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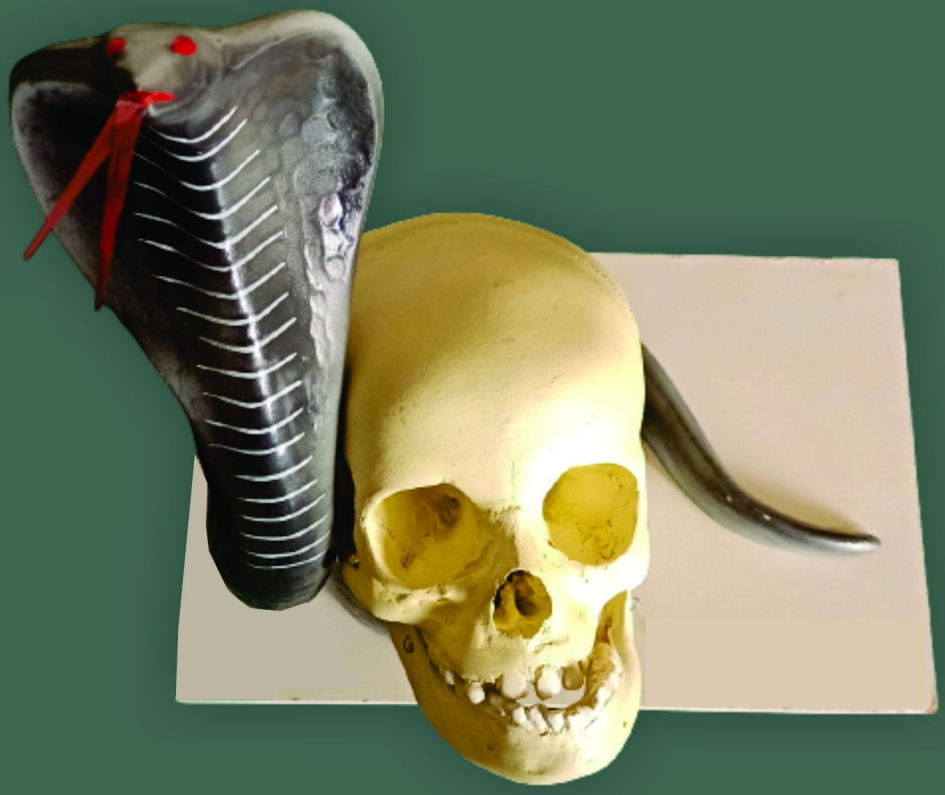
A HANDBOOK ON FORENSIC TOXICOLOGY

JV'n Dr. Gaurav Gupta



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HANDBOOK

ON

FORENSIC TOXICOLOGY

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PREFACE

Forensic Toxicology is an essential knowledge that should be possessed by every medical practitioner. The aim of the teaching Forensic Toxicology is to provide knowledge to a physician about toxins, their effects on human body and medico-legal responsibilities. Physician will also be capable of making observations and inferring conclusions by logical deductions to set enquiries on the right track in criminal matters and connected medico-legal problems. It provides knowledge of toxins, different types of toxins, their fatal dose, and fatal period, symptomatology, treatment and related law.

The aim of writing this book is to concise all essential information in easily comprehensible arrangement that can easily understood by students without missing any essential information. It is tried to keep chapter arrangement in a uniform format so that it can become easy to learn.

I would like to give my gratitude towards our Honorable Chancellor Ma'am Jv'n Vidushi Garg and our Honorable Founder and Advisor JV'n Dr. Panckaj Garg for providing me an opportunity to write this book and publish it in University press.

I would also like to thank my family and colleagues who encouraged and supported me in writing this book.

I wish that that this book will help students to understand the topic and will also guide them in the preparation of PG examination.

JV'n Dr. Gaurav Gupta
Author

ABOUT AUTHOR

Dr Gaurav Gupta is a Homoeopathic academician and consultant having B.H.M.S. M.D. from Jaipur. Currently he is working as Assistant Professor in Department of Forensic Medicine and Toxicology at Faculty of Homoeopathic Science under Jayoti Vidyapeeth Women's University, Jaipur. He has a vast clinical experience of more than 4 years. He has written and published 3 research papers in SCOPUS indexed international Journal, 2 in UGC Care listed Journals and 4 research papers in peer reviewed journals. He has written multiple book chapters in many edited volumes. He has also published 6 patent applications. He has knowledge of the subject and has unique style of teaching.

CHAPTER-1 FORENSIC TOXICOLOGY- AN INTRODUCTION

TOXICOLOGY: It can be defined as study of poison i.e. science dealing with the properties, actions, toxicity, effects on biological system, fatal dose, detection, estimation and treatment of poisons. According to Paracelsus (1493-1541), Father of Toxicology, everything is poisonous, it is the dose which differentiate a poison and a remedy.

Some other terms are:

Forensic toxicology: This science includes the use of toxicology in medical and legal field to study the harmful effects of substance on human being.

Poison: It is a substance whether solid, liquid or gas which causes injury, harmful effect on organs or even death when absorbed in body.

Clinical toxicology: It deals with effects produced by chemical substance in human body.

Toxinology: Science which deals with toxins produced by plants, animals, bacteria and fungi having *Fulminant poisoning*: It is caused by massive dose of poison resulting in rapid.

Acute poisoning: It is caused by single large dose or several dose of a poison at short interval.

Chronic poisoning: It is caused by taking smaller doses over a period of time, resulting in slow development of symptoms.

MEDICOLEGAL ASPECTS OF POISONS:

In law, the difference in medicines and poisons are the intention which is it is given. If intention of giving a substance is to harm someone or to cause death and if it is proved in court, than it is a punishable offence.

Some laws related with drugs, narcotics and psychotropic substances are:

(1) THE DRUGS AND COSMETICS ACT, 1940: It regulates the import, manufacture, distribution and sale of all kinds of drugs. The aim of this act is to control the quality, purity and strength of drugs. Any patent or proprietary medicine should display on the label or container, either the true fromula or a list of ingredients contained in it. This Act empowered the Central Government to form a Drugs Technical Advisory Board, and to establish a Central Drugs Laboratory, to help and advice both the Central and State Governments. This Act was amended in 1964.

(2) THE DRUGS AND COSMETIC RULES 1945: These rules are incorporated in the Drugs Act, 1940, to regulate the import of drugs, the functions and procedures of the Central Drugs Laboratory, the appointment of licensing authorities, and the manufacture, distribution and sale of drugs. These rules have classified drugs into Schedules.

(3) THE PHARMACY ACT, 1948: This act was designed with the aim to make regulations of the profession of pharmacy and to constitute Central Council of Pharmacy and State Councils of Pharmacy. This Act allows only the registered pharmacists to compound, prepare, mix or dispense any medicine on the prescription of a medical practitioner. This act is not applicable on dispensing medicine by a doctor to his own patients.

(4) THE DRUGS CONTROL ACT, 1950: This act provides control to government to control sale, supply and distribution of drugs. It allows the government to control maximum selling price of any drug.

(5) THE DRUGS AND MAGIC REMEDIES (Objectionable Advertisement) Act, 1954: This Act bans advertisements which offend decency or morality, and to prevent self-medication and treatment which cause harmful effects. It also prohibits the advertisements of magic remedies for procuring abortion or prevention of conception, increase of sexual potency, correction of menstrual disorders, and treatment of venereal diseases.

(6) NARCOTIC DRUGS AND PSYCHOTROPIC SUBSTANCES (NDPS) ACT, 1985: It was amended in 1989 and 2001. It revokes three acts:

(1) The Opium Act, 1857.

(2) The Opium Act, 1878.

(3) The Dangerous Drugs Act, 1930.

The Act strengthens the existing laws related to narcotic drugs, strengthens the existing controls over drugs of abuse, enhances the penalties particularly for illegal trading offences, makes provision for exercising effective control over psychotropic substances, and makes provision for the implementation of international conventions relating to narcotic drugs and psychotropic substances. This schedule lists 77 psychotropic substances.

(7) PREVENTION OF ILLICIT TRAFFIC IN NARCOTIC DRUGS AND PSYCHOTROPIC SUBSTANCES ACT, 1988- This act defines punishment if a person produces, possesses, transports, imports, exports, sells, purchases, or uses any narcotic drug or psychotropic substance except Ganja.

TYPES OF POISONS

A. Corrosive Poisons
    a. Strong Acids
    b. Strong Alkalis
    c. Metallic Salt
B. Irritants Poison
    a. Inorganic
    b. Organic
    c. Mechanical
C. Neurotics Poison
    a. Cerebral
    b. Spinal
    c. Peripheral

D. Cardiac Poison
E. Asphyxiant Poison
F. Others Poison
    a. Agrochemicals
    b. Drug of Dependence
    c. Petroleum Products
    d. Food Poisoning

NATURE OF POISONING

(1) IDEAL HOMICIDAL POISON: It should be:

i. Cheap,
ii. Easily available,
iii. Colourless, odourless and tasteless,
iv. Can be mixed easily.
v. Highly toxic,
vi. Resembling to natural disease
vii. Lacking antidote,
viii. No postmortem changes,
ix. Should not be undetectable and
x. Must be rapidly destroyed.

(2) IDEAL SUICIDAL POISON: It should be:

i. Cheap,
ii. Easily available,
iii. Highly toxic,
iv. Tasteless or agreeable taste,
v. Can be taken in food or drink,

vi. Producing painless death.

(3) STUPEFYING: Datura, cannabis indica etc.

(4) ABORTION: Calotropis, oleanders. aconite. croton, semecarpus, cantharides, ergot. lead.

(5) ACCIDENTAL: Household poison.

(6) RARE: Bacteria, insulin.

(7) CATTLE POISONING: To harm cattle of an enemy. e.g.- abrus precatorius, oleanders, calotropis, etc.

(8) ARROW POISONS: e.g. Abrus precatorius, croton oil etc.

(9) APHRODISIACS: Cantharides, cocaine, cannabis etc.

ROUTES OF ADMINISTRATION:

(1) By Inhalation.

(2) By injection.

(3) Intramuscular, subcutaneous and intradermal injection.

(4) Application to a wound.

(5) Application to a serous surface.

(6) Application to a bronchotracheal mucous membrane.

(7) By Swallowing.

(8) Insertion into the natural orifices, e.g. rectum, vagina, urethra, etc.

(9) Application to unbroken skin.

FATE OF POISONS IN THE BODY:

Maximum part of poison is removed from the body in vomiting and purging. The remaining part is absorbed goes into liver in less soluble form, which either partially metabolizes or completely destroys. Remaining part enters into circulation and either acts on the body as a whole, or on the particular organs with which it has special affinity. Few

inorganic poisons like arsenic and antimony remains in certain tissues, such as nails, hair, bones, etc., for long duration while few others like chloroform, phosphorus, nitrates etc destroys rapidly and can't be traced if postmortem is delayed.

ACTION OF POISONS:

(1) LOCAL: Poison acts on part where it comes in contact with body. E.g. – burn by corrosives, congestion and inflammation by irritants etc.

(2) REMOTE: Poison acts producing shock due to severe pain or acting on specific organs with specific affinity to poison after getting absorbed. E.g., cantharides acting on kidneys produces nephritis, nux vomica acting on the spinal cord causes tetanic convulsions etc.

(3) COMBINED: Poisons produces local as well as remote action. E.g. - carbolic acid, oxalic acid, phosphorus, etc.

FACTORS MODIFYING ACTION OF POISONS

(1) QUANTITY: More the quantity, more severe are the toxic effects. Large dose may produce death, while moderate doses may produce irritation, and small doses produce therapeutic action.

(2) FORM:

(A) PHYSICAL STATE: Action of poisons are most rapidly in gaseous state than in liquid and then in solids. Action of solid poisons depends on their solubility as easily soluble poisons acts more rapidly than less soluble poisons.

(B) CHEMICAL COMBINATION: The action of a poison depends on the solubility of poison. Inert substance may also become poisonous after chemical reaction if it produces a toxic soluble compound.

(C) MECHANICAL COMBINATION: The action of a poison may be affected if combined mechanically with other substances.

(3) MODE OF ADMINISTRATION: The route through which poison is administered also modifies the action of poison. e.g.- Inhaled poisons acts more rapidly than other modes.

(4) CONDITION OF THE BODY:

(A) AGE: Effect of poison also depends on age. It has maximum effect at both extremes of age.

(B) IDIOSYNCRACY: It is inherent personal hypersensitivity to an substance. The substance can be food also.

(C) HABIT: The effect of certain poisons decrease with long term usage as the tolerance to that substances increases and user may become addict to it.

(D) STATE OF HEALTH: Sickness, general debility, senility, chronic or disabling decreases the tolerance of a person to poison.

(E) SLEEP AND INTOXICATION: The action of a poison is delayed if a person goes to sleep soon after taking it.

DIAGNOSIS OF POISONING:

(1) IN THE LIVING: There is no definite single or group of symptoms to diagnose the poison. The following conditions should give rise to suspicion of poisoning.

A. Symptoms appear suddenly.
B. Symptoms appear immediately or within a short period after food or drink.
C. The symptoms are uniform and increases rapidly in intensity.
D. When several persons are affected with similar symptoms after eating or drinking from the same source
E. The discovery of poison in food or in vomit or in excreta.

Group of symptoms suggesting acute poisoning:

i. The sudden onset of abdominal pain, nausea, vomiting, diarrhoea and collapse.

ii. The sudden onset of coma with constriction of pupils.
iii. The sudden onset of convulsions.
iv. Delirium with dilated pupils.
v. Paralysis, especially oflower motor neurone type.
vi. Jaundice and hepatocellular failure.
vii. Oliguria with proteinuria and haematuria.
viii. Persistent cyanosis.
ix. Rapid onset of the neurological or gastrointestinal illness in persons having occupational exposure to chemicals.

Group of symptoms suggesting chronic poisoning:

i. The symptoms increases after taking suspected food, fluid or medicine.
ii. Malaise, cachexia, depression and gradual deterioration of general condition of the patient.
iii. Repeated attacks of diarrhoea, vomiting, etc.
iv. removal of patient from his usual surroundings results in disappearance of symptoms.
v. Traces of poison may be found in the urine, stool or vomit.

(2) IN THE DEAD PERSON: Collect the information from the inquest report and from the relatives of the deceased.

(I) POSTMORTEM APPEARANCES:

EXTERNAL:

A. Stains or marks of vomit, faeces or the poison itself on clothes or body.
B. Color of postmortem staining
C. Smell about the mouth and
D. The natural orifices, e.g., mouth, nostrils, rectum and vagina may show the presence of poisonous material or the signs of its having been used.

E. Injection marks.

F. Lesions on skin.

G. Marks of violence, such as bruises, or wounds.

INTERNAL: All organs and their content must be examined and preserved. Findings may found in:

A. **Smell:** Peculiar smell may be observed.

B. **Mouth and Throat:** Signs of inflammation, erosion or staining on the tongue, mouth and throat.

C. **Oesophagus:** Swelling or softening or desquamation of mucous membrane must be examined.

D. **Upper Respiratory Tract:** Examine the larynx, trachea and bronchi for evidence of volatile irritants or inhaled poisonous matter. Oedema, congestion and desquamation of mucous membrane may be observed.

E. **Stomach:** Toxic substances may be found in stomach. Following condition may found:

   a) **Hyperaemia**
   b) **Softening**
   c) **Ulcers**
   d) **Perforation**

F. **The Duodenum and Intestine:** Any sign of perforation or any other characteristic changes must be observed.

G. **Liver:** Necrosis of liver or fatty changes or signs of jaundice may be found.

H. **Respiratory System:** Oedema, congestion and desquamation of the mucous membrane of the trachea and bronchi may be seen.

I. **Heart:** Subendocardial haemorrhages in the left ventricle may occur.

J. **The bladder, and in females the vagina and uterus** should be examined for the poison as in case of insertion, traces of poison may found.

DUTIES OF A DOCTOR IN A CASE OF SUSPECTED POISONING

MEDICAL: Treatment to save life of the patient is the most important duty.

LEGAL: Helping the police in investigation of poisoning.

