions
- Symptomatic treatment.

Autopsy Findings

- Brownish stains may present over skin
- Brownish froth at mouth and nostrils
- Signs of asphyxia
- Tobacco smell may present
- Brownish discoloration of esophagus and gastricmucosa
- Stomach may contains fragments of tobacco leaves
- Pulmonary edema

Medicolegal Importance

- Accidental poisoning may occur due to overdose
- Occupational hazard
- Malingering to induce illness
- Suicide or homicide is rare

COMMON OLEANDER

Botanical name: *Nerium odorum*

Common names: Common oleander, white oleander, Kaner.

Features

- Shrub grows all over India and bears lanceolate leaves with white or pink flowers.
- The leaves give clear thick juice.
- ***Toxic part of plant***: all parts
- ***Toxic Principles***
  - Oleandrin (glycoside)
  - Nerin

- Folinerin

Mechanism of Action

The glycosides have digoxin like action and inhibit sodium potassium ATPase

Fatal Dose

15 to 20 gm root

5 to 15 leaves.

Fatal period: 20 to 36 hours

Clinical Features

- Nausea and vomiting
- Diarrhea
- Tachycardia
- Breathlessness
- Ventricular fibrillation
- Delirium
- Drowsiness.

Management

- Gastric lavage with activated charcoal
- Atropine for AV block and sinus tachycardia
- Symptomatic treatment.

Autopsy Findings

- Congestion of organs
- Petechial hemorrhages over heart.

Medicolegal Importance

- Root used for causing abortion

- Accidental death occurs due to consumption of folk medicine containing oleander
- Cattle poison
- Suicide
- Homicide is rare
- Common oleander resist decomposition and burning, thus can be detected from decomposed bodies or ash.

YELLOW OLEANDER

Botanical name: Cerberathevetia

Common name: Yellow oleander, pila Kaner, exile.

Features

- This shrub resembles common oleander but have largebell-shaped yellow color flowers.
- The plant bears fruits, which are diamond to globularin shape and 4 to 5 cm in length. The fruits are initially greenish in colour but turns yellow and then black when becomes ripe. The fruit contains a singlenut, which is elongated triangular with deep groove along the edge. Each nut contains 5 pale yellow seeds.
- Milky juice (sap) exudes from all parts of plant.

- ***Toxic part of plant:*** all parts but seed and root are more toxic.
- ***Toxic Principles***
  - Thevetin
  - Thevetoxin
  - Cerberin
- ***Fatal Dose***
  - 8 to 10 seeds

- 15 to 20 gm of root
- 5 to 10 leaves

➢ ***Fatal period:*** 2 to 3 hours if powdered root taken.

Clinical Features

➢ The milky juice (sap) if applied to skin may cause inflammation in sensitive individuals

➢ Numbness in mouth and tongue

➢ Vomiting

➢ Diarrhea

➢ Headache

➢ Giddiness

➢ Loss of muscle power

➢ Tachycardia

➢ Jaundice4

➢ Renal failure

➢ ***ECG Changes***

- Sinus bradycardia
- A-V block I and II^{o}
- Flattening or inversion of T wave
- ST depression
- Ventricular and atrial ectopics.

Management

➢ Gastric lavage with multiple dose activated charcoal is said to be effective since charcoal binds glycoside in the gut lumen and promotes their elimination

➢ Bradyar rhythmias are treated with intravenous boluses of atropine

and intravenous infusion of isoprenaline.

- Temporary cardiac pacing
- Administration of antidigoxin Fab fragment is consider as effective but expensive and not widely available.

Autopsy Findings

- Congestion of organs
- Subendocardial and perivascular hemorrhage with focal myocardial edema

Medicolegal importance: Same as for common oleander.

ACONITE

Common name: mithazahar, bish, bikh, monk's hood

Botanical name

Aconitum napellus — European variety

Aconitum columbianum — American variety

Aconitum ferox — Indian variety.

Features

- Perennial plant with deeply cut leaves and long spikes of deep blue color flowers, with on upright downy stalks. The upper sepal of flower resembles a hoodor helmet or cowl, hence the common name monkshood.
- Aconitum ferox variety found in India and grows in Himalayas.
- The root is stout and dark, conical and shows scars of broken rootlets, shriveled with longitudinal wrinkles. The root is about 5 to 10 cm long and 1.5to 2 cm thick at upper end. The root may resemble horseradish root. However, horseradish root is cylindrical and pungent.

- ***Toxic Parts***

- Root (more toxic)
- Seeds and Foliage.

➢ ***Toxic Principles***

- Aconitine
- Mesoaconitine
- Hypoaconitine
- Pseudoaconitine
- Ind-aconitine
- Bikh-aconitine
- Aconine.

Mechanism of Action

➢ Aconitine acts on nerve axons by opening sodium channels.It also inhibits complete repolarization of them embrane of myocardial tissue causing repetitive firing.

➢ It stimulates vagal medullary center.

Metabolism

Metabolism of aconitum alkaloids is mainly carried out by the enzyme esterase. Aconitine is converted into benzoyaconine through hydrolysis in C-8 position and into aconine.

➢ **Fatal Dose**

- 1 to 2 gm of root
- 3 to 5 mg of a conitine

➢ ***Fatal period***: 2 to 6 hours.

Clinical Features

➢ Nausea and vomiting

➢ Salivation

- Tingling and numbness in mouth and lips
- Diarrhea
- Palpitation
- Weakness
- Hypotension
- Ventricular ectopics
- Arrhythmias
- Vertigo
- Blurring of vision, hippus, mydriasis, xanthopsia
- Convulsions.

Management

- Gastric lavage with activated charcoal
- Benzodiazepines for convulsions
- Symptomatic treatment.

Autopsy Findings

- No specific findings
- Organs are congested.

Medicolegal Importance

- Aconite is considered as near ideal homicidal poison, as it is sweet in taste and can easily be given with pan or other foodstuff.
- Suicide
- Accidental poisoning may occur due to mistaken with horseradish. On cut section, aconite appears pink where as horseradish appears white
- Root used to procure abortion
- Arrow poison

- Cattle poison
- Aconite gets easily destroyed by decomposition and may not be detected in chemical analysis.

HYDROCYANIC ACID

Cyanide occurs as solid, liquid or in gaseous state as:

Gas form — Hydrogen cyanide (HCN)

Liquid form — Hydrocyanic acid (Prussic acid)

Solid form — occurs as salts such as potassium cyanide/sodium cyanide.

Source

1. Plants: cyanide is present in form of cyanogenic glycosidein wide variety of plants such as:

Bitter almond

Sorghum

Johnson grass

Bamboo

Apricot

Peach.

2. Combustion: such as

Burning of plastic furniture

Burning of silk or wool

Cigarette smoking.

Absorption, Metabolism and Excretion

- Ingestion: Salts of cyanide releases hydrogen cyanide in stomach due to action of hydrochloric acid and the nabsorbed as cyanide ion (CN-).

It is concentrated in RBCs. Enzyme rhodanase (present in mitochondria of liver and kidney) converts them to thiocyanate. This reaction needs sodium thiosulfate. Some cyanide is converted into cyanocobalamin (vitamin B12) in presence of hydroxocobalamin (vitamin B12a). Small amounts ofcyanide are excreted in breath and sweat.

- Inhalation: Cyanide gas is rapidly absorbed from respiratory system.
- Dermal: Hydrocyanic acid is also absorbed through the skin.

Mechanism of Action

Cyanide reversibly inhibits ferric iron containing enzymes. Cyanide attaches to the iron of the prosthetic group of cytochromeoxidase resulting in disturbance of the transport andutilization of oxygen in cells and causing a cytotoxic anoxia.

- **Fatal Dose**
  - 50 to 100 mg for hydrocyanic acid
  - 200 to 300 mg for sodium/potassium cyanide
  - 50 to 80 bitter almonds
  - Inhalation of 1 part in 2000 — hydrogen cyanide
- ***Fatal period:*** 2 to 10 minutes.

Clinical Features

- ***Inhalation***
  - Constriction about throat
  - Dizziness
  - Loss of consciousness
  - Coma
  - Death
- ***Ingestion***

- CNS: Headache, anxiety, agitation, dizziness, confusion, convulsions, coma
- CVS: Initially bradycardia and hypertension and lattertachycardia and hypotension, arrhythmias
- RS: Tachypnea followed by bradypnea
- GIT: Nausea, vomiting, abdominal pain, numbness.

➢ ***Skin***

- Perspiration
- Cherry red color
- Bullae.
- Chronic poisoning: Chronic low level of exposure leads to:
- Headache
- Amblyopia
- Optic atrophy
- Peripheral neuropathy
- Ataxia
- Deafness
- Glossitis
- Stomatitis.

Management

➢ Ingestion: gastric lavage with 5 percent sodium thiosulfatesolution

➢ Antidote- amyl nitrate — inhaled

➢ Sodium nitrate slowly I.V.

➢ Sodium thiosulfate 25 percent solution I.V.

➢ Mechanism of action of nitrites: nitrates induce methemo globinemia, which causes detachment of cyanide fromthe heme group of

cytochrome oxidase since methemoglobinhas a higher binding affinity for cyanide. Cyanidecombines with methemoglobin and form non-toxic cynmethemoglobin.

➢ Mechanism of action of sodium thiosulfate: sodium thiosulfate serves as a substrate for the enzyme rhodaneseto catabolise cyanide to non-toxic thiocyanate, which is excreted in the urine.

Autopsy Findings

- Bitter almond like smell
- Cherry red color of postmortem lividity
- Cyanosis
- Froth at mouth and nose
- Bright red color blood9
- Pulmonary edema
- Serosal surface may show hemorrhages
- Stomach mucosa may be eroded and blackened due to formation of alkaline hematin.

➢ Samples to be preserved for chemical analysis

- Blood
- Routine viscera
- Lung
- Brain
- Heart
- Spleen is considered as best specimen due to presenceof more RBCs.

Medicolegal Importance

➢ Suicidal use of cyanide is not common and is usually limited to

specific occupational groups. Suicide with this agent is more common in those persons who are employed in electroplating, chemistry, mining and metalheat treatment and have ready access to this chemical.

- Homicide — rare
- Accident — industrial/laboratory mishaps
- Embalming interferes with cyanide detection, there for einterpretation in postmortem period becomes difficult.

SOMNIFEROUS POISONS

Narcosis means to induce sleep. Somniferous poisons referto agent capable of inducing sleep. Narcotic drugs were theterm employed to categorize these agents.

Examples are:

- Opium
- Morphine
- Heroin
- Codeine

Opium

Common name: Afim

Opium (afim) is the dried extract of the poppy plant (*Papaver somniferum*). The word "opium" is Greek for "Juice".

Features

➢ Opium plant grows up to 0.3 to 1.5 meter in height. The plant bears whitish color flowers with 5 to 8 capsules.

➢ The unripe opium capsules are incised to obtain the extract, which is milky fluid.

➢ The milky fluid on drying yields opium. Crude opium is irregular mass of brownish in color with a characteristic smell and bitter taste.

➢ Poppy seed (khaskhas) are white seeds used as condiment in India for cooking.

➢ Opium plants are cultivated under license in India in stateof Rajasthan, UP and MP.

Fatal dose of various opiates

Active principles

Opium contains alkaloids, which are divided into two groups:

1. Phenanthrene group: have narcotic properties

Morphine

Codeine

The baine (non-analgesic).

2. Benzoisoquinoline group: have mild analgesic but non arcotic properties

Papaverine

Noscapine (narcotine).

Classification

Opium and its derivatives are classified as:

- Natural: e.g. morphine, codeine
- Semi-synthetic: e.g. heroin, hydromorphine, oxymorphine
- Synthetic: e.g. meperidine, methadone, fentanyl etc.

Absorption, metabolism and excretion

- Morphine is N-demethylated and O-demethylated along with unchanged drug are conjugated with glucuronic acid. The unchanged and un conjugated morphine are excreted by the colon and by the kidneys. A small amount is excreted into the milk.
- Heroin is reduced to morphine by the liver.

Mechanism of action

- Opioids act by acting on specific opioid receptors. Opioid receptors are μ (MU), δ (DELTA) or κ (KAPPA) located at spinal and supraspinal sites in CNS.
- Opioid receptors are part of family of G-protein-coupledreceptors and act to open potassium channels and prevent the opening of voltage-gated calcium channels, which reduces neuronal excitability and inhibits the release ofpain neurotransmitters.
- The μ receptors are important and two subtypes are recognized. The μ1 receptors are associated with analgesia, euphoria and dependence whereas μ2 receptors are associated with respiratory depression and inhibition of gut motility.
- The κ receptors are responsible for analgesia at the level of spinal cord. The role of δ receptors in humans is not clear.

Clinical features

The effects occur in three stage

- 1. Stage of excitement:

- The stage is short
- The person feel better with increased sense of wellbeing
- Talkativeness
- Restless or hallucinations
- Flushing of face.

➢ 2. Stage of stupor

- Headache
- Nausea and vomiting
- Giddiness
- Drowsiness
- Miosis
- Stupor.

➢ 3. Stage of narcosis

- Patient passes into deep coma
- Muscles becomes flaccid
- Diminished or absent reflexes
- Hypothermia
- Hypotension
- Bradycardia
- Bradypnea
- Non-cardiogenic pulmonary edema
- Convulsions
- Respiratory depression

- Death.

➢ The **classic triad** for opioid poisoning is **miosis, comaand respiratory depression**. Miosis is due to para sympathetic stimulation (of Edinger-Westphal nucleus).However, once brain develops anoxic insult, there maybe mydriasis.

➢ There may be abdominal distention

➢ Opium contract smooth muscle. The tone of pylorusis increase, colon is made spastic and small in testine becomes more tonic with increase in rhythmic activity but decrease in propulsive rate. This results in constipation.

➢ Retention of urine occurs because bladder sphincter is made tonic preventing micturation.

Management

➢ Oxygen/assisted ventilation

➢ Fluid and vaso pressors

➢ Gastric lavage is effective since the opium is slowly absorbed from gastrointestinal tract

➢ Ventricular tachyarrhythmia can be managed by lidocaine

➢ Naloxone is a potent antagonist and does not cause respiratory or circulatory depressant action. The aim of naloxone administration is to reverse the respiratory and central nervous system depression. Caution should be exercised while giving nalox one because it can precipitate acute withdrawal syndrome in chronic opioids users.

➢ A recently introduced antidote is nalmefene. Nalmefene has pure opiate antagonistic effects and could prove superior to nalox one.

Differential diagnosis

- Alcohol intoxication
- Barbiturate poisoning
- Carbolic acid poisoning
- Carbon monoxide poisoning
- Uremic coma
- Diabetic coma
- Hysteria
- Cerebral hemorrhage
- Head injury
- Cerebral malaria
- Meningitis/encephalitis
- Heat hyperpyrexia.
- ***Fatal dose***
  - Crude opium — 500 mg
  - Morphine — 200 mg
  - Heroin — 50 mg
  - Pethidine — 1 gm
- Fatal period: 6 — 12 hours.

Autopsy findings

- Signs of asphyxia
- Froth at mouth and nostrils
- Smell of opiate may present

- Injection marks/skin abscess/scarring in addicts
- Emaciation
- Pulmonary edema
- Cerebral edema.