1. Noting down the particulars of the patient and history.
2. In case of suspected homicidal poisoning, the doctor must confirm his suspicion before expressing an opinion. For this he must:
   i. Collect vomitus and urine and submit it for examination.
   ii. Carefully observe and record the symptoms in relation to food, any change in color, taste or smell of food & drink, and other persons affected at the same time.
   iii. Consult in strict confidence a senior practitioner and keep him informed about the case.
   iv. Shift the patient to the hospital and if refused by patient, then doctor should appoint a nurse of his confidence who should administer the medicine and food and not to allow anyone with the patient alone.
3. Once the poisoning is confirmed, patient should be shifted to the hospital.
4 Any suspected articles of food, excreta and stomach wash samples should be preserved for examination.
5. A government medical officer is required to report all cases of suspected poisoning to police, whether accidental, suicidal or homicidal.
6. If a private practitioner is convinced that the patient is suffering from homicidal poisoning, he is bound under Sec. 39 CrPC to inform the police or Magistrate.
7 Non-compliance is punishable under Sec. 176 IPC (simple imprisonment of 1 month or fine of ` 500/- or both). Giving false information on such matters is punishable under Sec. 177 IPC (simple imprisonment for 6 months or fine of ` 1000 or both).
7. If the private practitioner is sure that the patient is suffering from suicidal/accidental poisoning, he is not bound to inform the police

8. If the condition of the patient is serious, he must make arrangement to record the dying declaration.

9. If the patient dies, he should not issue a death certificate, but should inform the police.

10. Any opinion about the nature of poison should be given only after getting the report from the forensic science laboratory.

11. If the practitioner is summoned, he is bound to provide all information regarding the case.

TREATMENT OF POISONING

(I) IMMEDIATE RESUSCITATIVE MEASURES- They should be adopted which are:

A= Airway should be clear and if neede intubation can be done

B= Breathing can be supplemented by oxygen therapy either by a ventimask or through endotracheal tube if needed.

C= Circulation must be stabilized by giving I.V. fluid.

D= Depression of CNS should be corrected.

(II) REMOVAL OF UNABSORBED POISON:

(1) Inhaled Poisons: If the poison is inhaled, remove the patient into fresh air and oxygen should be given. The air-passages should be kept free from mucus.

(2) Injected Poisons: If the poison has been injected subcutaneously from a bite or an injection, a tourniquet should be applied immediately above the wound and keep looseing it every 10 minutes to prevent gangrene. Excision of wound should be followed by removal of poison sucking it out. Proper antidote should be given.

(3) Contact Poisons: The part that came in contact with poison should be washed for 30 minutes or neutralized by suitable chemical. Clothing must be removed immediately.

(4) Ingested Poisons:

a. **Gastric Lavage:** It is useful any time wihin 3 hours after ingestion of a poison.

b. **Emetics** may be used.

(II)ADMINISTRATION OF ANTIDOTES:

These substances counteract or neutralize the effects of poisons. Common modes of action of antidotes are:

(1) Inert complex formation.

(2) Accelerated detoxification.

(3) Reduced toxic conversion.

(4) Receptor site blockade.

(5) Toxic effect bypass

(A) MECHANICAL OR PHYSICAL ANTIDOTES: They neutralizes poisons by mechanical action or preventing their absorption.

(1) Activated charcoal.

(2)Demulcents forms a protective layer on the gastric mucous membrane, thus preventing damage.

(3) Bulky food acts as an mechanical antidote to glass powder.

(B) CHEMICAL ANTIDOTES: They reacts with poison and forms a harmless or insoluble compounds.

(C) PHYSIOLOGICAL OR PHARMACOLOGICAL ANTIDOTES: They act on the tissues of the body and produce symptoms exactly opposite to those caused by the poison. Some of the antidotes are:

1. **B.A.L.** (British anti-lewisite; dimercaprol; dimercaptopropanol): It is used as a physiological antidote in arsenic, lead, bismuth, copper, mercury, gold, thallium and antimony.
2. **E.D.T.A.** (ethylenediarninetetraacetic acid: calcium disodium versenate; edathernil; edetic acid; versene): It is a chelating agent and is antidote of lead, mercury, copper, cobalt, cadmium, iron and nickel.
3. **Penicillamine** (cuprirnine; dimethyl cystine): It is a hydrolysis product of penicillin and antidotes copper, lead and mercury.
4. **DMSA, succimer** (Meso-2, 3-dimercaptosuccinic acid): It is used in lead, mercury and arsenic poisoning.
5. **DMPS** (2,3-dirnercaptopropane 1-sulfonate): It is used in mercury, lead and arsenic poisoning.
6. **Desferrioxamine:** It is used in acute iron poisoning.

(III) ELIMINATION OF POISON BY EXCRETION: Indications are:

a. Severe poisoning.
b. Progressive deterioration
c. High risk of morbidity or mortality.
d. When normal route of excretion of the toxic compound is impaired.
e. When the poison produces delayed serious toxic effects.
f. When the patient is having other systemic diseases e.g. cardiovascular, respiratory etc.

The methods of excretion are:

(1) Renal Excretion

(2) Purging

(3) Whole-Bowel Irrigation

(4) Diaphoretics

(5) Forced Alkaline Diuresis

(6) Peritoneal Dialysis

(7) Haemodialysis

(8) Charcoal Haemoperfusion

(IV) SYMPTOMATIC TREATMENT: The symptoms should be treated on general lines.

(V) FOLLOW-UP: Follow-up is must to treat the complications. Psychiatric treatment is needed in suicidal cases.

CHAPTER-2 CORROSIVE POISONS- INORGANIC- SULPHURIC ACID

It is:

- Heavy,
- Odourless,
- Colourless,
- Non-fuming,
- Hygrascopic, oily liquid,
- Tendency to carbonise organic substances.

SIGNS AND SYMPTOMS:

- The lips are swollen and excoriated.
- Flow of acid on drinking may causes brown or black streaks extend in from angles of the mouth to the sides of the chin, and may also to the front of the neck.
- Corrosion of mucous membranes of mouth, throat and oesophagus.
- Burning pain, stridor, drooling, odynophagia and dysphagia.
- Pain in epigastric region extending to whole abdomen.
- Pharyngeal pain is the most common symptom.
- Eructation, nausea and vomiting. Vomitus is brown or black, mucoid, strongly acid, and may contain shreds of the charred wall of the stomach.
- Thirst is intense, but any attempt to drink causes vomiting.
- Teeth are chalky-white and tongue becomes swollen, sodden and black.
- Distension and tenderness of abdomen with severe constipation and tenesmus.
- The voice becomes hoarse and husky.
- The eyes are sunken with dilated pupils.

- Mind is not affected till the death.
- It may cause death from asphyxia due to oedema of the glottis or collapse due to perforation of stomach or circulatory collapse or toxaemia. Death may also result from pneumonia or secondary infection or starvation due to stricture of oesophagus.
- It may lead to complications such as:

(A) Acute:

(1) Upper airway obstruction and injury.

(2) G.I. haemorrhage.

(3) Oesophageal and gastric perforation.

(4) Sepsis.

(5) Tracheobronchial necrosis, atelectasis and obstructive lung injury.

(B) Chronic:

(1) Oesophageal obstruction.

(2) Pyloric stenosis.

(3) Vocal cord paralysis with airway obstruction.

VITRIOLAGE (vitriol throwing):

Vitriolage is throwing of any corrosive substance on someone. It is usually thrown on enemy to take revenge or to teach someone a lesson by disfiguring the face and harming them. The burns are penetrating and painless. Healing is a slow process and resulting in scar formation causing contracture. It may also result in blindness if eyes are involved. Death may result from shock or toxaemia. Corrosive substances that used are sulphuric acid, nitric acid, carbolic acid, corrosive alkali or juice of marking nut or calotropis.

FATAL DOSE: 5 to 10 min.

FATAL PERIOD: 12 to 24 hours.

TREATMENT:

(1) Gastric lavage and emetics should be avoided.

(2) Neutralize the acid by diluting it with milk or milk of magnesia or lime water. Carbonates and bicarbonates should not be used as it may result in distension of abdomen.

(3) Demulcent

(4) Prednisolone to prevent oesophageal stricture and shock.

(5) Correct circulatory shock.

(6) Tracheostomy, if there is oedema of glottis.

(7) Nothing should be given by mouth.

(8) Wash skin burns with water and apply magnesium oxide or sodium bicarbonate.

(9) Wash eyes Eye with water or sodium bicarbonate solution.

(10) Symptomatic.

POSTMORTEM APPEARANCES:

EXTERNAL-

- Corrosion of mucous membranes of lips, mouth and throat, and of the skin over the chin, angles of the mouth, and hands.
- Necrotic areas are grayish-white which become brown or black and leathery.
- Bums and stains can be found on clothes.

INTERNAL:

- Only upper digestive tract and the respiratory system are affected.
- Inflammation and oedema of upper GIT with severe interstitial haemorrhage.
- Lip and mouth escape injury if acid is taken from spoon.
- Squamous epithelium of oesophagus is comparatively resistant and superficial mucosal reaction is produced.

- Acid affects columnar epithelium of the stomach, causing superficial erosion and coagulation with eschar formation.
- Perforation of the oesophagus is rare.
- The greater part of stomach may become soft, spongy, black mass and disintegrates when touched.
- Stomach may perforate resulting in escape of gastric contents into the peritoneal cavity resulting in chemical peritonitis and corrosion of other organs.
- Swelling of the liver and kidneys.

MLI:

- Accidental Poisoning.
- Suicidal Poisoning.
- Not used for homicide,
- As abortifacient.

CHAPTER-3 CORROSIVE POISONS- INORGANIC- HYDROCHLORIC ACID & NITRIC ACID

HYDROCHLORIC ACID POISONING

Hydrochloric acid (muriatic acid) is

- Pungent,
- Colourless,
- Fuming liquid.
- A natural constituent of the stomach and bowels.

SIGNS AND SYMPTOMS

ACUTE POISONING:

- It is less corrosive in its action in compare to sulphuric acid.
- It destroys mucous membrane.
- The mucous membrane becomes grey or grey-white and becomes brown or black due to the production of acid haematin.
- Inhalation of fumes causes intense irritation of throat and lungs, suffocation, coughing, dyspnoea and cyanosis.

CHRONIC POISONING:

- It is caused by prolonged exposure to fumes of hydrochloric acid.
- The symptoms are coryza, conjunctivitis, corneal ulcer, pharyngitis, bronchitis, inflammation of gums and loosening of teeth.

FATAL DOSE: 15 to 20 min.

FATAL PERIOD: 18 to 36 hours.

TREATMENT:

It is same as sulphuric acid.

POSTMORTEM APPEARANCES:

- These are similar to sulphuric acid but less severe in its action.
- The stomach contains brownish fluid with brownish discoloration of folds of the whole stomach mucosa.
- Perforation of the stomach is rare.
- Acute inflammation and oedema of respiratory passages and lung tissue are common.

MLI:

- Suicidal poisoning.
- Accidental poisoning.
- Homicidal poisoning.
- As abortifacient

NITRIC ACID POISONING

Nitric acid is

- Clear,
- Colorless,
- Fuming,
- Heavy liquid,
- Has a peculiar and choking odor.

ACTION:

Its combination with organic matter produces picric acid which results in yellow discoloration of tissue.

SIGNS AND SYMPTOMS

- Similar to sulphuric acid.
- Eructation and abdominal distention due to gas formation.
- Yellow discolouration of the tissues, crowns of the teeth.
- Yellow stains on the clothes.
- Inhalation of fumes causes lachrymation, photophobia, irritation of air-passages and lungs producing sneezing, coughing, dyspnoea and asphyxia.
- Brown urine

FATAL DOSE: 10 to 15 min.

FATAL PERIOD: 12 to 24 hours.

TREATMENT:

It is same as for sulphuric acid.

POSTMORTEM APPEARANCES:

- These are similar to sulphuric acid but less severe in its action.
- Tissues are stained yellow except in the oesophagus and stomach, where they appear brown or brown-black due to formation of acid haematin.
- The stomach wall is soft, friable and ulcerated.
- Perforation of the stomach is not common.
- In death from inhalation of fumes, congestion of larynx, trachea, and bronchial tubes with oedematous lungs.

MLI:

- Suicidal poisoning.
- Accidental poisoning.

- Homicidal poisoning is rare.

CHAPTER-4 CORROSIVE POISONS- ORGANIC ACIDS- OXALIC ACID

It is-

- Colourless,
- Transparent,
- Prismatic Crystals,
- A natural part of plants such as spinach, cabbage, etc in the form of oxalate.
- Used as a bleach to remove stains
- Used to remove writing and signature illegally.

ACTION:

LOCAL:

- 10% of oxalate concentrated solution or crystal are corrosive in nature.
- It corrodes the mucous membrane of GIT.
- Less than 10% of concentration is a strong irritant, and produces systemic effects when absorbed.

SYSTEMIC:

(a) Shock: It is produced by large dose and results into death.

(b) Hypocalcaemia: It combines with Calcium ion and its level decreases in body resulting in hypocalcaemia and may result in death within 12 hrs.

(c) Renal damage: Tubular necrosis occurs and results into death due to uraemia in 2-14 days.

SIGNS AND SYMPTOMS:

A. FULMINATING POISONING:

- Large concentrated dose produces immediate symptoms and death within minutes.
- Burning, sour, bitter taste in the mouth with a sense of constriction around the throat and burning pain from the mouth to the stomach.
- Pain starting in the epigastric region radiates to whole abdomen. Tenderness may also be present.
- Nausea, eructation and vomiting. Vomitus contains altered blood and mucus and has a "coffee-ground" appearance.
- Diarrhoea may develop.
- Ultimately may result into death.

B. ACUTE POISONING:

- Large dose produces acute poisoning.
- Hypocalcaemia is prominent symptoms.
- GIT symptoms are less prominent.
- Muscle irritability and tenderness is present.
- Tetany and convulsions may also develop.
- Numbness and tingling of the fingertips and legs.
- Signs of cardiovascular collapse appear.

C. DELAYED POISONING:

- Symptoms of uraemia are very prominent.
- Urine may be scanty or suppressed
- Urine may contain traces of blood, albumin and calcium oxalate crystals.

- Metabolic acidosis and ventricular fibrillation may also be present.

FATAL DOSE: 15 to 20 g.

FATAL PERIOD: 1 to 2 hours.

TREATMENT:

(1) Gastric lavage carefully with calcium lactate or gluconate in water.

(2) Calcium preparation as antidote.

(3) Calcium gluconate 10%.

(4) Dialysis in case of renal failure.

(5) Parathyroid extract 100 units i.m. in severe case.

(6) Demulscents.

(7) Bowel wash.

(8) Symptomatic.

POSTMORTEM APPEARANCES:

- Strong solution causes white discoloration of the tongue, mouth, pharynx and oesophagus as if bleached, may also be red due to irritation.
- Oesophageal membrane shows longitudinal erosions.
- The mucous membrane of the stomach is reddened or punctate from erosions or almost black. It may be softened in patches but perforation is very rare.
- The stomach contents are gelatinous and brownish due to acid haematin formation.
- Usually intestines are not affected except upper part of the duodenum.
- Kidneys have oedema, congestion and the tubules are filled with oxalate crystals.
- Necrosis of tubules, chiefly proximal convoluted tubules.
- Congestion of the lungs, liver, kidneys and brain, without any local appearances.

MLI

- Accidental poisoning.
- Suicidal poisoning is rare.
- Homicidal poisoning is rare.
- As abortifacient.

CHAPTER-5 CORROSIVE POISONS- ORGANIC ACIDS- CARBOLIC ACID

It is:

- Colourless,
- Prismatic,
- Needle-like crystals,
- Burning sweetish taste,
- Turns pink and liquefy when exposed to air.
- Characteristic phenolic smell.
- Slightly soluble in water and easily soluble in glycerine, ether, alcohol and benzene.

SIGNS AND SYMPTOMS:

Poisoning by carbolic acid is called *carbolism*.

LOCAL:

1. **SKIN:**
    - Burning and numbness.
    - Bleaches the skin resulting in hard, cracked, whitish surfaces.
    - Deep burns are black.
    - It produces a painless white opaque eschar which is leaves a brown stain.
    - Necrosis and gangrene
    - Lysol results in brownish-purple discoloration of tissues.

2. **GIT:**

- Hot burning pain from the mouth to the stomach followed by tingling and anaesthesia.
- Dysphagia
- Painful and difficult speech.
- The lips, mouth and tongue corrodes and become white and hardened.
- Nausea and vomiting.

3. RESPIRATORY TRACT:

- Pulmonary and laryngeal oedema
- Slow and difficult breathing leading to respiratory failure.
- Bronchitis and bronchopneumonia due to aspiration of vomitus.

SYSTEMIC EFFECTS:

- Depresses the nervous system, especially the respiratory centre.
- Headache, giddiness, unconsciousness and coma.
- Contracted pupils, Noisy and difficult breathing.
- Rapid, feeble and irregular pulse.
- Cold sweat on face.
- Dusky cyanosis, respiratory alkalosis and metabolic acidosis.
- Liver may be damaged.
- Haemolysis and methaemoglobinaemia in severe cases.

CHRONIC POISONING (PHENOL MARASMUS) - It is characterised by

- Anorexia, weight loss.
- Headache, vertigo.
- Dark urine.
- Pigmentation of skin and sclera.

- Oochronosis: A condition with pigmentation in the cornea and various cartilages

It can cause death due to syncope or asphyxia due to failure of respiration or oedema of glottis or due to complications such as bronchopneumonia.

FATAL DOSE: 10 to 15 g.
FATAL PERIOD: 3 to 4 hours.

TREATMENT:

1. Gastric lavage
2. Magnesium sulphate is left after lavage
3. Emetic are useless due to the anaesthetic effect.
4. Demulcents.
5. Sodium bicarbonate in saline is given i.v. to promote excretion of carbolic acid by increasing dialysis and to correct circulatory dialysis.
6. Haemodialysis in case of renal failure.
7. Methylene blue to treat severe methaemoglobinaemia.
8. In case of local application, wash skin with undiluted polyethylene glycol or soap and water and apply olive oil.

POSTMORTEM APPEARANCES

EXTERNAL:

- Corrosion of the skin where it touches skin.
- Smell of phenol about the mouth.
- The tongue and mucous membrane of the lips, mouth and throat is corrugated, swollen, whitened or ash-grey and partially detached with numerous small submucous haemorrhages.

INTERNAL:

- The mucosa of the esophagus becomes tough, white or grey, corrugated and arranged in longitudinal folds.
- The mucosa folds of stomach are swollen and covered by opaque, coagulated, grey or brown mucous membrane.
- A reddish fluid mixed with mucus and shreds of epithelium may found in stomach having phenolic smell. The duodenum and upper part of the small intestine may show similar but milder changes.
- At the site of contact with stomach, the liver and spleen usually show a whitish, hardened patch due to the gradual discharge of phenol through membranes.
- The kidneys may show haemorrhagic nephritis.
- The brain is congested, may be oedematous.
- If vomit or poison has been inhaled, coagulation necrosis of the mucosa and severe congestion of the submucosa of the air-passages may be seen. Laryngeal and pulmonary oedema also occurs.

MLI

- Suicidal poisoning.
- Accidental poisoning.
- Rarely used for homicide.
- As abortifacient.

CHAPTER-6 CORROSIVE POISONS- CAUSTIC ALKALIS

- It includes ammonia, potassium hydroxide, sodium hydroxide, calcium hydroxide, ammonium carbonate, potassium carbonate and sodium carbonate etc.
- These are common chemicals used in industries.
- Mostly they are found as white powder but may also be colourless gas such as Ammonia which have very pungent, choking odour or can be liquid as Ammonium
- Household bleaches contains 5% sodium hypochlorite solutions which causes mucosal irritation.

ACTION: It causes cellular dehydration and produces heat that results in burn. It produces eschars (liquefaction necrosis) and penetrates skin more deeply with saponification of fat and oedema.

SIGNS AND SYMPTOMS:

- The lesions caused by caustic alkalis are similar to those of produced by corrosive acids.
- It has acid caustic taste and feeling of burning from the throat to the stomach.
- Vomited matters are alkaline. Vomitus is thick and slimy, but may contain blood and shreds of mucosa.
- Purging is present with severe pain.
- Stool also contains mucus and blood.
- Contact with skin causes greyish, soapy, necrotic area.
- In case of ingestion, abrasions, blisters and brownish discoloration on lips and around the mouth are present. The mucosa of the GIT is swollen, soft and a grey.

- Major complication is Oesophageal stricture.

FATAL DOSE:

Potassium or sodium hydroxide - 5g.

Potassium carbonate - 18g.

Sodium carbonate - 30 g.

Ammonia - 5 to 10 ml.

FATAL PERIOD: Usually 24 hours.

TREATMENT:

(1) Demulcents.

(2) Gastric lavage and emetics is usually contraindicated but may be performed carefully in mild cases as stomach may be soft.

(3) Oxygen in poisoning by ammonia vapour with moist enviornment.

(4) Protect the airway and if necessary tracheostomy may be done.

(5) Parentral analgesics.

(6) Steroids to relive laryngeal inflammation.

(7) Antibiotics to prevent infection.

(8) Wash skin with undiluted polyethylene glycol.

POSTMORTEM APPEARANCES:

- Skin around the mouth become dark and parchment- like after death.
- Lips, mouth and throat show corrosion.
- Inflammatory oedema with corrosion and sliminess of the tissues of the oesophagus and stomach are prominent features.

MLI:

- Accidental poisoning.
- Homicidal poisoning is rare.
- Suicidal poisoning.
- Throwing caustic soda solution with evil intention on the face and body of an enemy.

CHAPTER-7 IRRITANTS – INORGANIC-PHOSPHORUS

It exists in two varieties:

- White/ yellow phosphorus
  - Fertilizers, insecticide, rodenticide, incendiary bombs, smoke screens, firework
- Red phosphorus
  - matchbox

DIFFERENCE IN RED AND WHITE PHOSPHORUS

Red phosphorus	White phosphorus
• Violet red/ reddish brown	• White or yellow
• Amorphous, solid mass	• Waxy, translucent, soft stick
• odourless, tasteless	• Garlicky taste and odour
• Non luminescent	• Luminescent in dark
• Soluble in carbon disulphide	• Insoluble
• Non toxic	• Highly toxic
• Does not ignite in air	• May ignite in air

MOA

- Protoplasmic poison
- Affect cellular oxidation
- Causes necrobiosis especially of liver (yellow atrophy of liver) and kidneys

1. **FULMINATING POISON**
   - Dose is more than 1gm.
   - Patient dies within 12 hrs due to shock and cardiovascular collapse.
   - If patient survives, becomes restless, delirious and may be maniacal before death.

2. **ACUTE POISONING**

- **FIRST STAGE(few minutes-8 hr)**
  - Skin: painful penetration, second and third degree burn.
  - Ingestion :
    - Nausea, vomiting , diarrhoea, thirst,
    - Burning pain in throat and abdomen,
    - Garlic like odour in breath, vomitus and faeces,
    - Luminescent smoking urine and faeces

- **SECOND STAGE SYMPTOMS(after 2-3 days)**
  - It is a symptom free period.

- **THIRD STAGE SYMPTOMS**
  - Severe abdominal pain
  - Nausea, vomiting, haematemesis, diarrhoea
  - Tender hepatomegaly, jaundice, pruritis
  - Oliguria, haematuria, albuminuria
  - Headache, insomnia, convulsions, coma
  - Carpopedal spasm, tetany, stridor, opisthotonus
  - Purpura, epistaxis
  - Death d/t cardiac dysrythmias, hypocalcemia and hyperkalemia.

3. CHRONIC POISONING

- It is caused if there is constant exposure to fumes for a long period.
- Necrosis of the lower jaw in the area of decayed teeth.
- Symptoms start with pain in tooth followed by swelling of jaw, loosening of teeth, necrosis of gums, affecting the bone in the mandible. This condition is called "phossy or glass jaw". There is osteomyelitis and necrosis of jaw with discharge of foul smelling pus from multiple sinuses.
- Other symptoms are:
  - Nausea, vomiting, anorexia, pain in stomach, indigestion, purging etc.
  - Pain in joints
  - Bronchitis
  - Loss of weight
  - Jaundice and anaemia

FATAL DOSE.: 60- 120 mg

FATAL PERIOD : 2-8 days.

TREATMENT

1. Airway support and fluid maintenance
2. External burns should be washed using 1% Cu sulphate solution.
3. Gastric lavage: $KMNO_4$ (antidote), charcoal
4. Demulscents are contraindicated
5. Purgatives
6. Vit. K or blood transfusion to treat hypoprothrombinemia.

7. Haemodialysis
8. Vitamins and noradrenaline to prevent liver damage.

POSTMORTEM APPEARANCE

- Signs of irritation in oesophagus, stomach and intestine and presence of luminous in stomach.
- Sign of jaundice.
- GIT contents may smell like garlic.
- Liver is swollen, yellow, soft, fatty and very fragile.
- Acute yellow atrophy can be seen if patient survives.

MLI

- Accidental
- Occasionally homicidal
- To produce abortion
- Occupational poisoning

CHAPTER-8 IRRITANTS- METALLIC POISONS- ARSENIC

Pure arsenic metal is not poisonous in nature, but becomes poisonous when combines with oxygen and forms vapors of arsenic trioxide.

POISONOUS COMPOUNDS:

(1) Arsenious oxide or arsenic trioxide

(2) Copper arsenite (Scheele's green) and copper acetoarsenite (Paris green or emerald green).

(3) Arsenic acid.

(4) Sodium and potassium arsenate.

(5) Arsenic sulphide

(6) Arsenic trichloride (butter of arsenic).

(7) Arseniuretted hydrogen or arsine

ACTION: It causes local irritation of mucous membrane and depression of nervous system. Arsenic combines with the sulphydryl groups of mitochondrial enzymes and interferes with cellular respiration. It acts on vascular endothelium and results into increased permeability, tissue oedema and haemorrhage.

SIGNS AND SYMPTOMS:

(1) THE FULMINANT POISONING: Large doses of arsenic cause death from shock and peripheral vascular failure. Hypotension develops with dilatation of capillaries. Arsenic acts directly on heart muscle. In this poisoning, gastrointestinal symptoms are absent.

(2) ACUTE POISONING

(A) THE GASTROENTERIC TYPE: It is acute poisoning of arsenic which resembles bacterial food poisoning. Symptoms appear half to one hour to several hours after ingestion. It has sweetish metallic taste.

G.I.T.:

- Constriction in the throat and difficulty in swallowing
- Burning and colicky pain in oesophagus, stomach and bowel.
- Intense thirst and severe vomiting which may be projectile.
- Purging with tenesmus, pain, and irritation about the anus.
- Stool is frequently and involuntarily, and dark-colored, stinking and bloody, but later become colorless, odorless and watery resembling rice-water stools of cholera.
- A garlicky odor in breath and faeces.
- Fatty infiltration of liver.

RENAL:

- Oliguria, uraemia.
- Albumen, RBCs and casts are present in urine.
- Painful micturition.

C.V.S.:

- Acute circulatory collapse with vasodilation.
- Increased vascular permeability.
- Ventricular tachycardia, ventricular fibrillation.

C.N.S.:

- Headache, vertigo, hyperthermia,
- Tremors, convulsions, coma, general paralysis.
- A peripheral neuropathy

SKIN:

- Loss of hair,

- Skin eruptions.

Death may occur due to circulatory failure.

(3) NARCOTIC FORM:

- Gastrointestinal symptoms are less.
- Giddiness, formication and tenderness of the muscles.
- Delirium, coma and death.
- Paralysis of the extremities.
- Haematuria, and acute tubular necrosis.
- Anaemia, leucopaenia and thrombocytopaenia can occur.

CHRONIC POISONING: It is due to repeated small doses in metal workers, or by food or drink having it in traces. Usually its chronic poisoning is accidental. It may also be used for homicidal purpose. Symptoms are:

- **C.N.S.:** Polyneuritis, anaesthesias, paraesthesia. encephalopathy.
- **SKIN:** Finely mottled brown pigmentation on the skin flexures, temples, eyelids and neck (raindrop type of pigmentation), which persists for many months. Hyperkeratosis of the palms and soles with irregular thickening of the nails and development of trasverse white lines in the fingernails called Aldrich-Mees lines.
- **EYES:** Congestion. lacchrymation, photophobia.
- **G.I.T.:** Nausea, vomiting, abdominal cramps, diarrhoea, salivation.
- **C.V.S. AND KIDNEYS:** Chronic nephritis, cardiac failure, dependent oedema.
- **HEPATIC:** Hepatomegaly. jaundice, cirrhosis of the liver.
- **HAEMATOLOGIC:** Bone marrow suppression, hypoplasia, anaemia, thrombocytopaenia, leukaemia.
- **RESPIRATORY:** Cough, haemoptysis, dyspnoea.

- **GENERAL:** Anaemia and weight loss, loss of hair, brittle nails.
- Arsenic is teratogenic and can result in lung and skin cancer, leukaemia, etc.,

FATAL DOSE: 0.1 to 0.2 g. of arsenic trioxide.

FATAL PERIOD: One to two days.

TREATMENT:

(1) Gastric lavage.
(2) Emetics are not suggested.
(3) Demulcents.
(4) Alkalis are contraindicated as they increase the solubility of arsenic.
(5) Freshly precipitated, hydrated ferric oxide as it converts toxic arsenic to non-toxic ferric arsenite.
(6) B.A.L.
(7) Penicillamine
(8) DMSA (succimer) or DMPS
(9) Demulcents lessen irritation.
(10) Castor oil or magnesium sulphate prevents intestinal absorption of arsenic.
(11) Glucose-saline with sodium bicarbonate.
(12) Haemodialysis in case of renal failure.
(13) Chelation therapy is ineffective.
(14) In case of chronic poisoning, remove the patient from the source of exposure.

POSTMORTEM APPEARANCES:

EXTERNAL:

- Sunken eyeballs.
- Cyanosis.

- The body may be shrunken due to dehydration.

INTERNAL:

- Inflamed or ulcerated mouth, pharynx and oesophagus may be found.
- The mucosa of stomach is swollen, oedematous and red either generally or in patches, especially in the pyloric region.
- The stomach mucosa resembles red velvet.
- The caecum and rectum show slight inflammation.
- Arsenic may penetrate stomach walls and found in liver, omentum and endocardium.
- In fulminating type, the stomach and intestines may not show any signs of inflammation.
- The liver, spleen and kidneys are congested, enlarged and show cloudy swelling and occasionally fatty change.
- Oedema of brain with patchy necrosis or haemorrhagic encephalitis. The meninges are congested. Subendocardial petechial haemorrhages of the ventricle are common.

MLI:

- Occupational poisoning.
- Homicidal poisoning.
- Mass homicidal.
- To perform criminal activities.
- Suicide poisoning is rare.
- Accidental poisoning.
- As abortifacient.
- Cattle poison.

CHAPTER-9 IRRITANTS- METALLIC POISON- MERCURY

It is:

- Liquid metal,
- Bright silvery
- Volatile at room temperature.

POISONOUS COMPOUNDS:

(1) Mercuric chloride

(2) Mercuric oxide (brick-red crystalline powder).

(3) Mercuric iodide (scarlet-red powder).

(4) Mercuric cyanide (white prismatic crystals).

(5) Mercuric sulphide (cinnabar, sindoor). Artificial preparation occurs as red crystalline powder and is known as vermilion.

(6) Mercurous chloride (calomel).

(7) Mercuric nitrite.

(8) Mercuric sulphate (white crystalline powder).

(9) Sulphate of mercury (lemon-yellow powder).

(10) Ammoniomercuric chloride.

(11) Organic compounds of mercury.