Medicolegal importance

- Drug abuse
- Accidental death due to drug overdose
- Suicide may be attempted for painless and peaceful death
- Cattle poison
- Doping for horse race
- Homicide — rare
- Infanticide
- Used in euthanasia.

Prolonged Use of the Opium

The prolonged use of the drug produces the following effects: (i) Emaciation (ii) Dry skin and hair (iii) Pigmentation around the mouth and eyelids (iv) Dry and furred tongue (v) Anorexia (vi)Nausea and marked constipation (vii) Impotence(viii) Neurasthenia (ix) Dementia or mania

Withdrawal Symptoms

The following are the withdrawal symptoms when opium ingestion is stopped:

1st few hrs : Psychological effects due to fear, anxiety

8-16 hrs : Nervousness, restlessness, anxiety

14 hrs : Yawning, sweating and running of eyes and nose

24 hrs : Dilated pupils, goose skin and shivering

36 hrs : Severe twitching of muscles, painful cramps in legs and abdomen, vomiting, diarrhoea, insomnia

3-4 days : The symptoms gradually sub sides

Treatment of Withdrawal Symptoms

1. Opiates should be gradually withdrawn

2. Cyclazocin 4mg to be given daily

3. Methadone 100mg daily orally (1mg Methadone= 4mg morphine = 20mg pethidine).

i. ***Methadone maintenance****:* It is usually used for intravenous heroin users. Methadone maintenance units differ in their selection procedures, dose, duration of treatment, their philosophy and rules as well as ancillary metods of treatment. It is the treatment of first choice and among other methods it enjoys a high retention rate of clients due to its effectiveness. Methadone was synthesized by a German scientist and came was brought in to clinical use at the end of World War II. Methadone hydrochloride in a bitter white powder, that is soluble in water and ethanol. Under the trade names as 'Dolophine' and 'Westadone 'It is available as 5mg and 10mg tablets for oral use and for 10mg/ml injections can be given intravenously After subcutaneous injection, methadone can be found in the plasma within 10 minutes and after oral ingestion, is found after 30minutes. The peak concentration occurs within 4 hours whereas in the brain, the peak is in 1-2 hours. Methadon is an antidote of morphine but due to its own depressive action on respiration and other side effects has made it unsuitable for the purpose. It is a synthetic opiate and is a narcotic agent. After use of methadone, patient should take bed rest. Movement precipitates dangerous side effects.

Side effects of methadone are excessive sweating, Lymphocytosis and increased prolactin, albumin and globulin in the plasma. In urine, the metabolites of methadone are excreted are Pyrolidine and Pyrolline.

ii. ***Clonidine:*** A centrally acting á-Adrenergicagonist can suppress the Opioid with drawal from low to moderate doses of methad one. It is a potent anti hypertensive.

iii. ***Propranolol:*** 80 mg effectively relieves anxiety and craving associated with opiate addiction

iv. ***Nalorphine (Nalline/N-Allyl-normorphine):*** It is a semisynthetic opiate. It causes analgesia, respiratory depression, dysphoria and hallucination. It was popularly used as antidose of morphine. But its ownde pressive action on respiration and other side effects has made it unsuitable for the purpose. Nalorphine hydrochloride 5mg i.v is given for 15-30 minutes, then 10mg i.v toa total dose of 40 mg

v. ***Naloxone Hydrochloride:*** 0.4-0.8mg i.m/I.V. is given in adults and 0.01mg/kg is givenin children.

vi. Psychological counselling of the patient has an important role.

HEROIN

Heroin is diacetyl morphine and is a semi synthetic preparation of morphine. It was first prepared at St. Mary's Hospital, London by acetylaion of morphine with acetic anhydride in 1898. Morphine was first used for the treatment of cough but soon it became the most potent drug used by drugaddicts due to its habit-forming properties.

Manufacture of Heroin and its use in Medicine is banned now a days but it is being illegally manufactured and smuggled, as its cost is very high in international market (1Kg of morphine costs about one crore). This is quite profitable to India as it lies in the 'golden triangle' on eastern side and 'golden crescent' on the Western side. India is one of the countries for

transit of morphine and other drugs. Cultivation of opium and production of Heroin is done in 'golden quadrangle' namely Varanasi, Lucknow, Bareily and Badauin districtof U.P, and adjoining states of Rajasthan andMadhya Pradesh. About 10kg of crude opium give1kg of standard opium. This can be converted into Chinese Heroin (called No.4 Heroin) and Brownsugar (called No.3 Heroin—60% pure) that is available in India. Brown sugar is also calledsmack and its colour is brown due to presence ofsugar of milk, starch, powdered coffee, tea, andcoco or brick powder. The smack of 'goldencrescent' is grayish and that used of 'goldenquadrangle' is blackish-brown in colour. Brownsugar is sold in small cellophane packets of116gms each. The amount of Heroin in eachpacket may be 1/16th, 1/18th, or ½ of a gram.These cellophane bags are kept in a matchbox toavoid suspicion.

Routes of Consumption

- (i) Sniffing in the form of snuff
- (ii) Smoking incigarettes or *bidis* after removal of some tobacco
- (iii) Chasing (a cigarette foil or RS. 5or 10 note isfolded and instead of tobacco, the drug is putinside, this is then lighted using match stick, whenthick smoke come out it is inhaled—called 'Dragonchasing")
- (iv) Injections by s.c (*skin poppers*) andI.V. route (*main-liners*)

Signs and Symptoms

- (i) Anorexia
- (ii) Nausea and vomiting
- (iii) Constipation
- (iv) Emaciation
- (v) pigmentation aroundcheek

- (vi) Impotency.

Complication of Consumption

- (i) Abscess formation along the routes of injection
- (ii) Formation of pigmented scars in cubital fossadescribed as 'rail-road track'
- (iii) Thromosed veins
- (iv) Septicemia
- (v) AIDS and Hepatitis.

PETHIDINE (MEPERIDINE, DEMEROL)

It is a synthetic opiate and is a narcotic. Some ofits actions are in variation with morphine. It is agood analgesic and sedative. In contrast with theaction of morphine it is mydriatic. It causes drynessof the skin. It has direct action on the heartmusculature which is inhibits. It liberates histaminefrom mast cells. Pethidine causes early loss ofnorneal reflex, due to its anaesthetic effect on thecorneas. It is highly addictive agent and doctorsand paramedical workers are the common victims.Pethidine is therapeutically contra-indicated incases of high intracranial pressure. It is also notgiven in toxemia or pregnancy.

INEBRIANT POISONS

Inebriant are the substances capable of causing intoxication.These are poisons which produce excitement and narcosis:

Classical inebriants—Alcohols (*ethyl alcohol, methylalcohol, ethylene glycol*, etc.)

Anaesthetics—*Chloroform, ether*, etc.

Sedatives and hypnotic poisons—*Chloral hydrate, barbiturates*, etc.

Benzodiazepines—*diazepam, flurazepam*, etc..

Hydrocarbons—Aliphatic (e.g.*diesel oil, petrol, kerosene,*etc.), aromatic (e.g. *benzene*) and halogenated (e.g. *carbontetrachloride*)

Insecticides—*Organophosphorous compounds, Carbamates and Organochloro compounds.*

Examples of inebriant compounds are:

- Alcohol
- Barbiturates
- Chloral hydrate
- Ether
- Ethyl chloride
- Chloroform
- Tetrachlorethane
- Paraldehyde

ALCOHOL

The word 'alcohol' is derived from the Arabic word "*Al kohl*"which means "something subtle". The term whiskey is derivedfrom USQUEBAVGH, Gaelic for "water of life". Alcohols arehydroxy

derivatives of aliphatic hydrocarbons. When unqualified,'alcohol' refers to ethyl alcohol or ethanol (C_2H_5OH).

There are three categories of alcohols:

- Monohydroxy alcohols — these alcohols have only one hydroxyl (OH) group. For example, ethanol, methanol, isopropanol.
- Dihydroxy alcohols — these alcohols have two hydroxyl (OH) groups and are known as glycols. For example, ethylene glycol, propylene glycol.
- Trihydroxy alcohols — they are not really alcohols, but are only derivatives. For example, propane derivative glycerol or glycerine.

ETHYL ALCOHOL

Synonyms: ethanol, grain alcohol

Features

- Alcohol is clear, colorless liquid with typical fruity odor and having burning taste.
- Ethanol is both — water and lipid soluble. The hydroxyl and ethyl moieties confer both hydrophilic and lipophilic properties. Thus ethanol is an "amphophyle".
- The aliphatic alcohol forms a homologous series beginning with methanol, ethanol, n-propanol, isopropanol etc. The first three are readily soluble in water in all proportions but as the carbon chain length increases, water solubility decreases and octanol (8 carbons) is almost insoluble in water.
- All alcohols have general formula R-OH.
- The specific gravity of ethanol is 0.79, i.e. 1 ml of alcohol weighs 0.79 gm.

➢ Alcohol beverages are primarily a mixture of water and ethyl alcohol with small amounts of other substances, which impart the characteristic odor and taste to the beverage. These substances are referred as **congeners** because they are simultaneously produced during the fermentation process. Congeners consist of organic acids and esters.

Sources and preparations

Alcohol is produced by fermentation of sugar by yeast. Theprocess halts at a concentration of alcohol, by volume, ofapproximately 15 percent because of the death of yeast abovethis level. The sugar from cereal, vegetable or fruit is used.If cereal is used as raw material for alcohol preparation thenit has to be malted first to convert the starch into maltosebecause yeast cannot ferment starch. Malt is produced bymoistening the barley and allowing it to sprout, which is thendried, ground and added to the cereal in water resulting inthe formation of mash. Beer is brewed by filtering mash andtreating the filtered liquid with yeast. Whiskey is preparedby adding the yeast directly to the mash. Strong alcoholicbeverages are distilled after fermentation. Distillation furtherincreases the alcohol concentration.

Types of alcohol beverages

Alcohol beverages are of various types as described below

➢ A. Malted liquors — they are fermented product but are undistilled liquids so alcohol concentration is low. Examples are beer, stout.

➢ B. Wines — prepared by fermentation of natural sugars such grape or fruits. These drinks are also un-distilled. Wines are called “dry wine” when all sugar present has been fermented and “sweet wine” when some sugar is left. They are of following varieties:

o Light wines — such as claret, cider. Alcohol content did not exceed 15 percent

- Fortified wines — such as port, sherry. Here distilled beverages are added from outside. Alcohol content varies from 16 to 22 percent.

- Effervescent wines — such as champagne. These drinks are bottled before fermentation is complete.The alcohol content varies from 12 to 16 percent.

➢ C. Spirits — these are distilled drinks and alcohol content varies from 40 to 55 percent. Example includes —rum, whiskey, gin, brandy, vodka etc.

Types of alcohol

➢ Absolute alcohol — contains 99 percent w/w ethanol (dehydrated alcohol)

➢ Rectified spirit — contains 90 percent w/w ethanol (distilled)

➢ Proof spirit — it is an old term and refers to a standard mixture of alcohol and water of relative density 12/13 at 51oF, i.e. 49.28 percent of alcohol by weight or 57.10 percent by volume. Proof strength of alcoholic beverages is expressed in degrees. The ethanol content of various alcoholic beverages is expressed by volume percent or by proof. The proof being twice the percentage of alcohol by volume. Nowadays, alcohol is referred by percentage alcohol by volume (% v/v). This is equivalent to the number of milliliter of pure alcohol per 100 ml of the drink.

Glossary

➢ Denatured means the alcohol is processed in a prescribed manner so as to make it unfit for human consumption.

➢ Liquor includes spirits, denatured spirits, wine, beer, toddy and all liquids consisting of or containing alcohol.

➢ Country liquor includes all liquors produced or manufactured in India as per specifications for country liquor

- Indian made foreign liquor is liquor produced in India as per specifications. Examples — rum, gin, whiskey, brandy etc.
- Foreign liquor means potable duty-paid foreign liquor
- Rectification includes every process whereby liquor is purified or refined for making it potable
- Spirit means any liquor containing alcohol and obtained by distillation
- Toddy means fermented or unfermented juice drawn from a coconut, brab, date or any kind of palm tree and includes sweet toddy
- Sweet toddy or nira or neera means unfermented juice drawn from a coconut, brab, date or any kind of palm tree into receptacles treated in the prescribed manner so as to prevent fermentation.

Uses of alcohol

- Beverage
- Solvent
- Medicinal and therapeutic
- Antidote in methanol poisoning
- Preservative
- Fuel.

Absorption, Metabolism and excretion

Alcohol is rapidly absorbed from the stomach (20%) andsmall intestine (80%). Due to thinner mucosa, better bloodsupply and a larger surface area, the upper small intestine— the duodenum and jejunum has maximum capacity forabsorption of alcohol than gastric mucosa. As

alcohol is lipidsoluble, it diffuses from stomach and intestine by simple diffusion.However, alcoholic beverages more than 20 percentare absorbed slowly because higher concentration of alcoholinhibits gastric peristalsis and thus delays gastric emptying.Alcohol is subjected to gastric "first pass metabolism" due topresence of gastric dehydrogenases but they are not presentin intestine. The female has less alcohol dehydrogenase thanmale. When alcohol is absorbed, a substantial fraction isremoved from the portal vein blood by first pass hepaticmetabolism. Hepatic metabolism of alcohol shows saturationkinetics at low alcohol concentrations, so the fractionof alcohol removed decreases as the concentration of alcoholreaching liver increases. Thus, if alcohol absorption is rapidand portal vein concentration is high, most of the alcoholescapes into the systemic circulation whereas with slowabsorption, more alcohol is removed by first pass metabolism.

This is one reason why drinking alcohol on an emptystomach produces a much greater effect. Vaporized ethanolmay be rapidly absorbed by inhalation.

There are various factors that affect absorption of alcoholand these factors are discussed below.

- Food — presence of food in the stomach prolongs the absorption of alcohol. Presence of starch, proteins and fatty food retards the absorption. It is stated that with presence of food in stomach, as much as 17 to 20 percent of alcohol ingested escapes absorption and never appears in the blood.

- Concentration — diluted alcohol or alcohol with high concentration are absorbed slowly whereas alcohol in the concentration of 10 to 20 percent are absorbed rapidly.

- Habit and tolerance — in habituated person alcohol is absorbed rapidly.

➢ Gastrectomy may cause rapid absorption of alcohol. Similarly alcohol is absorbed rapidly in persons who had truncal vagotomy and drainage operation due to increased gastric emptying. More than 90 percent of ethanol consumed is metabolized in the body and only 5 to 10 percent is excreted unchanged by kidneys (urine), lungs (breath), feces and skin (sweat).

Alcohol in the systemic circulation is metabolized throughthree pathways:

➢ Alcohol dehydrogenase (ADH) pathway — in the cell cytosol

➢ Microsomal Ethanol Oxidizing System (MEOS) — located on the endoplasmic reticulum

➢ Peroxidase-Catalase system — in hepatic peroxisomes.