ACTION: The mercuric ion affects cellular transport function as it binds with sulphadryl groups of enzymes and cellular proteins, nucleic acid and mitotic apparatus. It can cause renal damage. In the CNS, mercury acts mainly upon cerebellum, temporal lobe, basal ganglia and corpus callosum. Its manifestations might be similar to Kawasaki disease

SIGNS & SYMPTOMS:

FIRST PHASE:

- Acrid metallic taste.
- Sensation of constriction in the throat, hoarse voice, difficulty in breathing.
- The mouth, tongue and fauces become corroded, swollen and show a greyish-white coating.
- Hot burning pain in the mouth, extending down to the stomach and abdomen, followed by nausea, retching and vomiting.
- The vomit contains greyish slimy mucoid material with blood and shreds of mucous membrane. Vomiting is followed by diarrhea with bloodstained stools and tenesmus.
- Circulatory collapse.
- Inhalation of fumes produces nervous symptoms, e.g. ataxia, restriction of visual field, paresis and delirium.

SECOND PHASE:

- Second phase starts in one to 3 days.
- Glossitis and ulcerative gingivitis.
- Severe infection, loosening of teeth and necrosis of the jaw may occur.
- Renal tubules show necrosis and produce transient polyuria, albuminuria, cylindruria, uraemia and acidosis.
- After many days membranous colitis develops and produces dysentery, ulceration of colonic mucosa and haemorrhage.

CHRONIC POISONING (HYDRARGYRISM): The causes are

(1) Continuous accidental absorption in workers,

(2) Excessive therapeutic use,

(3) Recovery from a large dose, and

(4) If ointment is used as external application for a long time.

SYMPTOMS:

- It shows classic triad of gingivitis and salivation, tremors and neuropsychiatric changes.
- Occasionally a blue line at their junction with teeth.
- Sore mouth and throat, loosening of teeth.
- Anaemia, anorexia, loss of weight.
- Chronic inflammation of kidneys with progressive uraemia.
- Tremors (Danbury tremors) start first in the hands, then lips and tongue and finally involve arms and legs. The tremor is moderately coarse and is interspersed by jerky movements.
- The advanced condition is called **hatter's shakes or glass-blower's shakes**, as it is common in glass-blowing and hat industries workers. The patient is unable to dress himself, write legibly or walk properly.
- The most severe condition is **concussio mercurialis**, in which no activity is possible.
- **Mercurial erethism** is seen in persons working with mercury in minor manufacturing firms. This term is used to refer to the psychological effects of mercury toxicity. These include anxiety, depression, shyness, timidity, irritability, loss of confidence, mental depression, delusions and hallucinations, or suicidal melancholia, or manic depressive psychosis (mad hatter), emotional instability, loss of memory and insomnia.
- **Mercurialentis** is a peculiar eye change due to exposure to the vapor of mercury. It is due to brownish deposit of mercury through the cornea on the anterior lens

capsule. Slit-lamp examination demonstrates a malt-brown reflex from the anterior lens capsule. It is bilateral and has no effect on visual acuity.

- Renal damage results in membranous glomerulonephritis with hyaline casts and fatty casts in the urine. Kidney is the primary target.
- **Acrodynia or pink disease** is idiosyncratic hypersensitivity reaction commonly seen in children. The onset is insidious with anorexia, insomnia, sweating, skin h and photophobia. Hands and feet become puffy, paraesthetic with peeling of skin. Teeth may shed.

FATAL DOSE: One to 2 g. of mercuric chloride.

FATAL PERIOD: 3-5 days.

TREATMENT:

(1) Gastric lavage.
(2) Activated charcoal.
(3) Demulcents.
(4) Bowel wash.
(5) Pencillamine
(6) BALis used. BAL and pencillamine in combination should not be used as they may cause formation of a toxic compound.
(7) Ca-EDTA is contraindicated as it is nephrotoxic with mercury.
(8) Urine must be kept alkaline.
(9) Haemodialysis.
(10) Shift the patient away from the source of exposure.

POSTMORTEM APPEARANCES:

- The mucosa of the gastrointestinal tract shows inflammation, congestion, coagulation and corrosion.

- Large intestine shows necrosis due to the re-excretion of mercury into the large bowel.
- Acute tubular and glomerular degeneration or haemorrhagic glomerular nephritis is seen.
- The liver is congested and shows cloudy swelling or fatty change.

MLI:

- Accidental poisoning.
- As abortifacient.
- Homicidal poisoning is rare.
- Suicidal poisoning is rare.
- Occupational poisoning.

CHAPTER-10 IRRITANTS- METALLIC POISON- LEAD

Lead is-

- Heavy steel-grey metal.
- Pure metal as well as its salts are poisonous.

The salts which are toxic are:

(1) Lead acetate
(2) Lead carbonate (safeda)
(3) Lead chromate
(4) Lead monoxide
(5) Lead tetroxide (sindur)
(6) Lead sulphide.

USES:

Lead is used in storage batteries, solders, paints, hair dyes, electric cable insulations, pottery and ceramics and petrol.

ACTION:

Lead affects the action of enzymes that are responsible for haem synthesis, haemoglobin and cytochrome production. It interferes with mitochondrial oxidative phosphorylation, ATPases, calcium dependent messengers and enhances oxidation and cell apoptosis. It causes haemolysis.

SIGN & SYMPTOMS

ACUTE POISONING

- An astringent and metallic taste.
- Dry throat, thirst, burning abdominal pain, nausea, vomiting, sometimes diarrhea.
- Peripheral circulatory collapse, headache, insomnia, paraesthesias, depression, coma and death.
- Cerebellar ataxia.

CHRONIC POISONING (PLUMBISM/ SATURNISM)

Daily intake of 1-2 mg. of lead is responsible for chronic poisoning. Lead vapour is more dangerous than dust.

SIGNS AND SYMPTOMS:

(1) FACIAL PALLOR: The facial pallor about the mouth might be due to vasospasm is one of the earliest and most consistent sign.

(2) Anaemia: Polycythaemia with polychromatophilia in early stages. Anaemia associated with polychromasia, punctate basophilia, reticulocytosis, poikilocytosis, anisocytosis, nucleated red cells (sideroblasts) with increase in mononuclear cells, whereas polymorphonuclear cells and decrease in platelets.

(3) LEAD LINE: A stippled blue line, called Burtonian line, seen on the gums in 50 to 70% of cases.

(4) COLIC AND CONSTIPATION: Colic of intestines, ureters, uterus and blood vessels are late symptoms. The colic occurs at night and the pain may be very severe. Diarrhoea or vomiting may also occur.

(5) LEAD PALSY: It is a late symptom. Tremors, numbness, hyperaesthesia, and cramps before the actual muscle weakness occurs.

(6) ENCEPHALOPATHY: The symptoms are vomiting, headache, insomnia, visual disturbances, irritability, restlessness, delirium, hallucinations, convulsions, coma and death.

(7) CARDIOVASCULAR SYSTEM AND KIDNEYS: Lead causes vascular constriction, leading to hypertension and permanent arteriolar degeneration. Chronic arteriosclerotic nephritis and interstitial nephritis occur.

(8) REPRODUCTIVE SYSTEM: It causes menstrual abnormalities like amenorrhoea, dysmenorrhoea, menorrhagia, sterility of both sexes, and abortion.

(9) OTHER SYSTEMS: Dyspepsia, anorexia, emaciation, general weakness, exhaustion, irritability, foul breath, headache, vertigo, loss of hair and drowsiness.

FATAL DOSE:

20g. lead acetate;

40g. lead carbonate.

FATAL PERIOD: 1-2 days.

TREATMENT:

(1) Gastric lavage.

(2) Demulcents.

(3) The combination of B.A.L. and calcium disodium versenate or DMSA is effective.

(4) Penicillamine.

(5) Calcium chloride

(6) Peritoneal or haemodialysis.

(7) Symptomatic treatment.

POSTMORTEM APPEARANCES:

IN ACUTE POISONING

- Signs of acute gastroenteritis.

- The mucosa of the stomach may by thickened and softened with eroded patches and may be covered with a greyish-white deposit.
- Bone Marrow shows hyperplasia of leucoblasts and erythroblasts with a decrease in fat cells.

CHRONIC POISONING:

- A blue line may be seen on the gums.
- Paralysed muscles show fatty degeneration.
- The stomach and intestines may show ulcerative or haemorrhagic changes and are contracted and thickened.
- The liver and kidneys are contracted.
- The brain is very pale and greatly swollen.
- The heart may be hypertrophied and there may be atheroma of the aorta and aortic valves.

MLI

- Acute poisoning is very rare.
- Chronic poisoning is more common and is mainly occupational.
- Homicidal poisoning is rare.
- Not Suitable for suicide.
- As abortifacient.
- As cattle poison.
- Lead missiles remaining embedded in the tissues due to gunshot injuries may produce poisonous symptoms in 12 to 48 days.

CHAPTER-11 IRRITANTS- ORGANIC POISON-PLANTS

ABRUS PRECATORIUS

SIGN & SYMPTOMS:

ON INGESTION:

- Abdominal pain, nausea, vomiting, bloody diarrhoea.
- Vertigo, tinnitus, giddiness.
- Cold clammy skin.
- irregular pulse, laboured breathing.
- convulsions,
- Haemolysis, oliguria.
- and death d/t cardiac failure.

INJECTED:

- Resembles viperine snake bite.
- Inflammation, oedema, oozing of fluid.
- Painful swelling and necrosis of muscles and regional lymph node may occur.

TREATMENT:

- Gastric lavage
- Suis should be excised
- Anti abrin
- A mixture of dilute hydrochloric acid and pepsin

- Symptomatic treatment

RICINUS COMMUNIS

SIGN & SYMPTOMS:

DUST OF SEEDS CAUSES:

- Watering of eyes, conjunctivitis
- Rhinitis, acute nasal inflammation
- Headache, pharyngitis, bronchitis
- Dermatitis
- Gastric upset

INGESTION

- GIT: burning pain in throat, colicky abdominal pain, cramping, nausea, thirst, vomiting and diarrhea.
- CNS: vertigo, drowsiness, delirium, convulsions coma
- Uremia, jaundice, feeble pulse, shock and dehydration

- **FATA DOSE :** 10- 20 seeds
- **FATAL PERIOD :** 3-5 days

TREATMENT:

1. Gastric lavage
2. Emetics and demulscents
3. symptomatic

CROTON TIGLIUM

- Croton tiglium, known as purging croton, is a plant species in the family Euphorbiaceae. C. tiglium is also
- Major known chemical constituents are crotonoleic acid, glyceryl crotonate, crotonic acid, crotonic resin, and various carcinogenic phorbol derivatives.
- It is called Jamaal Gota in India.

SIGN & SYMPTOMS

- Hot burning pain from mouth to stomach.
- Salivation
- Vomiting
- Purging
- Vertigo
- Prostration
- Collapse & death.
- When applied to Skin causes burning, redness and vesication.

FATAL DOSE- 4-5 Seeds; 1-2 ml oil

FATAL PERIOD- 6 Hours to 3 Days.

TREATMENT:

1. Gatric Lavage
2. Emetics and demulscents
3. Morphine and atropine
4. symptomatic

POSTMORTEM APPEARANCE:

- Mucosa congested, softened, inflamed, erosions, fragment of seeds may be present
- Dilatation of heart, pleural Hg, oedema of Liver, kidney spleen and lung

CALOTROPIS GIGANTEAN

The leaves and stem when incised yield thick acrid, milky juice.

SIGNS AND SYMPTOMS: Applied to the skin, it causes redness and vesication. When taken by mouth, the juice produces an acrid bitter taste, and burning pain in throat and stomach, salivation, stomatitis, vomiting, diarrhoea, dilated pupils, tetanic convulsions, collapse and death.

FATAL DOSE: Uncertain.
FATAL PERIOD: 6 to 12 hours.

TREATMENT:

1. Gastric Lavage,
2. Ddemulcents
3. Symptomatic.

POSTMORTEM APPEARANCES:

Dilated pupils, froth at the nostrils, stomatitis, and inflammation of gastrointestinal tract are seen. The abdominal viscera and brain are congested.

PLUMBAGO ROSEA (lal chitra) and PLUMBAGO ZEYLANICA (chitra)

The root contains as an active principle, plumbagin, a crystalline glycoside. All parts of the plant are poisonous.

SIGN & SYMPTOMS: Applied externally, roots produce irritation and blisters. Taken internally, there is burning pain from mouth to stomach, vomiting, thirst, diarrhoea, collapse and death.

FATAL DOSE: Uncertain.

FATAL PERIOD: Few days.

TREATMENT:

1. Gastric Lavage,
2. Ddemulcents
3. Symptomatic.

POSTMORTEM APPEARANCES:

Signs of gastroenteritis and congestion of internal organs are found.

SEMECARPUS ANACARDIUM

SIGNS AND SYMPTOMS:

ON EXTERNAL APPLICATION:

- Irritation
- Painful blister which contains acrid serum and eczema increases as the fluid in it touches any other skin part.,
- Itching.
- The lesion resembles a bruise.
- Ulcer is produced, and there may be sloughing.

ON INGESTION:

- Less irritant action.
- Blisters on throat.
- Severe gastrointestinal irritation,
- Dyspnoea, tachycardia, hypotension, cyanosis,
- Absence of reflexes, delirium, coma and death.

FATAL DOSE: Five to ten g.

FATAL PERIOD: 12 to 24 hours.

TREATMENT:

1. Gastric lavage.
2. Demulcent drinks.
3. On Ecxternal application, wash with lukewarm water containing antiseptic.

POSTMORTEM APPEARANCES:

- Blisters are seen in the mouth, throat and stomach which are congested and inflamed.

ERGOT

SIGNS AND SYMPTOMS:

IN ACUTE POISONING

Nausea, vomiting, diarrhoe,

- Giddiness, tightness in the chest, difficulty in breathing, marked muscular weakness and exhaustion.
- Paraesthesias, twitchings or cramps in the muscles.
- Dilated pupils with dimness of vision,
- Bleeding from the nose and other mucous surfaces.
- Rapid and weak pulse, blood pressure is raised.

CHRONIC POISONING (ergotism):

- Tingling and numbness of the skin.
- Vasomotor disturbances leading to dry gangrene
- Sensation of insects creeping under the skin.
- Hallucinations, ataxia, and convulsions.

FATAL DOSE: 2 to 10 g.

FATAL PERIOD: One to several days.

TREATMENT:

1. Wash the stomach.

2. Activated charcoal.
3. (3Iipecac
4. Cathartics.
5. Nitroprusside
6. Diazepam
7. Vasodilators

POSTMORTEM APPEARANCES:

- Not characteristic.
- The internal organs are congested.
- Degeneration of intima of smaller arterioles and thrombus formation

CHAPTER-12 IRRITANTS- ORGANIC POISON- SNAKES

More than 3500 species of snakes are found, out of which around 600 are poisonous. In India more than 200 species are found, out of which more than 50 are poisonous. According to WHO, Approx. 2.5 million cases of poisonous snake bites are reported world-wide causing more than 1,25,000 deaths.

The poisonous snakes may be divided into five families.

(1) (A) Viperidae: Russell's viper, gaboon viper, saw scaled viper, puff adder etc.

(B) Crotalidae: Rattlesnakes, pigmy rattlesnakes, copperheads etc.

(2) Elapidae: Cobras, kraits, mambas, tiger snake, taipan, coral snakes etc.

(3) Hydrophidae or sea snakes: All sea snakes are poisonous.

(4) Colubridae: Boomslangs, bird snake.

(5) Atractaspididae: African and Middle Eastern burrowing asps or stilleto snakes.

SIGNS AND SYMPTOMS:

- Ophitoxaemia is poisoning by snake venom.
- The symptomatology depends upon:

(1) Nature, location, depth and number of bites

(2) Duration for which snake bites

(3) Intensity of anger or fear that motivates the snake

(4) Amount of venom injected

(5) Species and size of the snake

(6) Condition of fangs and venom glands

(7) Age and size of the victim

(8)Sensitivity of vicitim to the venom

(9) Pathogens in the snake

(10) Availability of first aid and medical care.

- The most common symptom is fright and fear of death. This fright and fear may cause victim become semiconscious with cold clammy skin, hypotension, feeble pulse and rapid breathing.
- It may produce psychological shock and death.
- Tetanus or gas gangrene may also develop.
- **COBRA:**
  - Local symptoms start within 6 to 8 minutes.
  - A small reddish wheal develops at the site of bite.
  - The area of bite is tender with radiating burning pain and oozing of bloodstained fluid.
  - Systemic symptoms appear after about 30 minutes.
  - The patient feels sleepy, slightly intoxicated, weakness of legs
  - Nausea and vomiting.
  - Ptosis is followed by ophthalmoplegia. Weakness of extraocular muscle and strabismus.
  - Weakness of the muscles leading to paralysis of the lower limbs. The paralysis extends to the trunk and head which falls forward. The eyelids hang down.
  - After half to one hour, there is excessive salivation, vomiting, headache, vertigo, paraesthesia around the mouth and myalgia.
  - This is followed by paralysis of the facial muscles, palate, jaws, tongue, vocal cords, neck muscles. Muscles of deglutition become progressively flaccidly paralysed due to which there is difficulty in speech and swallowing.

- After approx. two hours, the paralysis is complete.
- Respiratory arrest may occur due to obstruction of upper airway by the paralysed tongue or inhaled vomitus, or due to paralysis of intercostal muscles and diaphragm.
- Development of coma followed by arrest of respirations with or without convulsions and the heart stops.
- Necrosis may develop around the site of bite, if patient recovers.

- **KRAIT:**
  - Symptoms are similar to cobra poisoning, but swelling and burning pain at the site of the bite are absent
  - Convulsions are mild.
  - Feeling of drowsiness and intoxication is more intense.
  - Albumin appears in urine.
- **RUSSELL'S VIPER AND ECHIS CARINATE:**
  - Victim presents minimal or no poisoning.
  - Only few develop serious poisoning, but death is rare.
  - Bite site is red with severe pain.
  - Constant bleeding from bitesite.
  - If less amount of venom is injected, nausea, pain and swelling restricted to below the elbow or knee develops.
  - In moderate case, feeling of intense pain, vomiting, giddiness, sweating, abdominal pain, dilatation of the pupils, insensitivity to light and marked collapse and often complete loss of consciousness. Increased skin temperature with tingling and numbness over the tongue and mouth or scalp and paraesthesia around the wound. Haematuria may be seen.
  - In severe cases, chief symptom is the persisting shock. Blood may show haemoconcentration early followed by decrease in red cells and platelets, with presence of urine contains blood, sugar and protein. A haemorrhagic syndrome with blood-stained sputum, haemorrhages from the gums, rectum, the site of bite, etc., sets in.

- **SEA SNAKES:**
  - Patient develops pain, stiffness and weakness of the skeletal muscles.
  - Polymyositis develops.
  - Trismus occurs in early stage.
  - Later, paralysis beginning with ptosis develops.
  - Death may occur due to cardiac arrest or paralysis of respiratory muscles,

FATAL DOSE:

- Cobra 12 mg.
- Russell's viper 15 mg.
- Echis 8 mg.
- Krait 6 mg. of dried venom.

FATAL PERIOD:

- Cobra- half to six hours,
- viper one to two days.

TREATMENT:

1. Polyvalent antisnake venon (PAV)
2. Antivenene, if PAV is not available.
3. In case of viper bite, prothrombin time should be monitored.
4. Neostigmine with atropine, If neuroparalysis develops.
5. Heparin in case of clotting abnormalities.
6. Tetanus antitoxin or booster dose of tetanus toxoid.
7. Broadspectrum antibiotic
8. Sedatives in case of viper poisons to relieve pain and nervousness.
9. Stimulant to treat collapse

10. Mechanical ventilator in respiratory failure, if needed.
11. Infusion of normal saline or transfusion of blood or plasma.
12. Haemodialysis or peritoneal dialysis.
13. Analgesics for pain, but aspirin should be avoided.
14. Surgical debridement of the blebs, bloody vesicles, and superficial necrosis may be necessary.

POSTMORTEM APPEARANCES:

- Two or occasionally one fang mark.
- Non-poisonous snakes leave a semicircular set of tooth marks.
- Sometimes, the bite marks may not be visible.
- In colubrine bite, the site of bite contains fluid and haemolysed blood causing staining of vessels.
- In viperine bite, discolouration, swelling and cellulitis about the mark and haemorrhages at the site of bite.
- Haemorrhages into the bowel, lungs and in many tissues may be seen.
- Signs of inflammation and congestion in kidney.
- Acute renal failure is cause of death in viper bite.
- Internal organs are congested.

CHAPTER-13 AGRICULTURAL POISON-ORGANOPHOSPHORUS

Many chemicals are used to manufacture pesticides. Pesticides are preparations which are used to control the livestock which harms crops. It includes herbicides, insecticides, fungicides etc.

CLASSIFICATION:

(1) INSECTICIDES OF VEGETABLE ORIGIN: E.g.- nicotine, pyrethrins and rotenone.

(2) CHEMICAL INSECTICIDES:

Inorganic: Phosphorus and compounds of antimony, arsenic, barium, mercury etc.

(3) SYNTHETIC ORGANIC CHEMICAL INSECTICIDES: E.g. - Phosphate esters, Carbamates, Chlorinated hydrocarbons etc.

ORGANOPHOSPHORUS POISONS

They are esters of phosphoric acid. They are used to types of compound-

(A) Alkyl phosphates: E.g. – HETP, TEPP (Tetron), OMPA, Dimefox etc.

(B) Aryl phosphates: E.g. – Parathion, Paraoxon, Methylparathion, Chlorthion etc.

ABSORPTION: They are absorbed by inhalation, through skin, mucous membranes and the gastrointestinal tract..

ACTION: Organophosphates are powerful inhibitors of acetylcholinesterases.

SIGN AND SYMPTOMS –

Symptoms appears very quick on inhalation, and least rapid from absorption through the skin. First Involuntary muscles and secretory glands then voluntary muscles and finally

brain centres are affected. Respiratory symptoms resemble to asthma. Symptoms may appear within 5 minutes to 2-8 hours. Symptoms develop when the cholinesterase level drops to thirty percent of its normal level.

(I) MUSCARINIC MANIFESTATIONS:

SLUDGE:

S = Salivation,

L = lachrymation,

U = urination,

D = defaecation.

G = gastrointestinal distress, and

E = emesis.

Other symptoms are

(1) **Bronchial tree:** Bronchoconstriction, increased bronchial secretions, dyspnoea, cyanosis, pulmonary oedema.

(2) **G.I.T.** : Anorexia, salivation, nausea. vomiting, cramps, diarrhoea,

(3) **Sweat glands:** Increased perspiratin.

(4) **C.V.S.:** Bradycardia or tachycardia, arrhythmias, hypotension.

(5) **Pupils:** Miosis.

(6) **Bladder:** Urinary incontinence.

(II) NICOTINIC MANIFESTATION:

(1) **Striated muscle:** Initiallly contraction followed by paralysis due to persistent depolarisation. Accumulation of acetylcholine Muscle weakness is results in Muscular fasciculations, cramps, weakness.

(2) **Sympathetic ganglia:** Hypertension, tachycardia, pallor, mydriasis.

(III) CNS MANIFESTATIONS: Restlessness, mood swing, headache, tremors, anxiety, drowsiness, confusion, slurred speech, ataxia, generalised weakness, coma, convulsions, depression of respiratory and cardiovascular centres.

Usually all 3 combinations of manifestations apperars simultaneously.

Mild poisoning: Signs and symptoms are: nausea, malaise, fatigue, minimal muscle weakness, cramping without diarrhoea.

Moderate poisoning: SLUDGE and/or tremors, weakness, fasciculations, confusion, lethargy, anxiety.

Severe poisoning: SLUDGE, and respiratory insufficiency, weakness, fasciculations, coma, paralysis, seizures, autonomic dysfunction.

Death may result from respiratory arrest due to failure of respiratory centre, or paralysis of respiratory muscles, or intense broncho-constriction.

CHRONIC POISONING: It is seen in persons engaged in pesticide spraying of crops. Symptoms include weakness, anxiety, gait disorders, muscle cramps, paraesthesias, drowsiness, confusion, irritability and psychiatric manifestations.

FATAL DOSE:

TEPP 50 mg. i.m. or 100 mg. orally.

OMPA 80 mg. i.m. or 175 mg. orally.

Parathion 80 mg. i.m. or 175 mg. orally.

HETP 60 mg. i.m. or 350 mg. orally.

Malathion and diazinon one g. orally.

FATAL PERIOD: In untreated cases. Death occurs within 24 hours, and within ten days in unsuccessfully treated cases. In nonfatal cases, effect lasts for six to thirty hours which disappear in 2 to 3 days, but may persist for two weeks.

TREATMENT -

(1) Remove the patient from the source of exposure. Contaminated cloths should be removed and the exposed areas should be washed with soap and water, followed by ethanol and water.

(2) If eye is affected, it must be washed.

(3) The airway is to be protectrd and if needed tracheastomy may be performed.

(4) If ingested, gastric lavage should be done·with 1:5,000 potassium permanganate solution.

(5) Activated charcoal.

(6) Cathartic.

(7) Avoid physostigmine.

(8) Atropine to treat muscarine effects.

(9) Specific cholinesterase reactivators like diacetyl monoxime (DAM), or 2-pyridine aldoxime methiodide, or pralidoxime chloride or pyridine aldoxime methane sulphonate (P2S).

(10) HI-6 and H10-7 appear to have activity against all known organophosphates.

(11) Diazepam to control convulsions.

(12) oxygen, intubation, atropine and positive pressure ventilation to treat pulmonary oedema and bronchospasm.

(13) Antibiotics to prevent pulmonary infections.

(14) Symptomatic.

POSTMORTEM APPEARANCES:

- Signs of asphyxia are present.

- The face is congested with cyanosis of the lips, fingers and nose.
- Bloodstained forth around the mouth and nose.
- The stomach contents may smell of kerosene.
- The mucosa of the stomach is congested with submucous petechial haemorrhages.
- Respiratory passages are congested and contain frothy haemorrhagic exudate. The lungs show gross congestion, excessive oedema and subpleural petechiae.
- Heart is sometimes soft and flabby.
- The internal organs are congested.
- The brain is congested and oedematous; meninges are congested. Petechial haemorrhages are present. The cholinesterase in erythrocytes and at myoneural junctions is below normal.
- Organophosphorus can be detected in putrefied bodies.

MLI

(1) Persons engaged in the manufacturing and packaging.

(2) Users of these compounds who sprays or dusts in the open as insecticides.

(3) Children of users.

(4) Research workers.

(5) Suicide is very common.

(6) Homicide is rare.

CHAPTER- 14 AGRICULTURAL POISON- ENDRIN

- It is a polycyclic, polychlorinated hydrocarbon belongs to the group of cyclodiene insecticides.
- Its taste is bitter.
- It is commonly known as "plant penicillin", because of its action on various insect pests
- Products available in market contain about 20 to 50% of endrin mixed with petroleum hydrocarbon, such as aromax, which smells like kerosene.
- It is commonly used as sprays.

SIGN AND SYMPTOMS – These begin within one to 6 hours. They are

- Salivation, nausea, vomiting, abdominal pain, rarely diarrhoea,
- Hoarseness of voice, coughing, froth at the mouth and nose, dyspnoea,
- Headache, giddiness, restlessness, hyperirritability, dilated pupils, incoordination, ataxia,
- Mental confusion, tremors, tonic and clonic convulsions,
- Coma and death due to respiratory failure.

CHRONIC POISONING:

Long duration exposure causes cumulative toxicity. Symptoms are loss of weight, weakness, ataxia, tremors, mental changes, oligospermia. It increases the tendency of leukaemias, aplastic anaemia and liver cancer.

FATAL DOSE: 5 to 6 gm.

FATAL PERIOD: One to several hours.

TREATMENT -

(1) Remove exposed cloths and wash contaminated skin with soap and water.
(2) Gastric lavage.
(3) Emetics and cathartics
(4) Activated charcoal.
(5) Cholestyrarnine to increase faecal excretion of organochlorines.
(6) No specific antidote.
(7) Maintain airway, breathing and circulation.
(8) Dextrose, naloxone and thiamine to control mental state.
(9) Diazepam followed by phenobarbital to control convulsions.
(10) Calcium gluconate is useful.

POSTMORTEM APPEARANCES:

- The mouth and stomach contents smell of kerosene.
- Signs of asphyxia are present.
- Endrin resists putrefaction, so can be found in the viscera even some time after death.

MLI

- Occupational
- Accidental.
- Suicidal.
- Homicide is rare.

CHAPTER-15 CARDIAC POISON- ACONITE & DIGITALIS

These are the poisons which have their affinity chiefly on heart directly or indirectly.

These are-

1. Aconite
2. Digitalis
3. Quinine
4. Nicotine

ACONITE

It has different varieties and all varieties and all parts of the plant are poisonous.

Active Ingredient- Aconitine, pseudo-aconitine, indaconitine, picraconitine and aconine.

MOA-

It acts on sodium channels of the cell membranes of excitable tissues. It first excites and then causes paralysis of nerves affecting myocardial, smooth and skeletal muscle. Higher centers of the brain remains functional as victim remains consciousness till the end.

Sign & Symptoms

GIT- There is feeling of burning from mouth to stomach, tingling and numbness in mouth along with salivation, nausea, vomiting and diarrhea. Patient feels dryness in mouth and thirst but is unable to swallow.

Locomotor System- twitching of the muscles with darting pains, cramps, muscular weakness

Occular- Dimness of vision and diplopia, Paraesthesias

CNS- Tingling and numbness felt all over the body, headache, giddiness, profuse sweating, dilated pupils, subnormal temperature, ataxia and slurred speech, hallucinations and convulsions.

CVS- Hypotension, cardiac arrhythmia, initially there is tachycardia, but in later stages bradycardia develops, , oppression in the chest, and death occurs from paralysis of heart or respiratory centres or both.

Fatal Dose: One g. root;

2 to 5 mg. of aconitine;

250 mg. extract.

Fatal Period: Two to six hours.

Treatment:

(1) Gastric lavage with warm water, and weak solution of iodine in potassium iodide

(2) Multiple-dose activated charcoal.

(3) Cathartic.

(4) Atropine may be given.

(5) Symptomatic.

Postmortem Appearances: Findings are similar to asphyxia. Congestion of stomach and duodenum and occasionally ecchymosis may be seen. The lungs, kidneys and brain are congested.

MLI-

The Circumstances of Poisoning are:

(1) Accidental poisoning.

(2) Suicidal poisoning

(3) Homicidal poisoning.

(4) Root as an abortifacient.

(5) Cattle poison.

(6) Arrow poison.

DIGITALIS PURPUREA

Whole plant is toxic in character having multiple cardiac and steroidal glucosides.

Active Ingredient- Digitoxin, digoxin, digitalin and digitonin (glycosides).

MOA- It acts on heart muscles and in toxic doses causes increased excitability of heart muscles.

SIGNS AND SYMPTOMS:

GIT: Anorexia, nausea, vomiting, diarrhoea.

CARDIAC: Arrhythmias: extrasystoles, ventricular tachycardia and fibrillation, atrial flutter and fibrillation, SA· block, A V block.

ENDOCRINE: Gynaecomastia.

Ocular: Transient amblyopia, photophobia, diplopia, blurring, colour aberration, halos.

SKIN: Urticaria.

CNS: Headache, fatigue, muscle weakness, neuro-psychiatric disorders, confusion, anxiety, depression, disorientation, drowsiness, delirium, hallucinations, trigeminal neuralgia.

Cardiovascular collapse is the cause of the death.

FATAL DOSE: 15 to 30 mg. of digitalin:
Four mg. of digitoxin;
Digoxin 10 mg;
Leaf : 2 g.

FATAL PERIOD: 1-24 hours.

TREATMENT:

(1) Stomach wash with a solution of tannic acid.
(2) Evacuation of bowels.
(3) Activated charcoal.
(4) Digoxinspecific antibody fragments (Fab).
(5) Phenytoin to treat ventricular irritability
(6) EDTA to lower the S. Calcium.
(7) Potassium salts to reduce extrasystoles and tachyarrhythmias.
(8) Atropine to treat bradycardia.
(9) Symptomatic.

POSTMORTEM APPEARANCES:

Signs of slight inflammation of the gastric mucosa
Remnant of the leaves may be found in the alimentary canal.

MLI- Accidental Poisoning.

CHAPTER-16 CARDIAC POISON- NICOTIANA TABACUM & QUININE

NICOTIANA TABACUM

In this plant, except the ripe seeds all parts are poisnous. The dried leaves (tobacco, tambaku) contain one to eight percent of nicotine and are used to smoke or snuff or chewed.