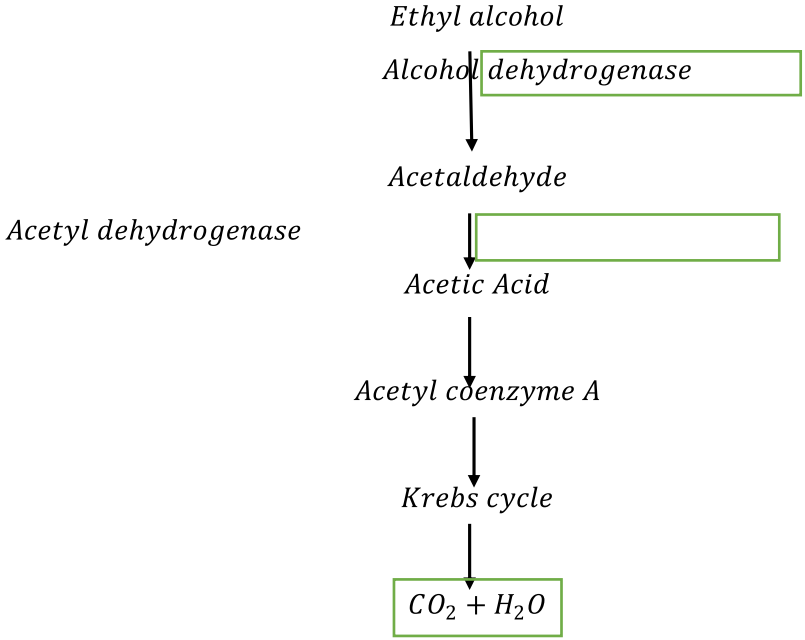
Drugs affecting gastric emptying
Drug with anticholinergic action like — atropine,chlorpromazine, Tricyclic antidepressants - Drugs with an adrenergic actions like — amphetamine - Drugs with opioid action like — codeine, heroin, methadone
Drugs hastening emptying of gastric contents - Metoclopramide - Cisapride - Erythromycin

ADH pathway is the principal pathway metabolizing more than 90 percent of the systemic ethanol. Ethyl alcohol is first oxidized to acetaldehyde by alcohol dehydrogenase and then this gets converted into acetic acid by aldehyde dehydrogenase which in turn is converted to acetyl coenzyme A and enters the Krebs (Citric acid) cycle where it is metabolized to carbon dioxide and water. Alcohol metabolism by alcohol dehydrogenase follows first order kinetics after the smallest dose. Once

the blood concentration exceeds about 10-mg/100 ml, the enzymatic processes are saturated and elimination rate no longer increases with increasing concentration but becomes steady at 10 to 15 ml/hour. Thus, alcohol is subject to dose dependent kinetics, i.e. saturation or zero-order kinetics.

Mechanism of action

- Alcohol is a central nervous system depressant that decreases activity of neurons with behavioral stimulation at low blood level.
- Alcohol produces simultaneous changes in many neurotransmitters and increases the fluidity of neuronal cell membranes.
- Alcohol in small doses interferes with cortical function like conduct, judgment, self-criticism and release of inhibitory tone but in larger doses depresses the medullary processes.



Alcohol Intoxication

Alcohol intoxication is a state associated with behavioral,psychomotor and cognitive changes in an individual. Acutealcohol intoxication is also called as **acute alcohol poisoning**or **inebriation**. The clinical features are described below.

Consumption of alcohol produces following stages.

- **Stage of excitement** characterized by a sense of wellbeing, sober, euphoria, increased confidence and excitement. There is depression of inhibitory controlling capacity of higher centers. The person is free in action, speech and emotions. But as the consumption of alcohol goes on, the person may land in the stage of in-coordination.

- **Stage of in-coordination** is characterized by muscle incoordination; sense perception and skilled movements are affected. The individual becomes irritable, quarrelsome with slurring of speech and unsteady gait. The face will be flushed and breath smells of alcohol. There is increase in reaction time.

features of intoxication and blood level		
Blood alcohol Concentration (mg/100 ml)	*Level of Intoxication*	*Clinical features*
0 – 50	Sobreity	Sober behavior
50 – 100	Euphoria	Feeling of well-being, increased self-confidence, decreased inhibition
100 – 150	Excitement	Free in action and emotion, talkativeness, fine movements affected, impairment in memory and comprehension
150 – 200	Confusion/In-coordination	In-coordination of muscle, skilled movements and perception affected, increased reaction time, slurred speech, staggering gait, confusion, disorientation, visual disturbances
200 – 300	Stupor	Diminished response to stimuli, inability to stand or walk, vomiting
300 – 500	Coma/death	Unconsciousness, abolished or diminished reflexes, subnormal temperature, incontinence of urine &feces, respiratory distress, death.

➢ **Stage of coma** as the consumption of alcohol continues, the person becomes drowsy and lands into coma.

o Alcohol causes mydriasis (dilatation of pupil) in the initial stages but as person lands in stage of coma, the pupil becomes constricted (meiosis). The so-called **McEwan** sign is positive in coma. Pinching of the skin orlight slapping of a person causes the constricted pupil ofperson to be dilated.

o Diagnostic criteria for alcohol intoxication — as perDSM IV developed by American Psychiatric Association.

diagnostic criteria for alcohol intoxication as per DSM IV
Recent ingestion of alcohol
- Clinically significant maladaptive behavioral or psychological changes (for example, inappropriate sexual aggressive behavior, mood labiality, impaired judgment, impaired social or occupational functioning) that developed during or shortly after alcohol ingestion
- One (or more) of the following signs, developing during or shortly after alcohol use: • *Slurred speech* • *Incoordination* • *Unsteady gait* • *Nystagmus* • *Impairment in attention or memory* • *Stupor or coma*
- *The symptoms are not due to a general medical condition or are not better accounted for by another mental disorder*

Alcohol intoxication and brain functioning

- ➢ Impairment of frontal lobe
  - o Impaired judgment
  - o Awkward motor coordination
  - o Decreased concentration
  - o Impaired abstraction
  - o Language is incoherent and disconnected
- ➢ Impairment of parietal lobe
  - o Depersonalization
  - o Abnormalities in orientation
- ➢ Impairment of temporal lobe

- Defect in recent memory
- Blackouts — lack of formation of new memory
- Defect in language reception
- Difficulty in understanding or listening

➢ Impairment of occipital lobe

- Impaired visual acuity
- Reduced ability to discern object and motion (blurredvision)

Differential diagnosis of alcohol intoxication

➢ Barbiturate poisoning

➢ Diabetic coma

➢ Carbon monoxide poisoning

➢ Hypoglycemia

➢ Head injury

➢ Cerebro-vascular episode

Death from alcohol intoxication

➢ Respiratory failure

➢ Circulatory failure

➢ Aspiration of gastric contents

➢ Hypoglycemia

Fatal dose

➢ Levels of blood alcohol above 500mg/100 ml are considered to be probably fatal.

➢ The fatal dose in adult is 6 gm of ethanol/kg body weight and in

children it is 3-gm/kg body weight.

Management

Principles of management of alcohol intoxication are:

- Secure airway
- Maintain adequate blood pressure and circulation
- Rule out head and spine injury
- Administer thiamine 100 mg intravenously followed by glucose 25 to 50 gm intravenously to protect against development of Wernicke-Korsakoff syndrome
- Manage hypothermia in usual way
- Nasogastric intubation and gastric lavage, may be helpful if patient presents within one hour after incident
- Access status of acidosis. A worsening acidosis after thiamine, glucose and fluid therapy should raise the possibility of ethylene glycol or other ingestion.

Drunkenness

A medical practitioner is frequently asked by law enforcingauthorities to examine a person for causing nuisance in publicplaces or driving vehicle under the influence of alcoholor had committed an offence under its influence. Under suchcircumstances, a doctor is expected to examine the personand opine regarding following points:

- Whether the examined person had consumed alcohol? And
- If he had consumed alcohol, whether he is under its influence?

The medical practitioner engaged for such job should alwaysexercise caution while furnishing an opinion. The RMPshould know what is

drunkenness? How to examine suchperson? How to collect exhibits which might be used asevidence in court of law. The following account providesguidelines for such procedure and practice.

Definition

Drunkenness is a condition produced in a person who hastaken alcohol in a quantity sufficient to cause him to losecontrol of his faculties to such an extent that he is unableto execute the occupation in which he is engaged at thematerial time.

Examination

Aims of examination

- Whether the person had consumed alcohol?
- If he had consumed alcohol, whether he is under its influence?
- Whether the state is due to alcohol intoxication or some other natural illness or due to other intoxicants or due to head injury?
- Whether it is safe for him to be detained in police custody or whether he should be admitted in the hospital for treatment?

Consent

Like other examinations, the consent should be obtainedfrom the person before proceeding for examination. Wherea person is too drunk or otherwise unconscious so that noconsent could be obtained, then the doctor should makeexamination as possible but the result of his examinationshould not be given to police until retrospective consenthas been obtained from the person when he has recoveredcompletely. However, if the person had been arrested for acriminal offence and is under the custody of police, then asper CrPC Sec. 53(1), the medical practitioner can proceedwith the examination of accused without his consent.

Preliminary particulars

Preliminary particulars such as name, age, sex, address, identificationmarks, time and date of medical examination, nameof accompanying person and brief history should be recorded.

Physical examination

1. General appearance — state of clothing (whether soiledby vomitus or feces), behaviour, disposition.

2. Record vital data such as — temperature, pulse, respiratoryrate and blood pressure. In alcohol intoxication thereis tachycardia and vasodilatation of cutaneous vesselsthat results in warm and flushed skin. Respiratory ratemay be increased. Low doses of alcohol causes a milddrop in blood pressure but more consumption results indose-dependent increase in blood pressure.

3. Speech — **normal, thick, slurred or over-precise**.Speech production is a complex motor activity andrequires coordination and is considered as sensitive indexof alcohol intoxication. Slight distortion of certain consonantsis one of the early signs of in-coordination ofmuscles of tongue and lips. Certain test phrases may beused to bring out this difficulty in speech such as "***Britishconstitution***", "***truly rural***" etc. A sober person may saythat he is not good at such phrases; the drunken personoften embarks to get them correctly.

4. Breath — smell of alcohol present or absent. The congenerspresent in alcoholic beverages impart peculiar odorthat could be appreciated in the breath of drunken person.However, certain other preparations may also impartsuch odor and the substances are given below:

- Ayurvedic preparations — contains alcohol up to 14 percent.
- Wincarnis — contains alcohol up to 20 percent.

5. Stance — does he stand properly or sway when standingerect with his feet together and eyes closed? A drunkenperson sways while doing such exercise. This is a – positive**Romberg sign.** (In Romberg's test, the person isasked to stand with his feet closely approximated, firstwith his eyes open and then with his eyes closed).

6. Gait — observe whether the gait is normal or staggering?A drunken person has staggering gate. The personis asked to walk on a straight line. His gait should beobserved. While walking straight, the person is askedsuddenly to turn to judge the reaction time. Drunkenperson will take more time for turning and takes one ortwo steps before turning.

7. Writing — the person should be asked to write few linesin a language familiar to him. Note the time taken towrite, repetition or omission of words, ability to readhis own handwriting. The drunken person often takesmore time to copy a sentence and may have difficulty inwriting the letters N, M or W. Often; he omits or repeatssome words or alphabets.

8. Eyes — note the state of conjunctiva, pupillary size andreflex, visual acuity and presence or absence of nystagmus.

- In drunken person:

- Conjunctivas are congested

- The pupils — in early stages, it is stated that pupils are dilated and as the level of intoxication advances, especially in coma, it becomes pinpoint.

- Pupillary reflex — the pupillary response to light is delayed or sluggish in drunken person.

- Convergence — in this test, the extrinsic muscles of eye are tested. The person is asked to follow a finger of medical examiner in all normal

directions and then ask to converge the eyes on a near object.

- Nystagmus — nystagmus is noted in alcohol intoxication. The presence of fine lateral nystagmus (alcohol gaze nystagmus AGN) indicates alcohol intoxication. Due to reduced visual acuity and dilated pupils, drunken person stares and tries to steady his gaze leading to alcoholic gaze nystagmus.

- Alcohol causes nystagmus by two mechanisms:

➢ Firstly by acting on vestibular system — it can cause positional alcohol nystagmus (PAN) detected when the patient is lying supine with the head turned to either the left or right side.

➢ Secondly by inhibiting the smooth pursuit system. There is impaired ability to maintain eccentric gaze brought about by alcohol's effect on ocular movements via neural mechanisms — results in horizontal gaze nystagmus (HGN).

- PAN occurs in two stages — PAN-I and PAN-II

- PAN-I is associated with acute elevation of blood alcohol tending to occur approximately 30 minutes after alcohol ingestion. In PAN-I, the first phase of nystagmus is in the direction toward which the head is turned.

- PAN-II — normally occurs at approximately 5 to 6 hours after drinking and is characterized by nystagmus in the opposite direction to that seen in PAN-I.

- HGN is a jerky eye movements noted when gaze is directed to one side. The first phase of HGN is in the direction of gaze and it becomes intensified at a more eccentric gaze position. It may be seen in normal individuals at extreme lateral gaze, when detected at lesser deviations, it is considered as pathological. An angle of onset of 40° or less from the midline is a sensitive indicator of a blood alcohol level in excess of 100

mg/100 ml. HGN is also noted in other conditions like ingestion of sedative and tranquilizing drugs.

9. Reflexes — in drunken person, knee and ankle reflexesare sluggish or delayed. Planter reflex may be extensoror flexor.

10. Muscular coordination — following are some test usedto determine muscle coordination

- Finger nose test — here the person is asked to touch nose by his index finger alternately with each upper limb
- Finger to finger test
- Unbuttoning and buttoning the shirt
- Picking objects from floor.

➢ ***Systemic examination***

- CNS — examination is carried out to access:
  - Memory — test the ability for recent memory
  - Orientation to time and place.
- Other systemic examination should be done in the usual way. Note presence or absence of injuries or any pathological condition resembling alcohol intoxication.

Collection and preservation of samples

➢ Blood — while collecting blood sample, spirit should not be used to cleanse the surface rather the surface should be washed with soap and water. Five ml blood sample should be collected in screw-capped bottle of universal size. The cap should be properly secured after adding appropriate preservative. five mg of sodium fluoride (acting as enzyme anticoagulant) for 5 ml of blood.

➢ Urine — for proper analysis, two samples of urine are required. The first sample should be collected and after 20 to 30 minutes later second sample should be collected. The concentration of alcohol in second sample reflects the blood alcohol during the inter-specimen interval. The difference in the alcohol concentrations in the two samples indicate whether the person was in absorptive phase, or at its peak or in the elimination phase. Multiplication of alcohol concentration in the second sample by 0.75 will give an approximate value of blood alcohol level at the time when the urine is being secreted. Phenyl mercuric nitrate or thymol is used as preservative.