ACTIVE INGREDIENT- Nicotine, anabasine and nornicotine. (all Alkaloids)

MOA-

It action is on autonomic ganglia, stimulating it initially, but depressed and blocked at later stage.

SIGN & SYMPTOMS

Local Application- It may causes dermatitis.

ACUTE POISONING:

G.I.T.: Burning acid sensation, nausea, vomiting, abdominal pain, hypersalivation**.**

CARDIOPULMONARY: Tachycardia, hypertension, tachyapnoea (early); bradycardia, hypotension, respiratory depression (late). Cardiac arrhythmias may occur.

C.N.S.: Miosis(excessive constriction of the pupil of the eye), confusion, headache, sweating, ataxia, agitation, restlessness, hyperthermia (early); mydriasis(dilation of the pupil of the eye), lethargy, convulsions, coma (late).

Death may occur from respiratory failure.

CHRONIC POISONING:

G.I.T.: anorexia, vomiting, diarrhoea,

C.N.S.: tremors, impaired memory, amblyopia(impaired or dim vision without obvious defect or change in the eye.)

CARDIOPULMONARY: cough, wheezing, dyspnoea, anaemia, faintness, irregularity of the heart with extrasystoles and occasionally attacks of pain suggesting angina pectoris.

FATAL DOSE: 50 to 100 mg. of nicotine.
15 to 30 g. of crude tobacco.

FATAL PERIOD: Five to 15 minutes

TREATMENT:

(1) Gastric lavage with warm water containing charcoal, tannin or potassium permanganate.

(2) Purgatives and bowel evacuation.

(3) Mecamylamine (Inversine) is a specific antidote given orally.

(4) Protect airway.

(5) Oxygen.

(6) Vasodilators can be given.

(7) Symptomatic.

POSTMORTEM APPEARANCES:

- Findings are similar to asphyxia.

- Brownish froth at mouth and nostrils.
- Haemorrhagic congestion of GI tract, and pulmonary oedema are seen.
- Remnants of leaf fragments may be found in stomach or it may smell like nicotine.

MLI-

The Circumstances of Poisoning are:

(1) Accidental poisoning.

(2) Suicidal poisoning

(3) Homicidal poisoning.

QUININE

The bark of the plant is the main source of alkaloids. Quinine is white needle-shaped, odourless crystals and has a bitter taste.

ACTIVE INGREDIENT- quinine, cinchonidine and other alkaloids.

MOA- Strong protoplasmic poison with anaesthetic and sclerosing action. It causes stimulation of CNS followed by depression.

SIGNS AND SYMPTOMS:

- **Ear:** Ringing in the ears, partial deafness,
- **Eyes:** Disorders of vision, fixed and dilated pupils.
- **GIT:** Abdominal pain, vomiting, diarrhoea,
- **C.N.S.:** Headache, giddiness, confusion of thought, muscular weakness,
- **Skin:** Itching, erythematous or urticarial rash on the skin,

- **C.V.S.-** Methaemoglobinaemia, tachycardia, hypotension, cyanosis, delirium and coma.

Death occurs from respiratory failure

CINCHONISM OR QUINISM- It is result of repeated therapeutic or overdose of quinine. Symptoms are tinnitus, vertigo, deafness, diplopia, scotoma, blindness, skin rash, hypoglycemia and cardiac arrhythmias

FATAL DOSE: 8 to 15 g.
Quinidine 4 to 6 g.

FATAL PERIOD: 6 hours.

TREATMENT:

(1) Gastric lavage should be performed and a concentrated solution of magnesium sulphate left in the stomach for rapid elimination of poison.
(2) Activated charcoal.
(3) I.v. flid for diuresis.
(4) Sodium bicarbonate to treat cardiac Toxicity.
(5) Oxygen
(6) Blocking of bilateral stellate ganglion can be done to protect vision.
(7) Symptomatic

PM APPEARANCES:

Congestion of organs
Haemolysis of red cells.
Renal tubules may be blocked by haemoglobin.

MLI-

The Circumstances of Poisoning are:

(1) Accidental poisoning.

(2) Suicidal poisoning

(3) Abortifacient.

CHAPTER-17 SPINAL POISON

STRYCHNOS NUX VOMICA

Strychnine (kuchila) is a powerful alkaloid obtained from the seeds of strychnos nux vomica, and other species of strychnos. The seeds of nux vomica in the ripe fruit are poisonous. It occurs as

- Colorless,
- odorless,
- rhombic prisms,
- bitter taste.

ACTIVE INGREDIENTS- Strychnine and brucine are chief ingredients. The seeds also contain a glucoside, loganin. The bark contains only brucine.

SIGNS AND SYMPTOMS:

- If swallowed uncrushed, symptoms are not developed as they are not dissolved and are passed entire in the faeces.
- When crushed seeds are taken, the symptoms are delayed for an hour or more.
- If the alkaloid is swallowed, the symptoms occur very rapidly.
- Bitter taste in the mouth, sense of uneasiness and restlessness, feeling of suffocation and dysphagia.
- The convulsion precedes by increased acuity of perception, increased rigidity of muscles, and muscular twitchings. Convulsions are at first clonic, but eventually become tonic. During the convulsions, the face is cyanosed with anxious look, staring eyes, prominent eyeballs and dilated pupils.
- In between the convulsions the muscles are completely relaxed, and the patient looks well but exhausted, and the breathing is resumed. The cyanosis lessens, cold perspirations cover the skin; dilated pupils may contract. After 5 to 15 minutes or on slightest impulse, e.g. a sudden noise, a current of air, or gently touching the patient, another convulsion occurs.

- Risus sardonicus results from contraction of the jaws and facial muscles in which the corners of the mouth are drawn back.
- The mouth is covered with bloodstained froth.
- The body arches in hyperextension (opisthotonos) position. The whole body becomes rigid attending a bow-like form.
- Consciousness is not lost and the mind remains clear till death.
- In fatal cases, the convulsions come rapidly one after another, and increase in severity and duration. Death usually occurs after four to five convulsions.

FATAL DOSE: 50 to 100 mg; one crushed seed.

FATAL PERIOD: One to two hours.

TREATMENT:

1. The first step is the effective control of convulsions. Diazepam then phenobarbital cis given.
2. Shift the patient in a dark room and silent room.
3. Short-acting barbiturates.
4. Gastric lavage.
5. Activated charcoal or Tannic Acid.
6. Acidify the urine.
7. Symptomatic Treatment.

POSTMORTEM APPEARANCES:

- They are not characteristic.
- Rigor mortis appears early.
- There may be signs of asphyxia.
- Extravasated blood may be found in the muscles.

- The mucosa of the stomach and duodenum may show patches of ecchymoses or congestion.
- The lungs, liver, kidneys, brain and spinal cord are congested.

MLI:

1. Homicidal poisoning.
2. Suicidal poisoning is rare due to painful death.
3. Accidental poisoning.
4. As cattle poison.
5. As arrow poison.
6. As an aphrodisiac.

CHAPTER-18 CNS DEPRESSANTS-ALCOHOL

Narcotic: Initially this term was used for opioids but now used for any illicit psychoactive substance by law enforcement agencies. These are substances which causes sleep.

- **OPIATE:** Natural alkaloids derived from the poppy plant. „
- **OPIOIDS:** Used for any substance which are capable of producing opium-like effects.
- **SEDATIVES:** These drugs decrease activity, moderate excitement, and produces a feeling of calmness.
- **HYPNOTICS:** A drug that produces drowsiness and produces a state of sleep, resembling natural sleep.

CLASSIFICATION OF CNS DEPRESSENTS:

(1) Ethyl alcohol.

(2) General anaesthetics.

(3) Opioid analgesics.

(4) Sedative hypnotics.

ALCOHOL

It produces intoxication.

CONCENTRATION:

The approximate percentage in some of varieties of alcohol is as follows:

- Vodka 60 to 65% Rum,
- liquors 50 to 60%
- Whisky, gin, brandy 40 to 45%
- Port, sherry 20%
- Wine, champagne 10 to 15%
- Beers 4 to 8%

A standard drink is roughly 45 ml of distilled spirits (15g. of alcohol) or 150 ml of wine (llg. alcohol) or 350 ml of beer (13g. alcohol). The safe limit of alcohol consumption is 210 g of alcohol in men and 140 g of alcohol in women per week.

ACTION

It acts on the CNS as a depressant of sensitive cells of cerebral cortex (centers regulating conduct, judgment and self-criticism), leading to unrestrained behavior. It depresses the vital centers of medulla producing coma and death. Alcohol also acts a hypnotic, diaphoretic, and in small doses as an appetizer.

SYMPTOMS:

ACUTE ALCOHOL POISONING

It results from large quantity in small doses at short intervals or in a large dose at a time. There are three phases of intoxication.

1. **STAGE OF EXCITEMENT :**
    - First a feeling of well-being and a certain slight excitation.
    - The actions, speech and emotions are less restrained.
    - There is increased confidence and a lack of self-control, which is a constant feature of alcoholic poisoning.
    - The person may disclose secrets. (‘in vino veritas’—in wine there is truth)
    - Normal good manners are forgotten.
    - Impairment of cognitive function, motor coordination and sensory perception occur above 30 mg% concentration and slurring of speech, unsteadiness, drowsiness, impaired reasoning and memory, reduced perception and decreased concentration above 50 mg%.
    - Mental concentration is poor and judgement impaired.
    - Deterioration of attention.
    - Recall memory is affected, person cannot recall certain situation, or names.
    - Sensetivity to pain decreases at 80 mg%.
    - The emotions are affected.

- Alcohol increases the desire for sex, but affects the performance

(2) STAGE OF INCOORDINATION: Alcohol content of the blood increases to 150 to 250 mg/100 ml.

- The sense perception and skilled movements are affected.
- The inhibitory action of the higher centre's loss increases resulting in alteration in the conduct of the individual.
- He may become carefree, cheerful, ill-tempered, irritable, excitable, quarrelsome, sleepy.
- There is clumsiness and incoordination in the fine and more skilled movements. Speech and fine finger movements are affected.
- Nausea and vomiting.
- The smell of alcohol in breath.
- Face becomes flushed and pulse is rapid.
- Sense of touch, taste, smell, and hearing are diminished.
- The temperature becomes subnormal. Heart rate is increased.

(3) STAGE OF COMA:

- It develops at or above 300 mg% of blood level of alcohol.
- Motor and sensory cells are deeply affected, speech becomes thick and slurring.
- Coordination is markedly affected, patient become giddy, stagger and possibly to fall.
- Coma with stertorous breathing.
- Rapid pulse with subnormal temperature.
- The pupils are contracted, but dilate on stimulation by pinching or slapping with slow return (Me Ewan Sign).

CHRONIC ALCOHOL POISONING- This condition arises in alcohol addicts who cannot stop drinking or who experiences withdrawal symptoms, if they stop drinking. These patients have irreversible somatic or brain changes caused by alcohol. Symptoms are:

- Impaired social or occupational functioning.
- Nausea, vomiting, anorexia, diarrhea.
- Jaundice.
- Tremors of the tongue and hands, insomnia, loss of memory, impaired power of judgement.
- Hypoproteinaemia and general anasarca.
- Peripheral neuritis and dementia.
- Coma, which may lead to death.

FATAL DOSE: 150 to 250 ml. of absolute alcohol consumed in one hour.
FATAL PERIOD: 12 to 24 hours.

TREATMENT:

ACUTE POISONING

(1) Gastric lavage.
(2) Bowel lavage.
(3) The patient must be kept warm, and if there is congestion of the brain, ice bags should be applied to the head.
(4) One litre of normal saline with 10% glucose, 100 mg. thiamine and 15 units of insulin are useful.
(5) If the coma deepens, nerve stimulants, such as caffeine and strychnine should be used.
(6) Artificial respiration, if there is difficulty in breathing.
(7) Haemodialysis or peritoneal dialysis.

CHRONIC POISONING

(1) Disulfiram (Antadict, Esperal) – This drug is used to develop aversion against alcohol. It inhibits aldehyde dehydrogenase. It inhibits the bio-transformation of ethanol beyond the acetaldehyde stage leading to accumulation of acetaldehyde in the blood and tissues producing unpleasant symptoms, such as flushing, palpitation, anxiety, sweating,

headache, abdominal cramps, nausea and vomiting, due to which the patient dislikes alcohol.

(2) Citrated calcium carbimide (Temposil).
(3) Chlorpromazine.
(4) Clonidine.
(5) Chlormethiazole.

POSTMORTEM APPEARANCES:

IN ACUTE POISONING

- Alcoholic odor on opening the body cavity.
- Acute inflammation of stomach with a coating of mucus.
- The brain, liver and lungs are congested.
- Smell of alcohol in the viscera.
- The blood is usually fluid and dark.
- Oedema and congestion of the brain and meninges.

IN CHRONIC POISONING

- Signs of malnutrition.
- Deep reddish-brown discoloration of gastric mucousa with patches of congestion or effusion.
- Congested, enlarged liver with fatty inftltration.
- Liver surface is pale and greasy with patchy yellowish areas within normal hepatic parenchyma.
- Cirrhosis of liver with 5 to 10 mm nodules. Liver becomes smaller and contracted to a hard, greyish yellow block.
- The kidneys show granular degeneration.
- The heart is dilated with fatty degeneration.

METHYL ALCOHOL

It is

- colorless,
- volatile liquid,
- odor similar to ethyl alcohol,
- has burning taste.

USES-

It is present in some home-made beverages, antifreeze, paint removers, dyes, resins, adhesives and varnish.

ACTION

It causes ethanol-like CNS depression and increased serum osmolality. It causes metabolic acidosis and retinal toxicity.

SIGNS AND SYMPTOMS:

- Symptoms of drunkenness.
- Nausea, vomiting and pain or severe cramps in the abdomen.
- Headache, dizziness, neck stiffness, confusion, vertigo.
- Muscular weakness.
- Depressed cardiac action and hypothermia.
- Dyspnoea and cyanosis.
- Odour in the breath.
- Delirium and coma.
- Toxic effect on the liver and kidneys (acute tubular necrosis) and on highly specialised nerve elements.
- Urine is acidic and contain acetone and a trace of albumin.
- The pupils are dilated and fixed.

- Visual disturbances like photophobia and blurred or misty vision (snowfield vision), seeing spots, central and peripheral scotomata, decreased light perception, concentric diminution of visual fields for color.
- An increased osmolal gap accompanied by visual symptoms suggest methanol poisoning.

FATAL DOSE: 60 to 200 ml.

FATAL PERIOD: 24 to 36 hours; may be delayed for 2 to 4 days.

TREATMENT:

(1) Gastric lavage
(2) Activated charcoal.
(3) Ethanol is the antidote.
(4) 60 ml of ethyl alcohol in 200 ml fruit juice can be given orally.
(5) 4 methyl pyrazole (4MP, or fomepizole).
(6) Folinic or folic acid 50 to 75 mg.
(7) Blood sugar should be measured frequently while ethanol is being given, as it may cause hypoglycaemia, especially in children.
(8) Crystalloid therapy, dextrose, thiamine, and phosphate for alcoholic ketoacidosis.
(9) Place patient in a left lateral decubitus position with head down to avoid aspiration of vomitus.
(10) Cover the eyes to avoid light.
(11) Keep the airway clear.

POSTMORTEM APPEARANCES:

- Cyanosis.
- Absence of postmortem clotting of the blood.
- The pyridine may give the skin a purple colour.
- Congestion and inflammation of mucousa of the stomach and the duodenum with small haemorrhages.

- Small or large intestine or both are contracted resembling a thick pipe of a very narrow lumen.
- The lungs are congested and oedematous.
- The brain is oedematous and shows local haemorrhage.
- The mucosa of the bladder is often congested.
- The liver shows fatty change and may show necrosis also.
- Tubular degeneration of the kidneys.

MLI

- Accidental Poisoning.
- Used for intoxication, when ethyl alcohol is not available.

CHAPTER-19 NARCOTIC POISONS-OPIUM

Opium (afim) is the dried juice of the poppy (Papaver somniferum). It is cultivated in India and other Eastern countries, only under the license. The unripe capsule is incised and the white juice collected dried to obtain opium.