Opinion

After examination of the person, the medical examinershould furnish opinion in one of the following ways:

1. The individual has not consumed alcohol.

2. The examined individual has consumed alcohol but heis not under its influence.

3. The examined individual has consumed alcohol and heis under its influence.

Interpretation of blood alcohol result

The proof strength of a liquid is obtained by dividing thealcohol percent (volume strength) by 0.571. For example,if we want to calculate proof strength of a wine containing10 percent alcohol, therefore 10 ÷ 0.571 = 17.5°proof. Similarly the percentage of alcohol in a liquid isobtained by multiplying the proof strength by 0.571. Forexample – whisky is 75° proof will contain - - 75 X0.571 = 42.8 percent by volume of alcohol.

Concentration of alcohol in blood is expressed as mg%,i.e. (w/v) or as

percentage. For example – 60 mg% (w/v)= 0.06% (w/v), i.e. 60 mg of alcohol per 100 ml blood(weight/volume).

Concentration of alcohol in solid tissue like viscera isexpressed as mg of alcohol per 100 grams of tissue(w/w).

The amount of alcohol consumed can be determinedfrom the blood alcohol level.

For calculating blood levels, various methods are usedand amongst them method advocated by Widmark ispopular. The Widmark formula is provided in equationis – **a = cpr**. Where a = amount of alcohol in gramsabsorbed in body, c = concentration of alcohol in bloodin grams/kg, p = weight of person in kilogram andr = constant. In men, the constant is 0.68 and in womenit is 0.55.

For urine analysis, the formula is – **a = ¾ qpr**. Theother factors are same and q = concentration of alcoholin urine in grams/liter.

Breathe analyzer

- Also called as alcometer, intoximeter or drunkometer
- Breath analyzer serve as on-spot test for police
- Legally admissible as per sec 185 of Motor Vehicle Act 1988
- In contemporary period, more sophisticated versions are available.

Principle

The concentration of alcohol in lung air is dependent onarterial blood. It is established that there is correlationbetween breath and blood alcohol and the ratio is assumedto be 2100:1, i.e. amount of alcohol in 2100 ml of alveolarair = amount of alcohol in one ml of blood. This is basedon Henry's law. The law state that when a volatile substance(alcohol) is dissolved in liquid (blood) and is broughtto equilibrium with air (alveolar air), there is fixed ratiobetween the concentration of the volatile substance

(alcohol)in air (alveolar air) and its concentration in liquid (blood)and ratio remains constant at given temperature.

Sources of errors in breatheanalyzer

- Variation in ratio between different individuals
- Use of ethanol containing products
- Belching or regurgitation of gastric alcohol contents
- Inadequate expiration (in unconscious or un-cooperative subject)
- COPD disease
- Use of metered dose inhalers
- Poor technique.

Alcohol and traffic accidents

It is established that driving vehicle under influence of alcoholcauses more accidents leading to increase in morbidityand mortality. Driving a vehicle under the influence of alcoholis an offense in India under the Motor vehicle Act 1988.A fine up to Rs. 2000 can be imposed or imprisonment canbe awarded that can be extended up to 6 months or both.The statutory limit of blood alcohol is **30 mg%** in India (asper Sec 185 of Motor vehicle Act 1988). The increase inaccidents are attributed to following factors:

1. Risk taking behavior
2. Impaired performance
3. Increased reaction time
4. Tracking
5. Poor mental coordination
6. Information processing

7. Visual — blurring of vision, decrease visual acuity,strong lights are needed to distinguish objects and dimobjects are not distinguished at all. There is also lackof color discrimination.

8. Psycho-motor performance

9. Altered time and space perception

10. Impairment of judgment

11. Skilled movements affected

12. Tendency to drive vehicle in the middle of road

13. Inaccurate cornering and poor judgment.

ALCOHOLISM

Also called as chronic poisoning, ethanolism, alcohol abuse,alcohol dependence

Definition

Alcoholism is a disorder characterized by excessive drinkingthat results in injury to person's health or inadequate socialfunctioning or both

DSM-IV defines alcoholism as repeated alcohol-related difficultiesin at least three of following seven areas of functioning:

1. Tolerance

2. Withdrawal

3. Taking larger amounts of alcohol over longer period thanintended

4. Inability to control use

5. Spending great deal of time associated with alcohol use

6. Giving up important activities to drink

7. Continued use of alcohol despite physical or

psychologicalconsequences.

Alcohol abuse is considered as repetitive problem with alcoholin any one of the following four areas of life withoutalcohol dependence:

1. Inability to fulfill major obligations

2. Use of alcohol in hazardous situation such as driving etc

3. Incurring legal problems

4. Use despite social or interpersonal difficulties.

Dipsomania is an irresistible desire to take large amounts ofalcohol until the person become almost unconscious fromits effect.

Complication of alcoholism

- CNS
  - Alcoholic blackouts
  - Fragmented sleep (restless sleep)
  - Peripheral neuropathy
  - Wrenicke's and Korsakoff's syndrome
  - Cerebellar degeneration
  - Cognitive problems
  - Permanent CNS impairment
  - Marchiafava-Bignami syndrome — it is rare idiopathic syndrome of dementia and seizures with degeneration of the corpus callosum. The syndrome is primarily reported in male Italian drinkers of red wine.
- CVS
  - Mild to moderate hypertension with heavy drinking
  - Alcohol cardiomyopathy

- Holiday heart — atrial or ventricular arrhythmias, especially paroxysmal tachycardia can occur after a binge in individuals with no evidence of heart disease.

➢ GIT

- Esophagitis
- Gastritis
- Duodenal ulcerations
- Upper GIT bleeding
- Mallory-Weiss syndrome
- Anorexia
- Abdominal pain
- Esophageal varices
- Atrophy of gastric cells
- Pancreatitis
- Mal-absorption
- Diarrhea secondary to increased small-bowel motility and decrease water and electrolyte absorption
- Fatty liver
- Alcoholic cirrhosis
- Alcoholic hepatitis.

➢ Genitourinary system

- Increase sexual desire in men but decrease erectile capacity (William Shakespeare wrote very aptly regarding alcohol and sexual function – " alcohol provokes the desire, but it takes away the

performance")

- Testicular atrophy
- Shrinkage of seminiferous tubules
- Decrease sperm count
- Amenorrhea
- Decrease in ovarian size
- Absence of corpora lutea with infertility
- Spontaneous abortion.

➢ Nutritional complications

- Folic acid deficiency
- Pyridoxine deficiency
- Thiamine deficiency
- Iron deficiency
- Zinc deficiency
- Vitamin A, D, K deficiency

➢ Hematopoietic system

- Increase in RBC size
- With folic acid deficiency — hypersegmented neutrophils, reticulocytopenia and hyperplastic bone marrow
- With malnutrition — sideroblastic changes
- Decrease granulocyte mobility and adherence
- Delayed hypersensitivity response to new antigen
- Toxic granulocytosis

- Mild thrombocytopenia
- Hypersplenism

➢ Others

- Alcoholic myopathy

Fetal effects of alcohol

Alcohol crosses the placental barrier and reaches fetus. Thefetal abnormalities may range from selected fetal alcoholeffect (FAE) to full-blown fetal alcohol syndrome (FAS).

The FAS are classified as follows:

1. Facial dysmorphology

2. Prenatal and antenatal growth deficiency

3. CNS involvement, including mental retardationSimilarly still births and spontaneous abortions are morecommon.

Cause of death

Following are the causes of alcohol related deaths

➢ Cirrhosis

➢ Fatty liver

➢ Hepatic failure

➢ Ruptured esophageal varices

➢ Alcoholic cardiomyopathy

➢ Pancreatitis

Withdrawal

Sudden cessation of alcohol by such person results in withdrawalreaction. The withdrawal reaction may manifest as:

- Abstinence syndrome develops 6 to 8 hours after cessation of alcohol and is characterized by tremors, agitation, sweating, nausea, headache and insomnia
- Alcohol hallucinations appear 24 to 36 hours after cessation of alcohol
- Seizures (also called as rum fits) occur 7 to 48 hours after cessation of alcohol. The seizures are clonic-tonic in nature with or without loss of consciousness
- Alcohol ketoacidosis
- Delirium tremens
- Wernicke's and Korsakoff's syndrome.

Delirium tremens

- The disorder appears after 3 to 5 days after cessation of alcohol and the onset is sudden
- The disorder is characterized by clouding of consciousness, disorientation with loss of recent memory.
- The disorder may be associated with vivid hallucinations
  - mostly visual or sometimes auditory in nature
- There is agitation, restlessness, shouting, tremor, ataxia and insomnia
- Autonomic disturbances are common and includes — sweating, fever, tachycardia, hypertension and dilated pupils
- When person is suffering from delirium tremens, he is not held responsible for any act because the he is considered to be of unsound mind during the state.

Wernicke's and Korsakoff's syndrome

It is alcohol-induced persisting amnestic disorder and occursdue to thiamine deficiency. Korsakoff's syndrome presentswith:

- Profound anterograde amnesia (inability to learn new things)
- Milder retrograde amnesia
- Impairment in visuo-spatial, abstract and conceptual reasoning
- Most of the patient demonstrates an acute onset of Korsakoff's syndrome in association with Wernicke's syndrome whereas rest person shows gradual onset.

Wernicke's syndrome

Wernicke's syndrome or encephalopathy is an acute form ofsyndrome characterized by drowsiness, disorientation, amnesia,ataxia, peripheral neuropathy, horizontal nystagmus, anddiplopia due to ophthalmoplegia.

Saturday night paralysis

An intoxicated person, while sitting in a chair, may hanghis arm over a chair and sleep. The resultant hanging ofarm over chair compresses brachial plexus causing paralysisof the muscles. The phenomenon is frequent on the weekendswhere a person may have binge drinking on Saturdayevening with subsequent paralysis.

Adverse effects of Disulfira

Alcohol palimpsest (alcoholic blackouts)

The patchy amnesia, which is not associated with unconsciousness,has been called as alcoholic palimpsest.

Management

- Adequate nutrition and rest
- Vitamin B supplementation
- Judicious use of benzodiazepines to combat withdrawalreaction
- Aversion therapy is meant for gradual weaning away theperson from habit of alcohol consumption. The therapyis instituted after taking care of withdrawal reaction.

Metallic taste
Malase
Abdominal discomfort
Rashes

Disulfiram has been used as an aversion technique. Itis assumed that disulfiram interferes with the oxidativemetabolism of alcohol (inhibits enzyme aldehyde dehydrogenase)with accumulation of aldehyde. Accumulationof aldehyde produces unpleasant symptoms (also calledas **aldehyde syndrome**) whenever person consumesalcohol therefore person prefer not to drink.

- Counseling and psychotherapy
- Rehabilitation.

Autopsy findings

- Smell of alcohol
- Congested conjunctiva
- Rigor mortis may be delayed

- Decomposition may be retarded
- Organs are congested
- Dark fluid blood
- In chronic alcoholics — fatty or cirrhotic liver, cardiomyopathy,pancreatitis, cerebellar degeneration, cerebellaratrophy, atrophy of gastric cells, testicular atrophy,degeneration of seminiferous tubules.

Medicolegal importance

1. As per Sec 85 of IPC, a person is not held responsiblefor his criminal act if at the time of doing the act theperson happens to be intoxicated and provided that thealcohol or intoxication was given to him without hisknowledge or against his will. According to this section,voluntarily drunkenness is no excuse for commission ofcrime.

2. As per Sec 86 of IPC, an act done is not an offenceunless done with a particular knowledge or intent, a personwho does the act in a state of intoxication shall beliable to be dealt with as if he had the same knowledgeas he would have had if he had not been intoxicatedunless the thing which intoxicated him was administeredto him without his knowledge or against his will.

3. According to Sec 510 of IPC — misconduct by adrunken person in public place is punishable withimprisonment up to 24 hours.

4. When person is suffering from delirium tremens, he isnot held responsible for any act because the he is consideredto be of unsound mind during the state.

5. Consent for examination of drunken person — vide supra

6. According to Sec 129-A, under Bombay ProhibitionAct 1949 (BPA) — the prohibition officer or policeofficer, who has reasonable grounds for

believing thatthe person has consumed alcohol, may produce suchperson for medical examination of drunkenness and/orcollection of blood. The medical examiner examines theperson and issues a certificate in the prescribed form“A” containing the details of clinical examination. Theblood samples collected by medical examiner forwardsthe same to Chemical examiner (Regional ForensicScience Laboratory) vide form “B”. The chemical analyzer,after analyzing the sample, forwards his report asprescribed in format “C”.

7. In State of Bombay Vs Balwant Ganpati, the BombayHigh Court held that Article 20 (3) of the Constitution ofIndia was not violated when blood for chemical analysiswas taken under Sec 129-A of the Bombay ProhibitionAct.

8. Sec 84 of the Bombay Prohibition Act 1949 providesthat any person, who is found drunk or drinking in acommon drinking house or is found there present for thepurpose of drinking, shall, on conviction, be punishedwith a fine which may extend to five hundred rupees.

9. Sec 85 of the Bombay Prohibition Act 1949 providesthat any person found drunk and incapable of controllinghimself or behaves in a disorderly manner underthe influence of a drink in any street or thorough fareor public place or in any place to which the public haveor are permitted to have access, shall, on conviction,be punished with imprisonment for a term which mayextend to one to three months and with fine which maybe extend to two hundred to five hundred rupees.

10. Sec 65 and 66 (1) of the Bombay Prohibition Act 1949provides penalty for illegal import, export, manufacture,sale, purchase or transport of an intoxicant withoutproper license, permit or authorization.

11. A medical practitioner may be sued for damages causedwhile treating a patient under intoxicated state or a surgeonmay be held negligent

for death of patient if thesurgeon performs operation under the influence of alcohol.The surgeon can be prosecuted under Sec 304-A ofIPC.

12. Treating the patient under the influence of alcohol isconsidered as infamous conduct (professional misconduct).

13. Many homicides are triggered by the aggressive behaviourengendered by alcohol.

14. Studies have shown that alcohol can be generated inputrefying bodies. Postmortem production of alcoholin decomposing bodies has been attributed to bacterialaction.Such production of alcohol in bodies is referredas endogenous alcohol. It is stated that the upper limitof endogenous production of alcohol16 is 0.15%.

15. Alcohol is highly hydrophilic, so once it enters thesystemic circulation; it is distributed evenly throughouttotal body water. Because women have more body fatcompared with men and fat contains no water, higherpeak alcohol levels are achieved in women than in menof the same weight.

clinical differentiation between ethanol and methanol poisoning			
Alcohol	*Odor*	*Acidosis*	*Visual changes*
Ethanol	Present	+	-
Methanol	Absent	++	+

METHYLALCOHOL

Synonyms: Methanol, wood spirit, wood alcohol, woodnaphtha

Features

Methanol is colorless and volatile liquid

Peculiar nauseating odor

Burning taste

It is obtained from the distillation of wood.

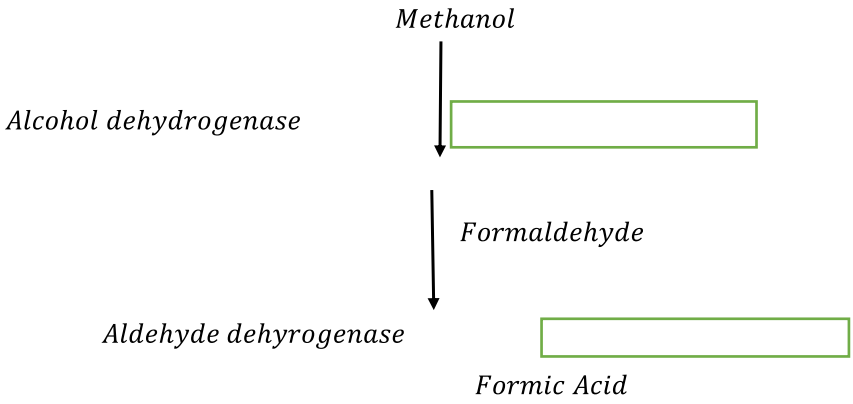
Absorption and metabolism

➢ Methanol is rapidly absorbed from the gastrointestinaltract, through lungs and skin

➢ It is metabolized in liver. It is first metabolized to formaldehydeby the enzyme alcohol dehydrogenase and thenthe formaldehyde is further metabolized into formic acidby the enzyme aldehyde dehydrogenase. Methanol follows zero order kinetics and the half-life isabout 20 to 60 hours.

Mechanism of action

Methanol itself is not toxic but two metabolites formed—formaldehyde and formic acid are highly toxic. Thesecompounds are responsible for causing profound metabolicacidosis and visual defect and blindness.



Fatal dose

60 to 100 ml.

Fatal period

24 to 36 hours.

Clinical features

The clinical features may be delayed for 12 to 24 hours.Patient may present with:

- Nausea
- Vomiting
- Abdominal pain
- Headache
- Breathlessness
- Dizziness
- Vertigo
- Tachycardia
- Hypotension
- Profound metabolic acidosis
- Convulsions
- Delirium
- Coma
- Visual disturbances are common and are attributed tothe toxic effects of formic acid. There may be blurringof vision, or frank blindness. There is retrobulbar degenerationin the form of necrosis of myelinated portion ofoptic nerve and supposed to be cause for visual loss.

- Photophobia
- Constriction of visual fields
- Fundoscopic examination reveals – hyperemia of opticdisk, papilledema, decrease pupillary light reflex, retinaledema.

Diagnosis

- High anion gap acidosis
- Fundoscopic examination — vide supra
- Blood methanol level — more than 50 mg/100 ml indicatessevere poisoning.

Management

- Protect eyes from light
- Maintain respiration, circulation and blood pressure
- Gastric lavage with sodium bicarbonate
- Antidote — ethanol is the specific antidote. Ethanolcompetes with the alcohol dehydrogenase enzyme andprevents metabolism of methanol, which is then excretedunchanged in urine. Ten percent ethanol is administeredthrough a Nasogastric tube; loading dose of 0.7 ml/kg isfollowed by 0.15-ml/kg/hour drip.18 Alternatively, 10%ethanol is administered at a dose of 10 ml/kg intravenouslyover 30 minutes, followed by 1.5 ml/kg/hour, soas to produce and maintain a blood ethanol level of100mg/100 ml.19
- Sodium bicarbonate for acidosis and it also preventsretinal damage.
- Folinic acid (folate therapy) intravenously — it enhancesthe removal of formic acid

- Hemodialysis
- Potassium chloride may be required if hypokalemiadevelops due to alkali therapy
- 4-methyl pyrazole is a specific inhibitor of alcohol dehydrogenaseand retards methanol metabolism. Slow intravenousinfusion of 100 mg has been found to effective.

Autopsy Findings

- Cyanosis
- Stomach and intestine may be hyperemic and inflamed.Patchy submucosal hemorrhages may be noted.
- Pulmonary edema
- Cerebral edema
- Retinal edema
- Necrosis of myelinated portion of optic nerve.

Medicolegal Importance

Most of deaths are due accidental consumption of methanoldue to non-available of regular alcohol or due to adulterationof regular alcohol with methanol.

BARBITURATES

Since the synthesis of diethylbarbituric acid andits introduction into medicine as a sedative andhypnotic agent many other structural analogueshave been prepared and investigated. Fisher &Von Mering introduced Diethyl Barbituric acid into medicine in 1904 under the name of *Venoral*(vera means true) that proved to be an effectivehypnotic without serious side reactions.

The barbiturates were used as sleeping tabletsand sedatives that led to their widespread abusetill 1960s when they were replaced by nonbarbituratehypnotics, such as the benzodiazepines.Unfortunately, barbiturates are still available in themarket either alone or in combination with othersubstances such as amphetamines.

Classification of Barbiturates

The classification of barbiturates into short, intermediateand long acting is arbitrary and may bemisleading. The onset of action is about ¼–½ hourbut the duration varies up to 8 hours or so forPhenobarbitone.

Fatal period→1-2 days.

Pharmacological Action of Barbiturates

Pharmacological action of barbiturates of all typesis the same that is depression of central nervoussystem. However, the structural variations resultin differences in rate of absorption and distribution.

By altering the dose, the degree of depressioncan be altered. Phenobarbitone has a specificdepressant action on cerebral motor cortex thatmakes it a valuable drug for epilepsy. In hypnoticdoses, it has no analgesic action and if prescribedalone in painful conditions, there may beexcitement, restlessness, mental confusion anddelirium. Barbiturates act as synergists withanalgesics and potentiate the action of alcohol.As barbiturates are cumulative drugs, they arecontraindicated in hepatic and renal disorders.After the oral ingestion, the peak concentration inblood and brain of:

1. Medium and short acting barbiturates is after→1-2hours
2. Long acting is after→4-8hrs after ingestion
3. Ultra short acting is after→30seconds to a fewminutes

They are rapidly absorbed from the gastrointestinaltract including

rectum and from thesubcutaneous tissues. They are concentrated inthe liver for a short time and evenly distributed inthe body fluids. Lipid solubility is the primarydeterminant of binding. Ultra short acting is 80%more bound to plasma proteins or stored in thebody fat from which they are subsequently clearedand degraded in the liver. Long acting is only 5%bound to plasma proteins and mainly cleared byurinary excretion. Medium acting are bound morethan the long acting and they are detoxified inliver and tissues and finally excreted quickly. Shortacting barbiturates are also excreted quickly.

Highly lipid soluble barbiturates such asthiopental, methohexal undergo rapidly to thevascular areas of brain and first to the gray matter.Maximum uptake occurs within 30 seconds andsleep may be induced within few minutes. Within30 minutes, there is redistribution to the lessvascular areas of the brain and other tissues.

Other factors that affect the binding capacity arethe dose absorbed and the patient's habit

***Excretion of barbiturates*:** The Barbiturates areremoved from the body by two differentmechanisms:

- Long acting Barbiturates aremainly excreted by the kidneys. As much as 85%of these compounds may be recovered from theurine. Excretion is slow and takes place in severaldays
- The liver mainly metabolizes short actingBarbiturates. Other tissues may also participatein this process. These compounds are notrecovered from the urine if taken in sedative doses.

Mode of action: Barbiturates are cellular,histotoxic agents. They:

- Produce histotoxic ortissue anoxia
- Partially inhibit the cytochromeenzyme system

➢ Toxic action may occurfollowing a large single dose or repeatedmedication in slow excreting Barbiturates.

Signs and Symptoms

The clinical staging of barbiturates has beenclassified in to five stages by 'Sunshine andHackett':

- ➢ Awake, competent and normally sedated
- ➢ Sedated:
  - o reflexes are positive
  - o preferssleep
  - o answers question when aroused
  - o doesnot cerebrate properly
- ➢ Comatose with positivereflexes
- ➢ Comatose and areflexia
- ➢ Comatose;difficulty in respiration and circulation and deathis from respiratory failure.

***Central nervous system*:**

(i) Drowsiness

(ii)Transient period of confusion, excitement, deliriumand hallucinations (iii) Ataxia, vertigo and slurringsleep (iv) Headache (v) Consciousness is depressedin a variable degree; can be assessed byrubbing the clenched fist on patient's sternum.The response of the pupil and peripheral nervereflexes are so erratic that it has a little help inassessing. (Pupils are constricted than dilated interminal hypoxia) (vi) “Rising up” sign (vii) Babinskisign is positive (viii) The degree of consciousnesscan be expressed by the following classification

Grade I: Response to vocal commands

Grade II: Maximum response to minimal painfulstimuli

Grade III: Minimal pain to maximum stimuli

Grade IV: Total unresponsiveness to maximalpainful stimuli

Respiratory system: They directly depress themedullary centers. The rate and depth of respirationis reduced and towards the end Cheyne-Stoke's type of breathing and then death. If comacontinues then infection, pneumonia andpulmonary oedema may develop

Cardiovascular system: Barbiturates exert directtoxic effect on the myocardium and interfere withthe myogenic tone of peripheral arterioles (i) Fallin cardiac output (ii) Permeability of arterioles isincreased leading to transudation and increase inextracellular fluid volume (iii) Cyanosis andhypotension (iv) Weak and rapid pulse (v) Coldand clammy skin.

***Pupils*:** Pupils are little contracted and reactingto light; may be dilated and unequal in terminalasphyxia.

Body temperature: Barbiturates interfere with thecontrol of body temperature; hypothermia isproduced requiring the use of symptomaticmeasures. During recovery the patient maybecome febrile.

***Gastro-intestinal tract*:** Bowel sounds may beabsent during severe poisoning and this is a badsign. When bowel functions return further drugabsorption may take place leading to fluctuatinglevels of consciousness. Incontinence of faecesmay occur

Renal symptoms: Renal functions may failespecially with hypotension and hypothermia.Incontinence of urine may occur and sugar andalbumin may be present in the urine.

Dermatological symptoms: The skin lesionsdevelop in 6% of the cases; are diagnostic andvery commonly present in poisoning with

mediumacting barbiturates. Bullous lesions occur wherethe skin surface rubs the other part of the skinsuch as inner aspects of thigh and pressurebearing areas like hands and feet. Initially thereare slightly raised areas of erythema and later onbullous eruptions are formed. The blister containsserous fluid, the rupture of which leaves a red rawsurface that may be mistaken for burns.It issuggested that bullous formation is either due totoxic effects of the drug or patient is undulysensitive to the drug itself.

Differential diagnosis of bullous eruptionsin Barbiturate poisoning

1. *CO poisoning*: The eruptions are present underthe pressure areas, sacrum, spine, inneraspect of knee and ankle. They are produceddue to impairment of circulation.

2. *Thermal heat:* The blisters due to burns willshow the effect of heat and the hair will beseinged

3. *Pemphigus:* Pemphigus blisters are non-tense,larger and the bullous spreading test is positive

4. *Methaqualone (Mandrax) overdosage*: Thefollowing symptoms are present; hypoproteinemia,gastric bleeding and cardiacarrhythmias.

5. *Glutathimide, meprobamate and tricyclicsantidepressants overdosages*

6. Prolonged contact with petrol and paraffin

Clinical Diagnosis of Barbiturate Poisoning

1. In an unconscious patient, one has to rule outother causes of coma such as:

- *Acutealcoholic poisoning*
  - *(a)* odour of alcohol ispresent in the breathing

- (b) eyes are congestedand pupils dilated
- (ii) *Carbolic acid poisoning*
- (a) the odour is characteristic
- (b) white patchescan be seen on lips and mouth
- (c) carboluria isdiagnostic
- (iii) *Co poisoning*
- *(a)* history ofexposure to the carbon monoxide gas is there
- (b) intermittent convulsions
- (c) cherry red colourof the skin
- (d) carboxyhaemoglobin is presentin the blood
- *(iv) Epileptic coma*
- *(a)* there ishistory of fits
- (b) pupils are fixed and dilated:
- (a) Froth at mouth
- (b) cyanosis
- (v) *Diabeticcoma*:
- (a) gradual onset
- (b) odour of acetone ispresent
- (v) Sugar and acetone is present in theurine
- (vi) *Brain trauma*:
- (a) history ischaracteristic
- (b) injuries and bleeding from thenose
- (c) pulse is rapid

- (d) paralysis may bepresent.

2. Clear history of ingestion of barbiturates

3. Findings of general anaesthesia with lowrespiration and decreased respiratory function

4. Presence or absence of bowel sounds

5. Urine or first stomach wash is to be tested forbarbiturates

6. Blood levels by gas chromatography,calorimetric methods and spectrophotometryare: (a) For long acting—8-10mg% (b) Formedium acting—4-7mg% (c) For short acting—2-4mg% (d) For ultra short acting—0.8-1mg%.

7. ECG findings show inverted and flattened 'T'wave and depressed ST segment

8. **EEG Findings in Barbiturate poisoning**

- *Mild intoxication*: Normal activity is replacedby fast activity in the range of 20-30Hzappearing first in the frontal regions andspreading to the parietal and occipital regionsas intoxication worsens.

- *More severe intoxication*: The fast wavesbecome less regular and interspersed with 3-4Hz slow activity.

- *Still more advanced cases* : There are shortperiods of suppression of all activity, separatedby bursts of slow (delta) waves of variablefrequency

- *Extreme overdoses*: All electrical activityceases in extreme of overdosage of the drug.This is one instance in which a flat EEG cannotbe equated with brain death and the effects arefully reversible unless anoxic damage hassupervened.

Treatment

Mild cases need no treatment but should be keptunder observation. Before treatment in comatosepatients, other causes of coma should beexcluded. The treatment includes the general andspecific measures.

General measures:

- *First part:* Clear the airway by tracheo-bronchialsuction, oxygen inhalation, physiotherapy ofthorax. X-ray of lungs shows evidence of collapse.No prophylactic antibiotics to be given unlessinfection is present

- *Second part:* Gastric lavage and suction: (i) It ismore useful if it is done within 4hours of theingestion of the poison (ii) Done with warm watermixed with potassium permanganate andsuspension of animal charcoal and tannic acid(iii) First sample is to be obtained in plain water(iv) Magnesium sulphate is used for purgation asit minimizes absorption.

- *Third part:* Regular charting of pulse and bloodpressure along: (i) With correction of dehydration(ii) Nor adrenaline 2mg with 500ml of 5% glucose(iii) Intravenous saline drip for correction of shockand hypotension (iv) Patient should be kept warm.

Specific measures:

- No specific antidote isknown

- In prolonged coma with retention ofcarbon dioxide, mechanical respirator andtracheostomy may be considered

- Treatmentof pulmonary oedema by relieving heart failure byaminophylline, digoxin etc. may be considered.500 ml of 10% Mannitol should be given I.V.Fureosemide is used as diuretic

- ***Analeptics:***Analeptics stimulate the central nervous systemespecially respiratory center so they are used inthe treatment of narcotic poisoning. Their use isopposed in barbiturate poisoning due to

thefollowing measures:

- (a) Are generally ineffectivein severe poisoning
- (b) Awakening effect istransient and followed by greater depression
- (c)Leads to cardiac arrhythmias and convulsions;cerebral ischemia and depression and thenirreversible brain damage
- (d) Overall results withouttheir use show a much reduced mortality rate
- (e)10 ml of 0.5% Bemigride (50 mg) and 1ml of 1.5%Amiphenazole (15 mg) are added to the 5%glucose saline drip for two hours at 5 minutesinterval or till consciousness returns whichever isearlier or return of pharyngeal or laryngeal reflexes.If vomiting and muscular twitching is seen thetreatment should be stopped (f) Some useCoramine (Nikethemide) 5ml i.v at 15 minutesinterval and then 10ml at 30 minutes interval tillreflexes return. If muscle twitching are presentstop the treatment.