Ripe and dry poppy capsules contain a trace of opium and are used for their sedative and narcotic action. Their warm decoction is used locally as a sedative fomentation and poultice. Poppy seeds (khaskhas) are white, harmless, demulcent and nutritive and are used as food. The oil from the seeds is used for cooking purposes.

ACTIVE INGREDIENTS:

It contains about 25 alkaloids. These form two chemically different groups:

(a) The phenanthrenes: morphine (about 10%), codeine (about 0.5%), and thebaine (about 0.3%), which are narcotic, and

(b) The isoquinoline group: papaverine (about 1%), and narcotine (about 6%), which have mild analgesic but no narcotic properties.

CLASSIFICATION:

(1) Natural: morphine, codeine.

(2) Semi-synthetic: heroin, hydromorphone, oxymorphone, oxycodone.

(3) Synthetic: meperidine, methadone, levarphanol tartrate, paregoric, diphenoxylate, fentanyl, propoxyphene.

ACTION:

They are similar to endorphins. The drug activates the natural receptor site of endorphins and depresses all centres except oculomotor, vomiting and sweating. It is a peripherally acting analgesic.

SIGN & SYMPTOMS:

Local contact of morphine causes erythema, urticaria and itching dermatitis in sensitive person. Symptoms appears within half hour of ingestion of opium and within 3 or 4 minutes if injected. It first stimulates, then depresses and finally paralyses the nerve centres.

(1) STAGE OF EXCITEMENT:

- This stage is absent in case of large dose.
- Increased sense of well-being.
- Increased mental activity, freedom from anxiety, talkativeness, restlessness or even hallucinations.
- Flushing of face.
- Maniacal condition may be present.

(2) STAGE OF STUPOR:

- Headache, nausea, vomiting.
- Incapacity for exertion, a sense of weight in the limbs, giddiness and drowsiness.
- The subject lies motionless, with eyes closed as if in a sound sleep from which he may be aroused at first, but soon passes into stupor and coma.
- Contracted pupils.
- Cyanosis of face and lips.
- Itching sensation all over the skin.
- The pulse and respirations are normal.

(3) STAGE OF COMA:

- The patient passes into deep coma from which he cannot be roused.
- The muscles become flaccid and relaxed and all reflexes are abolished.
- The face is pale.
- Conjunctivae congested.
- Pupils are contracted to pinpoint size and do not react to light.
- Pupils dilate during the agonal asphyxial phase caused by respiratory depression and ultimate paralysis.
- All the secretions become absent except sweat with cold skin.
- Temperature is subnormal.
- Hypotension with slow pulse

- Breathing is slow and stertorous and may be reduced to 3 to 4 per minute.
- Odor of opium in breath.
- In case of fatal termination, lividity of the surface increases with slow, irregular and imperceptible pulse. Respiration becomes Cheyne-Stokes in type, and finally dies from asphyxia in deep coma.

CHRONIC POISONING (MORPHINISM; MORPHINOMANIA):

Its use as an aphrodisiac and to produce a sense of euphoria develops habit. Opioid dependence is may also be present in patients with chronic pain syndromes. Physicians, nurses and pharmacists may also be abusers due to easy availability.

Usually dependence is produced by morphine and heroin. Heroin is more addicting than morphine. Addicts can tolerate 3 to 6 g. per day. Symptoms are:

- Dry skin
- Initially user feels feeling of relief and well-being, but on taking large doses, there is disinterest, and recurring periods of depression follow.
- Restless and irritable and sleep is disturbed by dreams.
- Insomnia.
- Loss of memory, mental fatigue and gradual intellectual and moral deterioration .
- Hallucinations may occur.
- Constipation, contracted pupils, anorexia, emaciation and weakness.
- Impotence.

TREATMENT:

IN ACUTE POISONING

(1) Gastric lavage.
(2) Activated charcoal.
(3) Bowel wash.
(4) Establish adequate airway, endotracheal intubation can be done, if necessary.
(5) Atropine is not recommended as it can cause death by paralysing the motor and sensory nerves just like morphine.

(6) Naloxone hydrochloride is a specific opioid antagonist.

(7) Nalmefene as opioid antagonist.

(8) Coma Cocktail: In comatose patients where the identity of poison is not known, 100 ml of 50% glucose, 100 mg. thiamine and 2 mg. naloxone i.v. should be given.

(9) Dextrose 50 ml. of 50% solution i.v. and thiamine 100 mg.

(10) Physostigmine

(11) Amiphenazole

(12) In the early stage, patient should be made to walk in the open air to help excretion, but is contraindicated if poison is absorbed.

(13) If coma is deepen, artificial respiration should be given.

(14) Analeptics may be given.

(15) Bupernorphine and LAAM are considered effective in long-term management.

(16) Symptomatic treatment.

IN CHRONIC POISONING

(1) Gradual withdrawal of drug.

(2) Methadone.

(3) Dihydrocodeine or codeine.

(4) Propranolol.

(5) Tranquilizers or sedation at bed time.

(6) Psychiatric counselling.

POSTMORTEM APPEARANCES:

- Signs of asphyxia are prominent.
- The face and the nails are cyanosed.
- Froth is seen at the mouth and nostrils.
- Postmortem staining is well-marked and cyanotic.
- The smell of opium is noticed on opening body, if putrefaction is not set in.
- The stomach may show remnant of opium.
- The trachea and bronchi are congested and covered with froth.

- The lungs are oedematous and congested.
- The brain, meninges and abdominal organ are congested.
- The blood is usually dark and fluid.
- Opium disappears rapidly from the cadaver.

MLI

(1) As suicidal poison because death is painless.

(2) Rarely homicidal.

(3) Poisoning may occur in addicts.

(4) Accidental poisoning.

(5) Cattle poison.

(6) To dope race horses.

(7) Used to gain confidence for doing some bold act.

CHAPTER-20 SEDATIVE-HYPNOTIC-BARBITURATES

They are

- White,
- Crysalline,
- Odorless powders,
- Bitter taste.

ACTION: They act as CNS depressant.

CLASSIFICATION:

(1) **Long-acting:** Barbitone, phenobarbitone, methyl phenobarbitone, diallylbarbituric acid, mephobarbital, phenytoin.

(2) **Intermediate-acting:** Amobarbitone, butobarbitone, probarbitone, sodium amytal, a pro barbital, vinbarbital, allobarbitone.

(3) **Short acting:** Cyclobarbital, pentobarbital, seconal, ortal, amobarbital, cyclobarbitone, quinalbarbitone.

(4) **Ultra-short acting:** Pentothal sodium, kemithal sodium, thiamylal sodium.

SIGNS AND SYMPTOMS :

ACUTE POISONING

A single large or repeated small dose causes acute poisoning. The symptoms are-

- Drowsiness.
- Confusion, excitement, delirium, and hallucinations.
- Ataxia, vertigo, slurred speech, headache, paraesthesias.
- Subjective visual disturbances.
- A stupor progressing to deep coma, with inhibition or loss of superficial and deep reflexes, and gradual loss of response to painful stimuli.

- The Babinski toe sign may become positive.
- Respirations may be rapid and shallow or slow and labored.
- Fall in cardiac output and an increase in capillary permeability leading to an increase in the extracellular fluid.
- Cardiovascular collapse evidenced by cyanosis, hypotension, weak rapid pulse, and cold clammy skin occurs.
- Contracted pupils with sensitivity to light.
- Decreased peristalsis in deeply comatose patient is bad prognostic sign.
- Scanty or suppressed urination that may contain sugar, albumen and haematoporphyrin.
- Incontinence of urine and faeces may occur.
- Sub normal body temperature, fever indicates bronchopneumonia.
- Respirations become irregular, sometimes Cheyne-Stokes in character and finally stop.
- Delirium, hallucinations, ataxia. paraesthesias, loss of reflexes, hypotension, cyanosis, stupor progressing to coma.
- Blisters on the skin in sites where pressure has been exerted between two skin surfaces, often on an area of erythema strongly suggest barbiturate poisoning.

CHRONIC POISONING:

Tolerance develops rapidly and cross-tolerance with alcohol is also developed, when barbiturates are used therapeutically in epilepsy or psychoneurotic patients. Symptomds are:

- Symptoms resembles to symptoms of chronic alcoholism with progressive impairment of cerebral function, dysarthria, ataxia and depression.
- Tendon reflexes may be depressed.
- Hypertonia and tremors of Parkinsonian type may also be present.
- The impairment of mood, behaviour, and intellectual functions causes social deterioration.
- Withdrawal symptoms appear within a day and presents as anxiety, nausea, vomiting, weakness, hypotension, tremors and disturbances of vision. Convulsions may occur.

FATAL DOSE:

Short-acting: One to two g.

Medium-acting: Two to 3 g.

Long-acting: Three to 5 g.

FATAL PERIOD: 1-2 days.

TREATMENT:

(1) Gastric lavage.

(2) Bowels wash.

(3) There is no specific antidote.

(4) 'Scandinavian method', uses anti-shock measures, maintenance of patent airway, and adequate respiratory support.

(5) Fluid replacement therapy.

(6) Dopamine for shock.

(7) Normal saline with five percent glucose i.v. increases the rate of excretion.

(8) Artificial respiration and oxygen.

(9) Keep patient warm and remove mucus the throat.

(10) An endotracheal tube may be left in situ for the first 3 days, but after this a tracheastomy should be done.

(11) Noradrenaline to treat shock and low blood pressure.

(12) Haemodialysis

(13) Charcoal haemoperfusion.

(14) Forced alkaline diuresis.

(15) Mini-heparinisation, elastic stockings, and inflable cuffs for deep-vein thrombosis and thromboembolism.

(16) Antibiotics to minimise risk of pneumonia.

(17) Symptomatic treatment.

POSTMORTEM APPEARANCES:

- Signs of asphyxia.

- Cyanosis is present.
- Remnant of barbiturate may be seen in the stomach.
- The gastric mucosa may be eroded. The fundus may be thickened, granular and haemorrhagic. The cardiac end and lower oesophagus may be eroded from regurgitation.
- Congestion and oedema of lungs with black discoloration. Petechial haemorrhages may be present in the lungs and on the pleura and pericardium.
- Whole venous system is engorged with dark deoxygenated blood.
- Kidneys shows degeneration of the convoluted tubules.

MLI

- Suicidal poison.
- Rare homicidal poison.
- Accidental poisoning may result due to "automatism" (involuntary suicide).

CHAPTER-21 DELIRIANT- DATURA FASTUOSA

There are nine varities of this plan out of Two important varieties are:

(1) **Datura alba**, a white flowered plant, and

(2) **Datura niger**, a deep purple flowered plant.

Fruits are spherical in shape having spines. The fruits contains yellowish-brown seed. The flowers are bell-shaped. All parts of these plants including nectar are poisonous, especially the seeds and the fruit.

ACTIVE INGREDIENT- Hyoscine (scopolamine), hyoscyamine, and atropine

MOA-

It first stimulates the higher centres of brain, motor centres and then depresses and ultimately causes paralysis. The respiration is also first stimulated, then depressed, and the heart centre is stimulated.

SIGN & SYMPTOMS

LOCAL APPLICATION- It may causes dermatitis. The pollen can cause unilateral mydriasis (cornpicker's pupil).

INGESTION- In case of eating seeds, symptoms appear within half an hour, while in case of decoction, symptoms appears in a few minutes and if alkaloids are ingested, symptoms develops immediately.

MENTAL SYMPTOMS- Restlessness, agitation, confusion, giddiness, staggers (loss of balance) as if drunk. Patient is in state of delirium, he can’t recognise relatives or friends. Delirium is restless and purposeless; in early stages, excitement, talkativeness and incoherence are present. The patient may be silent but usually he is noisy, tries to run away from bed, picks the bed clothes,

(carphologia), tries to pull imaginary threads from the tips of his fingers, threads imaginary needles. Hallucinations of sight and hearing and delusions are present.

G.I.T - Bitter taste, dryness of mouth and throat, dysphagia, burning pain in the stomach.

EYES- Congested conjunctivae, dilatation of pupils with loss of accommodation for near vision, temporary blindness, photophobia and diplopia. Sluggishness of light reflex followed by absent reflexes.

C.V.S. - Tachycardia, full, bounding pulse, but becomes weak and irregular, and the respirations are increased.

C.N.S. - Muscle tone and deep reflexes are increased, and there may be muscular spasm or convulsions. Urinary retention and inability to pass urine occurs. With advancement in effect of poison, excitement suppresses in one to two hours, and then patient goes into deep sleep or coma which may end rarely in death from respiratory paralysis. The patient may also recover from this condition within 2 to 3 days but usually distinct improvement occurs in 24 hours.

SKIN: Scarlatinal rash or exfoliation of the skin. The skin is dry and hot.

OTHERS- Hoarseness of voice, difficulty in talking, flushed face, rise in temperature.

Remember 8 D's: Dryness of mouth, dysphagia, dilated pupils, dry; hot skin, drunken gait, delirium, drowsiness, death due to respiratory failure.

FATAL DOSE: 0.6 to one g. (100 to 125 seeds).

FATAL PERIOD: 24 Hours.

TREATMENT:

(1) Emetics.

(2) Gastric lavage with weak solution of tannic acid.

(3) Activated charcoal

(4) A cathartic.

(5) Frequent lower bowel evacuation.

(6) Physostigmine as antidote.

(7) Pilocrapine nitratre is useful.

(8) Morphine is contraindicated as it causes depression of the respiratory centre.

(9) Bromides and short-acting barbiturates to control Delirium. Ether or chloroform is more beneficial.

(10) Light diet, and free purgation to increase intestinal movement and removal of remnant.

(11) Symptomatic.

POSTMORTEM APPEARANCES:

- Findings are not characteristic, but are similar to asphyxia.
- Seeds or their fragments may be found in the G.I. tract.
- The stomach may show slight inflammation
- Oedema in lungs.
- Putrefaction is delayed for a long time by seeds.

MLI-

The Circumstances of Poisoning are:

(1) To stupefy a victim before attempting crime, crushed or powdered seeds or an extract is mixed with edible item.

(2) Homicide is very rare.

(3) As abortifacient.

(4) As an aphrodisiac.

(5) Accidental cases occur- In children by eating the fruits.

(6) The seeds and leaves are mixed with tobacco or ganja and smoked in a pipe.

(7) A decoction of seeds is mixed in liquor increase intoxication.

(8) As love philter.

A person suffering from delirium of datura is not criminally responsible for his acts.

CHAPTER-22 DELIRIANT-CANNABIS SATIVA OR INDICA

It is also known as Indian hemp. Its farming is restricted by law. The female plant is taller, about 4 to 6 metres, and has more darker and luxuriant foliage than the male.

COMMON NAMES- Pot, grass, dope, weed, hash, mary jone, M.J., hashish or bhang.

ACTIVE INGREDIENT- The active contents are present in its resin. Active ingredients are cannabinol, which is inert in nature but when heated, it is partly converted to the active isomere tetrahy-drocannabinols (THC). In both, male and female, all parts of the plant contain the active material except stem, root and seeds.

MOA- It is a psychoactive drug and a CNS stimulant. THC is metabolised in the liver and excreted in the urine and faeces.

USES- It is used as illegal drug. It is used in the following forms:

(1) Bhang (siddhi, sabji): It is prepared from the dried leaves and fruit shoots. It is used as decoction. It contains 15% of active principle. Fresh bhang is highly intoxicating and narcotic while stored bhang is mildly stimulating and pleasure-giving.

(2) Majoon: It is a sweet prepared with bhang used to increases the appetite and sexual desire.

(3) Ganja: It is prepared from the flower tops of the female plant. It is mixed and smoked with tabacco. It contains 15 to 25% of the active principle. Ganja mixed with tobacco in cigarettes is used for smoking and called Reefer or Joint.

(4) Charas or hashish: It is the resin discharged from the leaves and stems of the plant, and it contains 25% to 40% of the active principle It is mixed and smoked with tobacco in a pipe or hukka.

SIGNS AND SYMPTOMS:

Symptoms are developed soon after smoking and lasts for 1-2 hour and within half-an-hour after swallowing and lasts for 2 to 3 hours.