➢ Picrotoxin can be given2ml intravenously but if muscle twitching arepresent or corneal reflexes return, stop thetreatment

➢ Dialysis and exchange transfusionare at times life saving.

Autopsy Findings

➢ External findings are not characteristic; signsof asphyxia are present and cyanosis of face andnails is seen

➢ Traces of tablets and capsules ofbarbiturates may be found in the mouth,oesophagus and stomach

➢ Mucous membraneof stomach is congested and eroded badly fromthe alkaline attack of drugs like sodium amytalwhich, being the

sodium salt of a weak organicacid, hydrolyses in the stomach. The fundus maybe thickened, granular and haemorrhagic. Thecardia and lower oesophagus may be eroded fromthe reflux and if the victim regurgitates, then black,altered blood may appear at the nose and mouth

- Lungs are congested, oedematous and findingssuggestive of pneumonia may be present. Thecongested lungs in acute barbiturate poisoningare more intense than in any other condition. Thelungs are almost black and the whole venoussystem is engorged with dark, deoxygenated blood

- Petechial haemorrhages are seen in the pleura,pericardium and meninges

- Kidneys arecongested and degenerative changes of thetubules are present

- Brain is oedematous,softening of globus pallidus is seen and multiplepetechial haemorrhages are seen in the whitematter

- All other organs are congested

- Barbiturates blisters: These blisters are foundon the dependent parts of the skin surface, especiallybuttocks, backs of thigh, calve and forearmsFor chemical analysis of viscera, besides theother organs brain is to be preserved as venoral isretained in the brain.

Medicolegal Aspects

- Commonly used for suicide more by young malethan female, next only to organophosphoruscompounds. It is freely prescribed and easilyavailable

- Therapeutic uses: It is used for sleeplessness,anxiety states, epilepsy, strychnine,picrotoxin and cocaine poisoning

- Rarely usedfor homicidal purposes

- Repeated small dosescause addiction that leads to withdrawal symptoms,used for the relief of worries and anxiety ofmodern life

- Automatism: The accidental orsuicidal overdose may lead to the poisoning

- In ordinary doses, it induces natural sleep butoccasionally instead of sleep, there is mentalconfusion. It is likely to happen in those caseswhere insomnia is due to pain and an analgesicis not taken for its relief. In these cases, the useof barbiturates may lead to mental confusion. Asa result, to induce pain patient takes more of thedrug automatically for getting that he has alreadytaken a dose known as barbiturate automatism.In some cases, the patient continues to take thedrug that leads to overdose that is fatal.

However, some workers have a different concept;they feel that confusion and forgetfulness cannotaccount for overwhelming overdose that is foundin these cases. It is possible that there are casesof intentional suicide or alcoholics where confusionis more likely and the action of barbiturates ispotentiated by alcohol. It is now over 40 yearssince 'automatism' was offered as a sociallyacceptable explanation of self-administeredoverdoses of barbiturates. The idea was oftenaccepted without any real evidence that repeatedtherapeutic doses were taken by a patient whodid not remember taking the drug and in somestudies by Dorpat concluded that drugautomatism is a myth.

DELIRIANT POISONS

Deliriant are the poisons acting on the brain and inducingaltered consciousness with confusion, delusions, hallucinationsand agitation.

Examples are given below:

- Datura
- Deadly night shade (Atropa belladonna)
- Henbane (Hyoscamus)
- Indian hemp (Cannabis sativa)
- Cocaine

DATURA

Common name: Thorn apple, Jimson seed

Features

Datura is a wild shrub up to 3 to 5 feet and grows atwaste places

Plant bears dark green oval leaves with trumpet or bellshaped flowers. Two varieties of Datura fastuosa arefound in India:

Datura niger — deep purple color flowers

Datura alba — white color flowers

The plant bears fruits which are spherical and have multiplespikes, thus called as “thorn apple”. Thefruit contains brownish kidney shaped seeds.

Toxic part: all parts are toxic but seeds are more toxic.

Active Principle

Hyoscine (scopolamine) Together referred as

Hyoscyamine beladonna alkaloids

Atropine

Mechanism of Action

The alkaloids competitively inhibit the muscarinic effectsof acetylcholine.

Site of action are at all postganglionic parasympatheticand few postganglionic sympathetic (sweat glands,smooth muscles) innervations.

Majority of the CNS actions are due to blockage ofmuscarinic receptors in the brain viz. vagal stimulation,decrease in heart rate. High doses cause corticalexcitation, restlessness, disorientation, hallucinations anddelirium followed by respiratory depression and coma.

Absorption, Metabolism and Excretion

The alkaloids are quickly absorbed from all mucusmembrane and skin.

The alkaloids are excreted by the kidneys.

Clinical Features

The clinical features are best summarized in classical phrasequoted by Morton Still "**blind as bat, hot as hare, dry asbone, red as beet and mad as hen**". It can also rememberas **D**s. The progression of symptoms with loss of cholinergicfunction is dose related to atropine.

Dose related progression of symptoms	
Dose of atropine	*Symptoms*
0.5 mg	Dry mouth
1 mg	Dilatation of pupils Blurring of vision or Diplopia
2 – 4 mg	Increases cardiac and respiratory activity Elevate blood pressure
5 mg	Temperature elevation Inability to swallow Urine retention
10 mg or more	Restlessness Hyperactivity Agitation Clouded sensorium Disorientation Hallucinations Delirium Coma

Dryness of mouth (dry as bone)

Bitter taste

Difficulty in talking

Dysphagia

Dilated pupils

Diplopia

Difficulty in vision (blurring of vision, blind as bat)

Dry hot skin with flushing (red as beet)

Hyperpyrexia (hot as hare)

Drunken gait (ataxia)

Hyperreflexia

Convulsions

Delirium, hallucinations, agitation, amnesia, incoherence,visual or auditory hallucinations (mad as a hen)

Deficit of recent memory. Remote memory undisturbed

Dysuria

Distention of bladder (retention of urine)

Death.

Management

Treat the patient in quiet and dark environment

Gastric lavage with activated charcoal

Catheterize bladder

Cathartic is given, even if patient is treated 24 hoursafter ingestion because intestinal motility is decreased

Monitor and regulate patient's temperature

Hyperpyrexia — hydration, cold sponging

Agitation can be controlled with judicious use ofdiazepam/lorazepam

Antidote is physostigmine. Intravenous physostigmineis given slowly over 5–10 minutes if hyperthermia,delirium, convulsions, hypertension and arrhythmiasoccurred.

Drug Contraindicated

1. Phenothiazines
2. Antihistamines
3. Morphine
4. Tricyclics
5. Quinidine
6. Disopyramide
7. Procainamide

Autopsy Findings

Signs of asphyxia

Gastrointestinal tract shows inflammation

Seeds or fragments may be found in stomach

Difference between datura and chiliseeds		
Features	*Datura seeds*	*Chilli seeds*
Color	Brownish	Yellowish
Size	Bigger	Smaller
Shape	Kidney shaped	Round
Surface	Pitted	Smooth
Smell	Odorless	Pungent
Taste	Bitter	Pungent
On action	Embryo curves outward	Embryo curves inward towards hilum

Medicolegal Importance

1. Accidental death may occur since datura seeds may bemistaken for chilly seeds.

2. Suicide – rare

3. Homicide

4. Datura seeds are used as stupefying agent to rob people.Robbers usually mix the datura seeds with food or drinksand offer to travelers in train. Once the passengers arestupefied, they robbed them. Thus datura gains popularityas **railroad poison**.

5. Used as love philter or potions. In Anthony and Cleopatra,it was mentioned that Cleopatra employed the daturaextract in her famous wooing of Caesar.

6. Datura seeds are abused. The seeds are mixed with cigaretteand belladonna and smoked as hallucinogen.

7. Datura seed resist putrefaction of body

8. Criminal responsibility: Datura produces temporaryinsanity. Usually, the poison is administered without thevictim's knowledge. Hence the individual is not heldresponsible for his acts under the influence of Datura.

9. Scopolamine is used as truth serum.

Fatal Dose

50 to 100 seeds

10 to 100 mg of atropine

Fatal period: 24 hours

CANNABIS

Botanical name: *Cannabis sativa or Cannabis indica*

Common name: Indian hemp

Features

Cannabis sativa plant grows all over India however; thecultivation is

restricted by government.

The plant is dioecious, i.e. the sexes are separate. Thefemale plant is taller and grows about 4 to 6 meter andbears luxurious foliage than male counterpart.

Nabilone is a synthetic cannabinoid and possess antiemeticproperties. It is found to be useful in patientsreceiving cancer chemotherapy.

Preparations of Cannabis

The various preparations of Cannabis sativa, which is used,are as follows:

1. Bhang: also called as sidhi, patti, sabji. Bhang is madefrom dried leaves of plant pressed into cakes.

2. Ganja: is derived from flowering tops.

3. Charas: also known as hashish or hash and is derivedfrom resinous exudates of plant

4. Majun: a sweet prepared with any of the above preparationadded.

5. Marijuana: this term is used in America and many textsconsidered it as synonymous with ganja. It is preparedfrom the leaves and flowering tops of the plant.

Active Principle

Three active preparations are abundantly found and include:

Cannabinol

Cannabidiol

Several isomers of tetrahydrocannabional. The isomerresponsible for most of the characteristic effect of cannabisis l-Δ^9- tetrahydrocannabional (Δ^9- THC).

Fatal Dose

Charas — 2 gm/kg body weight

Ganja — 8 gm/kg body weight

Bhang — 10 gm/kg body weight

Fatal period: about 12 hours

Mechanism of Action

Cannabis act at cannabinoid receptors. Two types ofcannabinoid receptors have been identified and are CB1and CB2.

CB1 — widely distributed with highest concentration inbrain neurons

CB2 — found in cells of immune system, spleen, tonsilsand immune cells.

Absorption, Metabolism and Excretion

Cannabis (Δ^9- THC) is absorbed from the gastrointestinaltract and as smoke or vapor from the respiratory tract. Itis slowly absorbed from subcutaneous or intramuscularinjection.

Δ^9- THC is rapidly converted by liver microsomes intoan active metabolite 11-hydroxy- Δ^9- THC. 11-hydroxy-Δ^9- THC is converted into an inactive metabolite andexcreted in urine, feces and bile.

Clinical Features

Subjective perception of relaxation and euphoria

Some impairment in thinking, concentration and perceptualand psychomotor functions occur

Higher doses causes hallucinations, sedation and at timesdysphoria with unpleasant sensation

Excitement

Impulsive ideas/stray ideas

Size of objects and distance is distorted

Recent memory and selective attention is impaired

Changes in perception of color, shape and time

Increase in appetite

Nausea

Dry mouth

Tachycardia

Palpitations

Hypotension

Angina may be precipitated by ganja smoking in personswith coronary insufficiency.

Differential Diagnosis

1. Cocaine intoxication

2. Amphetamine intoxication

3. Sedative

4. Tricyclic antidepressants

5. Panic attacks

Management

Gut decontamination

Benzodiazepines for paranoia

Haloperidol for acute psychotic state

Supportive measures.

Autopsy Findings

Signs of asphyxia

Unabsorbed bhang may be identified in the stomach.

Medicolegal Importance

1. Drug of abuse

2. Acute intoxication causes impairment in motor skillsand judgment, affects vision and perception of time andspace. Therefore, it may be dangerous to drive a vehicleunder the influence of cannabis.

3. A case is reported where a young adult male died afterconsuming bhang, who was suffering from heart ailment.

4. Cannabis is known to induce suicidal ideation

5. Run amok: under the influence of the substance, a personget frenzy and goes on killing other persons who comesin his way until the homicidal tendency last. Thereafter,the person commits suicide or surrender.

COCAINE

Features

Cocaine is an alkaloid derived from plant Erythroxyloncoca.

It is produced as a salt (cocaine hydrochloride) or as analkaloid known as freebase or crack.

The free base (crack) is a colorless, odorless, transparent,crystalline substance. It makes cracking sound whenheated, thus known as crack.

Cocaine hydrochloride is a white powder. It is usuallyadulterated with other substances such as caffeine,amphetamine, strychnine etc. When it is adulterated withheroin, it is called as speed ball.

Routes of Administration

1. Chewing – coca leaves
2. Pyrolysis (smoking)
3. Snorting
4. Intravenous injection
5. Ingestion

Mechanism of Action

Cocaine is CNS stimulant. CNS stimulant effects andeuphoria are mediated through inhibition of dopaminereuptake in the nucleus accumbens. However, in chroniccocaine user, it causes dopamine depletion and impairmentof dopaminergic function in the brain.

Cocaine potentates' nor-epinephrine and epinephrine.Cocaine blocks the reuptake of neurotransmitters at thesynapse due to which there is increase in the concentrationof nor-epinephrine and epinephrine. The increase inconcentration of nor-epinephrine and epinephrine causessympathomimetic effects such as tachycardia, hypertension,hyperthermia, diaphoresis, mydriasis and vasoconstriction.

Absorption, Metabolism and Excretion

Cocaine is well absorbed from oral, nasal and respiratorysite.

With intravenous injection or inhalation, the onset effectsare rapid with peak level achieved in 3 to 5 minutes. Thehalf-life is approximately 1 hour.

It is metabolized by liver and plasma cholinesterase tobenzoylecgonine, ecgonine methyl ester and ecgonine.

The metabolites are excreted through urine.

Fatal Dose

Oral — 50 mg

Mucosal — 100 mg

Fatal period: few minutes — few hours

Clinical Features

Acute poisoning

Anxiety

Agitation

Restlessness

Tremors

Hyperthermia

Tachycardia

Hypertension

Convulsions

Hyperreflexia

Psychosis

Auditory or visual hallucinations

Altered tactile sensation: the person feel that somesmall insects are crawling on his skin. This is knownas **Magnan's symptom** or **cocaine bugs** (formication).

Mydriasis

Pulmonary edema

Rhabdomyolysis

Intestinal ischemia/abdominal pain/colitis

Blindness due to occlusion of retinal artery as a consequenceof vasoconstriction.

Myocardial ischemia/infarction due to coronary arteryvasospasm

Intracranial hemorrhage such as subarachnoid hemorrhageor intracerebral hemorrhage or infarction may occur.

Management

Hyperthermia managed by cooling blanket/ ice watersponging/ice water bath

Convulsions — diazepam/lorazepam

Tachycardia — beta-blockers

Chest pain — calcium channel blockers, nitrates

Ventricular arrhythmias — lidocaine

Supportive measures.

Autopsy Findings

Signs of asphyxia

Nasal septum ulceration and perforation may be notedin chronic snorting abuser.

There may be multiple scar marks at injection site inchronic abuser

There may be infective endocarditis as sequelae to septicinjection site and practice.

Hepatic necrosis may be present especially in coca pastesmokers.

Heart may show evidence of myocardial infarction.Microscopy shows lymphocytic infiltrate, coagulativenecrosis of myocardial fibers

and intimal proliferation.

Samples to be Preserved

1. Blood

2. Brain

3. Skin from injection site

4. Swab from nasal mucosa

Medicolegal Importance

1. Drug of abuse

2. Body may get decompose rapidly

3. Accidental deaths are common due to overdose or adulteration

4. Provoke the users for violent behaviour. Acute intoxicationmay cause person to be aggressive and paranoid.

5. Prostitutes may place cocaine solution into vagina toproduce local constriction and causes intoxication.

6. **Body packer syndrome**: persons engaged in smugglingof cocaine fill the drug in balloon or condom or polythenebag and swallowed to conceal the contraband. Thisact is called as body packing. The packets may causeintestinal obstruction.Sometime, sudden death maybe caused due to rupture of the bag or condom withingastrointestinal tract.

7. **Body stuffer syndrome**: in this syndrome, the personwho smuggled the cocaine, on the verge of being arrestedfor possessing cocaine, swallows the drug to conceal theevidence. This act causes cocaine poisoning.

SPINAL POISONS

This group of poisons mainly acts on the spinalcord, the cerebral symptoms being either slightor absent. The stimulation of spinal cord resultsin spasms and convulsions, while depression causesparalysis and loss of sensation. Nux vomicais a spinal stimulant whereas and Gelsemium isspinal depressant.

Examples of spinal poisons are:

- Strychnos nux vomica
- Physostigmatis semina or calabar bean
- Physostigmine (eserine)
- Gelsemium sempervirens or jasmine

Strychnine

Botanical name: *Strychnosnux vomica*

Common name: Kuchila, dog buttons

Physical features

Belongs to family Loganiaceae

A vine found in South India

Plant bears oval dark green leaves

Fruits are globular and contain disk-shaped seeds. Theseeds are round, disk shaped, concave on one side andconvex on the other side. Seeds are ash gray in colorand covered with silky fibers. The seeds are about 2.5cm in diameter and 5 mm in thickness. The pericarp ofseed is

tough.

Uses

- Rodenticide
- For killing stray dogs
- As folk medicine
- Arrow poison for hunting

Toxic parts of plant

- Leaves
- Fruits and seed
- Root and stem
- Bark.

Toxic principles

- Seed contains two active principle namely:
- Strychnine
- Brucine
- Root, stem, bark and leaves contains:
- Brucine as toxic principle
- Lagonin as glycoside
- Strychnine is an odorless white crystalline prism thatmelts at 275 to 285oC with decomposition. It is verybitter in taste and more powerful than brucine.

Mechanism of action

➢ Strychnine antagonizes the inhibitory neurotransmitteramino-acid glycine at postsynaptic receptors.

➢ Inhibitory glycine receptors are abundant in the spinalcord and brain stem where they are mainly involvedin regulation of motor functions. When inhibitors areblocked, ongoing neuronal excitability is increased andsensory stimuli produce exaggerated reflex effects thusproducing powerful muscle contractions.

➢ Glycine receptors in higher brain centers such as substantianigra, neostriatum and hippocampus are commonlyinsensitive to strychnine, explaining why strychninesymptoms are largely spinal in origin.

Absorption, metabolism and excretion

Strychnine is well absorbed from gastrointestinal mucosaand nasal mucosa but not through the skin. It is metabolizedin liver. In non-fatal human poisoning, strychnine disappearancefollowed first order kinetics with a half-life of 10 hoursto 16 hours. It is excreted mainly by kidneys with traces inbile, milk and saliva.

Clinical features

➢ If seeds are swallowed uncrushed, the hard pericarp resistdigestion and seeds are passed in feces without any poisonoussymptoms.

➢ With crushed seeds, symptoms begin to appear within15 to 30 minutes.

➢ Bitter taste in mouth

➢ Sense of uneasiness, restlessness, fear and anxiety

➢ Increase difficulty in breathing and swallowing

➢ Muscle twitching and spasm of muscle followed by

convulsions.The convulsions last for 30 seconds to 2 minuteand are precipitated by slightest stimuli such as suddennoise, a current of air or gentle touching of patient. Theconvulsions are first clonic but eventually becomes tonic.In between the convulsions, the muscles are completelyrelaxed and it is an important diagnostic feature.

- The convulsions are more marked in anti-gravity musclesand body arches in hyperextension position and lies onheel and head. This position of body is known as **opisthotonos**.It is most common position. However, at time,the body may bend forward and the condition is calledas **emprosthotonos**. If body bends side wise (i.e. lateralbending), the condition is called as **pleurothotonos**.

- Contraction of the muscles of face causes widening ofthe angle of mouth with creases appearing around eyelids.This condition is known as **risus sardonicus**. Alsocalled as **sardonic smile** due to grimacing that occurreddue to muscle contraction of face.

- There is difficulty in breathing during convulsions due tocontraction of chest muscles and diaphragm.

- Patient remains conscious and maintains clear sensoriumduring and between convulsions.

- There may be frothing at mouth and pupils are dilated.

- Prognosis is good if interval for appearance of convulsionsincreases and period of convulsion decreases.Prognosis is bad if reverse occurs, i.e. when convulsionsappear rapidly and last longer.

Complications

- Hypoxia
- Hyperthermia
- Rhabdomyolysis

- Metabolic acidosis/ lactic acidosis

Causes of death

- Medullary paralysis due to hypoxia
- Respiratory failure due to spasm of respiratory muscles

Differential diagnosis

Showing difference betweenstrychnine and tetanus		
Features	*Strychnine*	*Tetanus*
History of onset	Poisoning sudden	Injury Gradual
Fever	Not usual	Usual
Convulsions	All muscles are involved at the time of convulsions	All muscles are not affected at same time
Lock jaw	Absent	Present
Muscle	Relaxed in between convulsions	Are stiff and not fully relaxed
Fatal period	1 – 2 hour	>24 hours
Laboratory findings	Chemical test for strychnine positive	No poison Bacteria present on microbiological investigation

1. Tetanus
2. Rabies
3. Meningitis
4. Cocaine intoxication
5. Phenothiazine poisoning

6. Phencyclidine

7. Hysteria

Fatal dose

- Seeds — 1 to 2 crushed seeds
- Strychnine — 50 to 100 mg

Fatal period

- 1 to 2 hour

Management

- Patient should be managed in calm environment.
- Emesis is contraindicated as the procedure may precipitateconvulsions. However, cautiously gastric lavage maybe carried out after protecting air way. Activated charcoalshould be administered and is considered as effective.
- Management of convulsions is important and can betreated by lorazepam or diazepam. If benzodiazepinesare ineffective, short acting barbiturate can be administered.Intractable convulsions may need muscle relaxantssuch as pancuronium.
- Rest symptomatic measures.

Autopsy findings

- Rigor mortis— appear and disappear early
- Postmortem caloricity
- Signs of asphyxia
- Froth at mouth
- Serosal surface may show hemorrhagic areas

- Occasionally muscle may show hemorrhages or evidenceof rupture
- Spinal cord is congested. Microscopy shows multiplehemorrhages in anterior and posterior horns with ringhemorrhages around the capillaries. Neurons may showchromatolysis.
- Organs are congested.

Samples to be preserved

- Routine viscera
- Blood
- Spinal cord

Medicolegal importance

- Accidental poisoning may occur in children who maychew the seeds out of curiosity.
- Accidental poisoning may occur in adults due to consumptionof folk/indigenous medicinal preparation.
- Accidental poisoning in adults may occur due to strychnineconsumption as it is considered as aphrodisiac.
- Homicide rare due to bitter taste and dramatic appearanceof symptoms, however cases have been reported.
- Used to kill cattle.

PERIPHERAL NERVE POISONS

Examples are:

- Curare
- Conium maculatum

CURARE

Curare is found in various species of strychnous plants andchondrodendrontomentosum plants.

Uses: Skeletal muscle relaxant

Active Principles

➢ Curarine

Mechanism of Action

➢ The action is on peripheral muscles. Curarine blocks thepostsynaptic nicotinic acetylcholine receptors at myoneuraljunction and thus causes paralysis of skeletal musclewithout affecting consciousness.

➢ Initially the small muscles of the eye, fingers and toes areaffected followed by paralysis of neck, upper and lowerlimbs and finally diaphragm and intercostal muscles areparalyzed causing respiratory failure.

Absorption, Metabolism and Excretion

Curare is slowly absorbed from GIT. Absorption is rapidwhen given by subcutaneous or intramuscular route. Curarineis metabolized by liver and excreted into urine.

Clinical Features

- Flaccidity of muscle
- Headache
- Hypotension
- Vertigo
- Mydriasis
- Blurring of vision
- Hyperthermia
- Convulsions
- Death is due to paralysis of respiratory muscle.

Complications

- Malignant hyperthermia
- Rhabdomyolysis
- Fatal dose: 30 to 60 mg
- Fatal period: 1 to 2 hours

Management

- Air way maintenance and respiration
- Atropine
- Neostigmine (antagonizes block).

Autopsy Findings

- Signs of asphyxia
- Organs are congested.

Medicolegal Importance

- Arrow poison
- Rarely used for homicide
- Anesthetic deaths due to overdose
- Derivative of curare in combination with barbiturate isused for euthanasia.

CONIUM MACULATUM

Botanical name: *Conium maculatum*

Common names: Poison hemlock, Socrates poison, commonhemlock, spotted hemlock

Toxic parts of plant: all parts of plant

Toxic Principles

- Coniine (alkaloid)
- Gamma-coniceine (alkaloid)

Mechanism of Action

Mechanism of action is two fold and the alkaloid act at:

- Neuromuscular junction as a non-depolarising blockers(similar to curare) and causes flaccid paralysis
- Autonomic ganglia producing nicotinic effects such assalivation, mydriasis, tachycardia followed by bradycardia.
- Fatal dose: 60 mg of coniine
- Fatal period: 1 to 3 hours

Clinical Features

- Nausea, vomiting

- Abdominal pain
- Salivation
- Tremors
- Sweating
- Convulsions
- Mydriasis
- Tachycardia followed by bradycardia
- Motor paralysis
- Hypotension
- Coma
- Respiratory failure.

Complications

- Rhabdomyolysis
- Acute renal failure
- Respiratory failure.

Management

- Air way maintenance and respiratory support
- GIT decontamination with activated charcoal
- Benzodiazepines for convulsions.

Autopsy Findings

- Signs of asphyxia
- Mousy odor (due to free coniine in plant)
- Organs are congested.

Medicolegal Importance

- In ancient times, poison hemlock was used for execution.Socrates was executed by giving poison hemlock.
- Poisoning is rare in India.
- Not popular suicidal agent
- Accidental poisoning may occur.

These are the agents which cause asphyxia.

Classification

Asphyxiants are classified as:

1. Simple asphyxiants
2. Respiratory irritants
3. Systemic asphyxiants
4. Volatile compounds

ASPHYXIANTS

Simple Asphyxiants

These are the inert gases and when these gases are breathedin high concentration, they act mechanically by displacingor excluding oxygen.

Examples are:

- Carbon monoxide
- Nitrogen
- Butane
- Ethane
- Methane.

Respiratory irritants

These agents, when inhaled cause destruction of the respiratorytract or lung or both and cause inflammatory changes.

Examples are:

- Ammonia
- Chlorine
- Formaldehyde
- Hydrogen sulphide
- Smoke
- Methyl isocyanate.

Systemic Asphyxiants

These agents cause systemic toxicity.

Examples are:

- Carbon monoxide
- Hydrogen cyanide gas
- Smoke.

Volatile compounds

These agents act as anesthetic agents.

Examples are:

- Aliphatic hydrocarbons
- Halogenated hydrocarbons
- Aromatic hydrocarbons.

CARBON MONOXIDE

Properties

- It is colorless, odorless, tasteless, non-irritant gas
- It is lighter than air
- Soluble in water
- Burns with blue flame.

Sources

1. Fire

2. Produced whenever there is incomplete combustion ofcarbon/fuel such as wood, charcoal, kerosene etc.

3. Automobile exhaust

4. Tobacco smoke

5. Generates in gun-powder, mines and detonation ofexplosive.

Mechanism of action

➢ Carbon monoxide (CO) has 230 to 270 times greateraffinity for hemoglobin than that of oxygen. Thus, itdisplaces oxygen from its combination with hemoglobinand forms a relatively stable compound known as **carboxyhemoglobin**.Formation of carboxyhemoglobinresults in reduced arterial oxygen content and causestissue anoxia.

➢ Furthermore, CO causes a leftward shift of the oxyhemoglobindissociation curve affecting the offloading ofoxygen from hemoglobin to the tissue.

➢ The resultant effect is decreased ability of blood to carryoxygen and deliver to the tissues.

Showing percentage ofcarboxyhemoglobin and signs and symptomsproduced in carbon monoxide poisoning

% of CoHb	*Clinical Features*
0 – 10	No symptoms
10 – 20	Tightness across forehead Slight headache Dilatation of cutaneous blood vessels
20 – 30	Headache Throbbing in temples
30 – 40	Severe headache Weakness Dizziness Dimness of vision Nausea and vomiting Collapse
40 – 50	Same as above plus greater possibility of collapse or syncope Increased respiration and pulse
50 – 60	Syncope Coma with intermittent convulsions Cheyne-strokes respiration
60 – 70	Depressed cardiac function and respiration Possible death
70 – 80	Weak pulse and slowed respiration Respiratory failure and death

Clinical features

➢ ***Acute poisoning***

o The clinical features progresses as the percentage ofsaturation of CO increases in the blood. Initial symptomsare breathlessness, mild headache and as percentageincreases, there is impairment of higher intellectualfunctions.

o Several types of skin lesions may be produced in carbonmonoxide poisoning. The lesions vary in degree fromareas of erythema and edema to marked blister and bullaformation. The bullae are geographic in shape and maybe few or more in number and are not necessarily locatedat pressure sites.

o There may be development of metabolic acidosis andrhabdomyolysis.

o Eye: There may be venous engorgement, tortuosity, diskedema and flame-shaped retinal hemorrhages.

o Carbon monoxide has direct cardiac toxicity and inducesatrial and ventricular arrhythmias. Angina or myocardialinfarction may be induced or precipitated.

Differential diagnosis

1. Alcohol intoxication
2. Cerebrovascular episode (CVE)
3. Meningitis/encephalitis
4. Epilepsy

Complication after acute exposure

1. Dementia

2. Blindness

3. Amnesia

➢ ***Chronic poisoning***

- Headache
- Confusion
- Dizziness
- Weakness
- Paresthesia
- Visual disturbances
- Hypertension
- Hypertension
- Hyperthermia
- Palpitation
- Aggravation of angina
- Parkinsonism
- Incontinence
- Ataxia
- Polycythemia

Management

➢ Remove patient from vitiated environment

➢ Oxygen administration. Hyperbaric oxygen is consideredto be beneficial

➢ Convulsions should be treated with benzodiazepines

- Physical activity curtailed — to minimize the incidenceof cerebral demyelination
- Mannitol for cerebral edema.

Autopsy findings

- Pinkish coloration of skin and mucosa
- Pinkish colorpostmortem lividity
- Blisters may present on dependent parts such as calves,buttocks, wrist, knee
- Froth at mouth and nostrils
- Features of asphyxia
- Blood — pinkish red and fluid
- Internal organs — pinkish red
- Pulmonary edema
- Petechial hemorrhages in white matter and putamen
- Necrosis of basal ganglia especially globus pallidus.

Medicolegal importance

- Accidental death
- Suicide — it is popular agent in western countries tocommit suicide. The victim usually uses motor vehicleexhaust for the act of self-destruction.

CARBON DIOXIDE

Properties

- Atmosphere contains about 0.4 percent

- Heavy, odorless, colorless gas
- In solid form called as “dry snow”

Sources

1. Combustion: produced by complete combustion of carboncontaining compounds

2. Respiration

3. Decomposition of organic matter

4. Mine explosions

5. Manholes, wells, soil or cellar

Clinical Features

The clinical features vary with concentration of carbon dioxidegas inhalation.

Management

- Administer oxygen
- Supportive measures.

Autopsy findings

- Cyanosis
- Froth at mouth and nostrils
- Congestion
- Petechial hemorrhages
- Dark fluid blood
- Venous engorgement

Medicolegal importance

Accidental deaths — worker working in deep well, dampenpit, overcrowding in ill-ventilated room etc.

HYDROGEN SULFIDE

Properties

- Colorless, heavy, inflammable gas
- Rotten egg like smell
- Hydrogen sulfide, carbon dioxide and methane gases areformed in severs and called **sever gas**.

Sources

1. Formed during decomposition of organic substances containingsulfur

2. Found in sewer, cesspool, privy vaults.

Mechanism of action

It acts as a cytochrome oxidase poison, blocking the electrontransport chain that catalyzes the reduction of molecularoxygen to water.

- ***Fatal Dose***
  - 1000 to 3000 p.p.m.

Clinical features

- Breathlessness
- Cough
- Giddiness
- Nausea
- Lacrimation

- Photophobia
- Keratoconjunctivitis
- Muscular weakness
- CNS depression
- Delirium
- Convulsions
- Coma
- Death.

Management

- Shift the patient from vitiated environment
- Protect airway
- Oxygen administration
- Intravenous fluid and vasopressors
- Treat pulmonary edema with furosemide
- Supportive measures.

Autopsy findings

- Signs of asphyxia
- Rotten egg like odor
- Postmortem lividity is greenish blue due to partial formationof sulfhemoglobin.
- Blood and viscera — greenish —purple.
- Petechial hemorrhage.
- Evidence of respiratory irritation in form of erosion ofmucosa.

- Lungs are congested and edematous.
- Liver — shows greenish-gray or ashen tint due to postmortemcombination of hydrogen sulfide and methemoglobin.
- Urine thiosulfate and serum sulfhemoglobin levels canbe determined.

Medicolegal importance

- Accidental deaths
- Occupational hazard for workers working in sewer tanksetc.
- Putrefaction is rapid.

METHYL ISOCYANATE (MIC)

Properties

- Colorless liquid with pungent sweetish smell below 27oC
- Becomes gas at 39oC
- Highly volatile and inflammable

Uses

1. Manufacture of carbamate pesticide — carbaryl
2. Manufacture of adhesive
3. Manufacture of plastics

Mechanism of action

Causes carbamylation at biochemical level

Clinical features

- *Inhalation*
  - Lacrimation

- Irritation of eyes
- Blurring of vision
- Photophobia
- Corneal ulceration
- Cough
- Dyspnea
- Chest pain
- Hemoptysis
- Pulmonary edema
- Convulsions
- Coma
- Death.

➢ *Dermal*

- Erythema
- Vesication.

Management

➢ Decontamination of skin and eyes

➢ Oxygen administration

➢ Bronchodilators and steroids

➢ Supportive

Autopsy findings

➢ Signs of asphyxia

➢ Pulmonary edema

- Cerebral edema
- Visceral congestion
- In delayed death — degeneration of brain, heart, lung,liver and kidneys.

Medicolegal importance

- Accidental death
- MIC was involved in Bhopal tragedy that occurred in1984 causing more than 2000 deaths

FOOD POISONING

Food poisoning means illness resulting from ingestionof food with microbial or non-microbial contamination.In other words, it is an acute gastroenteritis caused byingestion of food or drink contaminated with either livingbacteria or their toxins or chemical substances andpoisons derived from plants and animals.

Food poisoning infections range from trivial intestinaldisorders to life-endangering bacterial invasions of thebloodstream.

Causes

- Poisoning by microbial contamination
- Poisoning by non-microbial contamination

Microbial contamination

I. Bacteria

- Bacillus cereus
- Staphylococcus aureus
- Salmonella group (except *S. typhi*)
- Shigella
- Vibrio
- Escherichia coli
- Campylobacter
- Yersinia enterocolitics
- Listeria monocytogenes
- Clostridium.

II. Viruses

- Rotavirus
- Adenovirus
- Parvovirus.

III. Protozoa

- Giardia lamblia.

IV. Fungi

- Aspergillus flavus
- Fusarium roseum.

Non-microbial contamination

I. Vegetable origin

- Lathyrus sativus
- Mushrooms
- Aggemone mexicana

II. Animal sources

- Poisonous fish like shellfish, scombroid fish etc.
- Mussel.

III. Chemicals

- Flavouring agents
- Coloring agents
- Preservatives.

1. Mycotoxins are toxic metabolites produced by molds.The disease caused by these metabolites, either by contactor by inadverant ingestion of

the toxin when presentin food or feeds, is called mycotoxicoses. Mycotoxicosesdiffer from mycoses. Mycoses means generalized invasionof living tissue by an actively growing fungus.

2. Food additive: Food and Agriculture Organization ofUnited Nations (FAO) defines food additive as "a nonnutritivesubstance added intentionally to food, generallyin small quantities to improve its appearance, texture orstorage properties".

3. Food poisoning outbreaks are associated with meat,sweat-marts (custards, cream confectionery, puddings,cakes etc), fish, egg and egg products, milk and milkproducts, vegetables and fruits.

Summarizing bacteria and the symptoms produced by them

Bacteria	*Symptoms*	*Food sources*
A. Incubation period: 1–6 hours		
S. aureus	Nausea, vomiting, diarrhea	Poultry, salads, cream, pastries
B. cereus	Nausea, vomiting, diarrhea	Meat, fried rice
B. Incubation period: 8–16 hours		
C. perfrigens	Abdominal cramps, diarrhea	Beef, poultry, legume, gravies
C. Incubation period:		
> 16 hours		
V. cholerae	Watery diarrhea	Shell fish, water, salad, cheese, meats
E. coli	Inflammatory diarrhea	Beef, poultry, egg, dairy products
Salmonella sapps	Dysentery	Egg, salad, lettuce, raw vegetables
Shigella sapps	Dysentery	Molluscs, crustaceans
V. Parahemolyticus		

Food poisoning by bacteria

Food-borne bacterial toxins causes illness by two way:

- Ingestion of food contaminated with microbial toxins or
- Ingestion of bacteria in contaminated food, whichleads to intestinal colonization of bacteria and endogenousproduction of toxins.

Some bacteria act through both mechanisms (describedabove) for example, B. cereus, C. botulinum, V. cholerae etc.

Food poisoning due to C. perfringens has a slightlylonger incubation period (8 – 14 hours) and results fromthe survival of heat resistant spores in an inadequatelycooked food.

Differential diagnosis

1. Ulcerative colitis
2. Amebiasis
3. Heavy metal poisoning
4. Irritable bowel syndrome

Autopsy findings

- In most of the cases (except botulism) there is gastrointestinalcongestion, in some cases; there may be ulcerationof mucosa or findings of gastro-enteritis.
- Abdominal organs are congested.

Samples to be preserved

1. Stool

2. Vomitus

3. Remnants of food

Medicolegal importance

1. Mass food poisoning may occur while having food atcommon eatery/function.

2. Public health authorities have to be informed.

Psychoactive drugs are agents capable of altering the mentalfunctioning of a person

There are four patterns of drug abuse:

1. Acute intoxication

2. Substance dependence

3. Harmful use

4. Withdrawal state

DRUG DEPENDENCE AND ABUSE

Acute Intoxication

Acute intoxication is a transient condition following theadministration of alcohol or other psychoactive substances,resulting in disturbances in the level of consciousness, cognition,perception, affect or behavior or other psycho-physiologicalfunctions and responses.

Substance Dependence

Substance dependence is a cluster of physiological, behavioraland cognitive phenomenon in which the use of a substance or aclass of substances taken on a much higher priority for a givenindividuals than other behaviors that once had greater value.

Club drugs and their street names	
Drugs	*Street Names*
MDMA (3,4-methylenedioxymethamphetamine)	Ecstasy, X, M, rolls
GHB (gamma-hydroxybutyrate)	G, liquid ecstasy, soap
Flunitrazepam (Rohypnol)	Mexican Valium, circles
Ketamine (Ketalar)	K, Special K, Jet

Harmful Use

Harmful use is characterized by:

1. Continued drug use despite awareness of harmful medicaland/or social effects of drug being used and/or

2. A pattern of physical hazardous use of drug.

Substance Withdrawal

Substance withdrawal is a condition where symptoms resultsfrom the

cessation of substance of abuse accompanied by amaladaptive behavior change.

Addiction

Addiction denotes a chronic disorder characterized by compulsiveuse of drugs resulting in physical, psychological andsocial harm and continued use despite evidence of that harm.

Classification

Psychoactive substances or drugs of dependence and abuseare classified as:

1. Ethanol
2. Tobacco
3. Opioids
4. Cannabis
5. Cocaine
6. Amphetamines and others
7. Hallucinogens — LSD, phencyclidine
8. Tranquillizers, sedatives and hypnotics — barbiturates,benzodiazepines etc.
9. Inhalants — solvents, ethers etc.
10. Miscellaneous: caffeine, Datura, analgesics etc.

Alcohol remains the preferred agent over the years, howevermany newer psychoactive drugs have joined it. Thesedrugs are called as recreational drugs and includes marijuana(ganja), LSD, amphetamine etc. The recent trend identifiesclub drugs as new sojourn. These are referred as

club drugsbecause of their prevalence at dance parties, rave partiesand nightclubs.

Mechanism of Drugs of Misuse

- Mimicking or substituting for natural transmitters as:
  - Opioids — endorphin/encephalin
  - Alcohol — GABA-A/endorphin
  - Benzodiazepines — GABA-A
  - Cannabis — Anandamide ('ananda' is a Sanskritword and it means bliss)
  - LSD — 5-HT.
- Increasing endogenous transmitter release
  - Cocaine — dopamine
  - Amphetamine — dopamine
  - Ecstasy — 5-HT/dopamine
  - Solvents — noradrenalin
- Blocking natural transmitters
  - Alcohol — glutamate
  - Barbiturates — glutamate.

Complications of Drug Abuse

- ***Medical complications***
  - Malnutrition
  - Self-neglect
  - Dental decay

- Thrombophlebitis
- Pulmonary embolism
- Cellulites/abscess/septicemia
- Transmission of HIV/hepatitis B and C
- Psychiatric complications
- Death.

➢ ***Social complications***

- Social aloofness/isolation
- Antisocial behavior
- Theft/violence/crime.

Examination of a Person with Substance Abuse

➢ Record Blood pressure
➢ Pulse rate
➢ Temperature
➢ Pupil size
➢ Pupillary reaction to light
➢ Conscious level – lethargy, stupor/coma
➢ Restlessness/agitation
➢ Glasgow coma scale
➢ Orientation in time/place/person
➢ Speech
➢ Pallor
➢ Flushing

- Tremors at rest
- Yawning
- Lacrimation
- Rhinorrhea
- Gooseflesh
- Bowel sounds
- Presence of needle tracks
- Disordered perception
- Coordination
- Gait
- Romberg's test
- Auscultation of the chest
- Other systemic examination.

Autopsy Findings

- Clothes may contain drug, packets/chilam/needle-syringe/tourniquet etc.
- Emaciation
- Injection marks in intravenous drug abuser, there may bemultiple fresh injection marks and/or old linear track scarswith fibrosis of veins often on the antecubital fossae, forearms,and dorsa of hands. Phlebitis of veins may be noted.
- Punctate areas of black discoloration (soot tattooing)may be noted and are caused by deposition of carbonaceousmaterials along the tract of needle. Suchtattooing is called "**turkey skin**" as it resembles theplucked

bird.

- Injection marks in subcutaneous drug user are in formof multiple circumscribed scars and are known as **skinpoppers**.
- Multiple abscesses may be present. Regional lymphnodes are enlarged.
- Nasal septum ulceration and perforation may be notedin chronic cocaine snorting abuser.
- Gastrointestinal tract may contain pills or capsules. Theremay be packets or containers as in the body packers andbody stuffers.
- There may be bowel ischemia or gangrene as in cocaineintoxication.
- Rhabdomyolysis.
- Pulmonary edema, tuberculosis may be present. Theremay be focal bleeding in lung, hemosiderin containinghistiocytes, focal fibrosis and angiothrombosis.
- There may be multiple spleen infarctions with secondarymixed bacterial infection and abscesses in chroniccocaine abuser.
- There may be infective endocarditis as sequelae to septicinjection site and practice.

WAR GASES

Introduction

War gases are the agents used to kill, injure or incapacitatethe enemies. In civil conditions, these gases are used to dispersethe unruly mob. The history of war gases begins withFirst World War where more than 100,000 people died andabout 1.2 million affected due to use of chlorine, phosgene,and nitrogen mustard. In Second World War, the Germansdeveloped and used number of nerve agents (tabun, sarinand soman together referred as "G" military agents) whereasEnglish developed VX in 1952.

Classification

- 1. Lacrimators: These are tear gases and causes tearing of eyes.
- Examples are:
  - Chloracetophenon (CAP)
  - Bromobenzyl cyanide (BBC)
  - Ethyl-iodo-acetate (KSK).
- 2. Lung irritants: These are asphyxiants or choking agents
- Examples are:
  - Chlorine
  - Phosgene
  - Diphosgene
  - Chloropicrin.
- 3. Vesicants: These are the agents that cause blisters
- Examples are:

- Mustard gas
- Lewisite.

➢ 4. Sternutators: These are nasal irritants

• Examples are:

- Diphenylamine chlorarsine (DM)
- Diphenyl chlorarsine (DA)
- Diphenyl cyanarsine (DC).

➢ 5. Paralysants:

• Examples are:

- Carbon monoxide
- Hydrogen sulfide.

➢ 6. Nerve gases: These agents have acetylcholine like action.

• Examples are:

- Tabun
- Sarin
- Soman
- VX.

Lachrymators (tear gases)

The gases are fired in artillery shells or pen guns to dispersethe rowdy mob.

Clinical features

➢ The vapor of gas causes intense irritation of eye andlacrimation

➢ Spasm of eyelid

- Nausea and vomiting
- Irritation of air passage and sore throat
- Rhinorrhea
- Bronchorrhea
- Cough
- Prolonged exposure leads to blistering of skin, conjunctivitis, corneal ulceration, keratitis, and pneumonitis
- In rare case, they may cause acute laryngotracheobronchitis or death.

Management

- Remove patient from exposure site
- Irrigation of eye
- Weak sodium carbonate solution may be applied to the affected skin.

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