In small dose, the symptoms are very mild including euphoria, passivity, heightening of subjective experiences, and disorientation.

In moderate doses these symptoms are intensified. It includes impaired immediate memory function & thought patterns, lapses of attention and a subjective feeling of unfamiliarity.

In high doses, body image changes with depersonalization and marked sensory distortion.

Symptoms of Intoxication:

(A) Psychiatric:

(1) Feelings of detachment, cleverness, depersonalization, euphoria, relaxation, feeling of well-being, dreaminess, sleepiness, self-confidence, jocularity, laughing, silliness, rapidly changing emotions.

(2) Thought processes: irrelevant thoughts, altered reality testing, decreased duration of concentration and attention, altered sense of identity, disorientation.

(3) Sensory novelty and increased sensitiveness, vivid images, illusions and hallucinations.

(4) Feelings of precordial distress and tightness in chest, fear of dying.

(5) Altered sense of time and space. altered sexual feelings.

(6) Maladaptive behavioural effects: sense of judgement altered, failure to meet responsibilities.

(7) Rapid speech, impaired, talkative, poor immediate memory. User feels that they can fly.

(B) Physical:

(1) G.I.T. - Increased appetite and thirst, slight nausea, dry mouth.

(2) C.N.S. - Heaviness and pressure in the head, dizziness, dysaesthesia (an abnormal unpleasant sensation felt when touched), sleepiness, paraesthesias (Tingling or prickling), restlessness, ataxia, tremors,

(3) C.V.S. – Tachycardia.

(4) Renal- Increased urinary frequency.

(5) Eyes- Injected conjunctivae (red eye caused by dilation of blood vessels).

The characteristic odor of it may be present in case of smoking. Psychiatric patient or sensitive individuals may become paranoid after a relatively low dose. Death is very rare due to respiratory failure.

CHRONIC POISONING: The use of the drug in small quantities for long period is not harmful due to development of tolerance and but individual becomes psychologically dependent. In case of excess use, degeneration of the central nervous system occurs. Chronic use causes gynaecomastia. There is loss of appetite, weakness, wasting, tremors, sleepy facial expression, vacant look, red eyes, impotence and moral and mental deterioration. Rarely auditory and visual hallucinations and delusions of persecution is present with insanity.

Run Amok- It is a condition that can develop in chronic users where he suffers from psychic disturbance having period of depression which is followed by violent attempts to kill people. Patient develops homicidal mania. He first kills a person against whom he

may have real or imaginary enemity and then kills anyone that comes in his way until the homicidal tendency lasts. Then he may commit suicide or may surrender himself.

MLI-

The Circumstances of Poisoning are:

(1) Accidental ingestion or inhalation.
(2) Overindulgence
(3) Majoon and charas are used to stupefy to commit crime.
(4) It is taken to strengthen the nerves by criminals before committing a crime.
(5) As aphrodisiac.
(6) To overcome hunger and thirst, and believe that it helps in the concentration of mind towards meditation.

CHAPTER-23 DELIRIANT- COCAINE

- It is obtained from the leaves of Erythroxylum coca, which grows wild in South America, India, Java, etc.
- It is a colourless, odourless, crystalline substance with bitter taste.
- **COMMON NAME-** Coke, snow, Cadillac, white lady, crack, pasta, bazooka, and speed-ball.

USES-

- Crack is prepared by combining cocaine with baking soda and water, which is suitable for smoking.
- It is used as local anaesthetic.

ACTIVE INGREDIENT- Ecgonine, hygrine and cinnamyl cocaine. The leaves contain about 0.5 to 1% cocaine.

MOA-

- It decreases the sensitivity of the terminal nerves and causes vasoconstriction at the site of application.
- It is a powerful stimulant of CNS for a short time, followed by depression.
- The euphoric effect is subjected to the release of dopamine, serotonin and other neurotransmitters.

SIGN & SYMPTOMS

- On inhalation it acts within 1-3 minutes; while when used i.v. or smoked, action appears within seconds and peak action is in 3 to 5 minutes; when applied

topically to the nasal mucosa, it peaks in 20 to 30 minutes and when ingested orally it peaks within 60 to 90 minutes.

- Its action is short, and as such it has to be taken every half to one hour to maintain a high.
- Symptoms appears in following stages-

(1) STAGE OF EXCITEMENT:

- **G.I.T. -** Bitter taste, dryness in the mouth, dysphagia,
- **MENTAL –** Patient feels well with loss of depression and fatigue. The patient may be excited, restless and talkative, but becomes calm and dull later. The patients may have hallucinations. Mania may develop.
- **CARDIOPULMONARY-** The pulse is rapid, respirations rapid and deep. It produces hypertension that may cause cerebral bleeding.
- **C.N.S. –** Headache may present. The reflexes are exaggerated, and there may be tremors or convulsions. There is often a feeling of tingling or numbness in the hands and feet, and a numb feeling at the place where the drug has touched, e.g. nose and back of throat, when it has been sniffed
- **EYES –** Pupils are dilated.
- **SKIN -**Pallor of the skin, cyanosis, sweating, and the temperature is raised.

(2) **STAGE OF DEPRESSION:**

- Within an hour or even less, respirations become feeble with profuse perspiration. Patient may collapse or may develop convulsions and death may occur due to respiratory failure, cardiac failure, or vascular collapse. Rupture of an aneurysm may occur.

- Large doses or a "binge" may result in anxiety and panic leading to paranoia.
- Its use may cause Foetal death or abortion if used during pregnancy. Its use can also result into premature delivery or child may suffer from developmental, behavioral and learning problems.

SPEEDBALL - A combination of cocaine and heroin taken by injection. It may cause death suddenly and unexpectedly during or immediately after a struggle.

COCAINE HABIT/ COCAINOMANIA/COCAINOPHAGIA

It is an intense, irresistible craving for cocaine. Addict takes its repeated doses to keep them high and to avoid the 'crash'. It is also used with other drugs like alcohol, tranquilizers or heroin to increase its effect. Abusers can tolerate upto 10 g/day.

SIGNS AND SYMPTOMS: „ Emaciation, anorexia, digestive disturbances, significant loss of libido, impotence, gynecomastia, galactorrhea and abnormality in menstrual cycle in women and infertility. Face is pale with sunken eyes, dilated pupils. Tongue and teeth becomes black with ulceration of nasal septum due to sniffing. Degeneration of CNS with hallucinations, convulsions and delirium may occur.

MAGNAN'S SYNDROME/COCAINE BUGS- It is present in cocaine addicts. It is a tactile hallucination characterized by feeling as if grains of sand are lying under the skin or small insects are creeping on the skin giving rise to itching sensation (formication).

FATAL DOSE:

One gm. orally.

Procaine is about half as toxic as cocaine.

FATAL PERIOD: Few minutes to few hours.

TREATMENT:

(1) In case of ingestion, gastric lavage should be done with warm water having potassium permanganate, charcoal or tannic acid.

(2) If sniffled, wash the mucous membrane with water.

(3) If injected, apply a ligature above the part.

(4) Chloroform or short-acting barbiturates to control convulsions.

(5) Amyl nitrite as antidote by inhalation.

(6) Airway should be protacted.

(7) Circulation is to be corrected.

(8) Thiamine 100 mg. i.v.

(9) Symptomatic.

POSTMORTEM APPEARANCES:

- Cocaine decomposes rapidly so blood should be preserved by adding fluoride.
- Brain is analysed as it is not hydrolysed into benzolecognine as in blood.
- Cocaine can be recovered from recent injection sites, or by swabs from the nasal mucosa.
- The signs are of asphyxia.
- Pulmonary congestion and oedema of lungs weighing 3 to 4 times of its normal weight.
- Heart may show foci of scarring.

MLI-

(1) It is rarely used for homicide or suicide.

(2) Accidental cases occur from addiction.

(3) It is used as an aphrodisiac.

4) Prostitutes inject cocaine solution into vagina to produce local constriction.

CHAPTER-24 PSYCHOTROPIC DRUGS

Psychotropic drug affects psychic function, behavior or experience of the user. It includes:

(1) Antidepressants.

(2) Neuroleptics.

(3) Hallucinogens.

CLASSIFICATION:

(1) Sedatives:

(a) Barbiturates and others.

(b) Minor tranquilisers.

(c) Alcohol.

(2) Stimulants:

(a) Amphetamines, methylphenidate.

(b) Cocaine.

(3) Opiates: Heroin, methadone, morphine, etc.

(4) Hallucinogens.

(5) Marihuana

(6) Tranquilisers: chlorpromazine and others.

(7) Antidepressants :

(a) Tricyclics.

(b) Monoamine oxidase inhibitors.

(8) Antimania drugs.

ANTIDEPRESSANTS

CLASSIFICATION:

(I)Tricyclic:

(a) Tertiary amines, amitriptyline, doxepin, impramine, trimipramine.

(b) Secondary amines: desipramine, nortriptyline, protriptyline.

(c) Tetracyclic: maprotiline.

(2) Dibenzoxazepine: amoxapine.

(3) Trizolphyridine: trazodone.

(4) Bicyclic: fluoxetine.

(II) Manoamine oxidase inhibitors (MAOI): nilamide, tranylcypromine.

(Ill) Lithium carbonate.

(IV) Miscellaneous: amphetamines, caffeine.

The antidepressants are toxic due to

(1) Anticholinergic effects producing supraventricular tachycardia, agitation, seizures, coma, hallucinations, and respiratory depression,

(2) Blocks reuptake of norepinephrine at the synapses, resulting in atrial and ventricular disturbances and hypertension,

(3) Quinidine-like membrane depressant effects on the heart by altering sodium influx resulting in conduction delays and myocardial depression,

(4) Peripheral alpha blockade causing hypotension, and

(5) inhibition of sympathetic reflexes centrally

METHAMPHETAMINE

ACTION: It increases the synaptic concentration of neurotransmitters dopamine and norepinephrine and blocks re-uptake of norepinephrine and also increases release of catecholamine. It produces euphoric effect similar to cocaine which lasts ten times more than cocaine. It also stimulates CNS and CVS.

SIGN & SYMPTOMS:

ACUTE POISONING:

(1) Mild: Restlessness, talkativeness, insomnia, tremors, sweating, dilated pupils.

(2) Moderate: Hyperactivity, confusion, hypertension, tachycardia, tachyapnoea, vomiting, sweating, hallucinations.

(3) Severe: Delirium, hyperpyrexia, convulsions, coma, arrhythmias.

CHRONIC POISONING:

(1) It develops psychosis manifested as stereotyped-compulsive behavior, paranoid personality, delusions of persecution and visual hallucinations, sometimes tactile also.

(2) Cardiomyopathy.

(3) Intracranial haemorrhage.

FATAL DOSE: 150 mg. to 2 gm.

TREATMENT:

(1) Gastric lavage.

(2) Acidification of urine.

(3) Symptomatic.

(4) Chlorpromazine for amphetamine psychosis.

POSTMORTEM APPEARANCES

1. Similar to asphyxia.
2. Myocardial fibrosis is seen in long-term use.

MLI:

- Long term use develops psychological dependence and tolerance.
- Withdrawal of drug can cause depression and sometimes suicide.

CYCLIC ANTIDEPRESSANTS

CLASSIFICATION:

(1) First generation: Imipramine, amitriptyline, desipramine, doxepin, nortryptiline, protriptyline, trimipramine.

(2) Second generation; Amoxapine, maprotiline.

(3) Newer agents: Bupropion, trazadone, netazodone, fluoxetine, paroxetine, sertraline.

ACTION: It Inhibits neurotransmitter reuptake. They are anticholinergic blockade, a-adrenergic blockade and myocardial depressant.

SIGNS AND SYMPTOMS:

C.N.S.: Depression of mental state and coma, delirium, altered sensorium, generalised, brief and self-limited convulsions, myoclonus, nystagmus, dysarthria and ataxia.

C.V.S.: Sinus tachycardia, conduction delays, ventricular arrhythmias, depressed inotropy, hypotension, atrioventricular block, bradycardia.

PARASYMPATHETIC: Dry skin and mucosa, ileus, urinary retention, mydriasis and hyperthermia.

FATAL DOSE: 2 to 5 gm.

TREATMENT:

1. Gastric Lavage.
2. Avoid emetics.
3. Activated charcoal
4. Cathartic.
5. Physostigmine

MONOAMINE OXIDASE INIDBITORS (MAOI)

Includes iproniazid, isocarboxazid, phenelzine, pheneprazine, nialamide and tranylcypromine.

ACTION: They block the action of monoamine oxidase, resulting in alterations of neurotransmitter metabolism.

SIGNS AND SYMPTOMS:

PHASE-1: CNS excitation. headache, dilated pupils, tremors, convulsions, hallucinations, confusion, nausea, hyperpyrexia, hypertension followed by hypotension.

PHASE-2: CNS and CVS depression: Coma, cardiovascular collapse.

PHASE-3: Complications: Haemolysis, rhabdomyolysis, pulmonary oedema, acute renal failure.

FATAL DOSE: 2 to 5 mg/kg.

TREATMENT:

1. Gastric Lavage.
2. Activated charcoal.
3. Symptomatic.

PSYCHEDELICS (HALLUCINOGENS)

Psychedelics substances alter perception and mood and affect numerous cognitive processes but the individual knows that what he is experiencing is not real. Person is alert and oriented but experiences different perceptual and sensational abnormalities. Sensory perception and thought processes are grossly distorted, and mood is altered. Reality (space, time, bodily dimensions) is distorted and a feeling of depersonalization makes the patient feel separated from the situation. The person may experience euphoria or dysphoria, can be emotionally labile but usually realises that he is under the influence of

a drug. Some drugs are LSD, cannabis, mescaline (from the pevote cactus), dimethyl tryptamine(DMT), psylocvbin (from mushrooms) etc.

LSD (LYSERGIC ACID DIETHYLAMIDE):

It is –

- Colorless,
- Tasteless,
- Odorless,
- semi-synthetic compound,

Lysergic acid portion of it is a natural product of the ergot fungus Claviceps purpurea.

ACTION:

Powerful antagonist of serotinin, and can also mimic its action.

SIGN & SYMPTOMS:

- The trip is felt usually after half to one hour, peaking after 2 to 6 hours and fading after 12 hours. The bad or good "trip" depends on the circumstances and mood of the patient.
- Dry mouth, sweating, dilated pupils.
- Tachycardia, hypertension.
- Eextreme mood change, visual hallucinations, alterations in time perception, etc.
- Altered changes in vision and hearing, like floating feeling, illusions, sensation of synesthesia or seeing smells and hearing colors.
- Tolerance develops in 2 to 3 days with daily dosing.
- Side-effects are depression, panic attacks, schizophrenic episodes and psychosis.
- User becomes violent and may attack others (urge to kill) or hurt himself.

- The feeling of being able to fly, user can jump out of windows.
- Depersonalisation, chronic dread, depression, mood swings and paranoid attitudes and beliefs may be found.
- **Flash-back symptoms,** the person experiences a recurrence of the emotional and psychological aspects of the previous 'LSD' trip. It occur most frequently with abuse of psychotomimetics, such as LSD, STP, tryptamines, mescaline and psilocybin. These delayed recurring symptoms may lead to eccentric behavior, suicide or even homicide.

FATAL DOSE: About 14 mg.

TREATMENT:

1. Low doses of anti-anxiety drug and benzodiazepines
2. Prolonged talking known as "talking the person down" which may extend up to 12 to 18 hours
3. Psychotherapy

CHAPTER-25 ASPHYXIANTS- CARBON MONOXIDE

CARBON MONOXIDE

Asphyxiant poisons are those poisons which decreases the oxygen concentration or replaces oxygen in breathing air which leads to suffocation and produces adverse effect in body.

CLASSIFICATIOIN:

1. **Irritants Asphyxiants-** Affects respiratory tract producing inflammation. E.g.- smoke, tear gas, ammonia etc.
2. **Chemical Asphyxiants-** They reacts with body tissue affecting supply of oxygen. E.g.- CO, hydrogen sulphide, cyanide etc.
3. **Simple Asphyxiants-** Inert gases when inhaled in high concentration acts mechanically to remove oxygen. E.g.- Nitrogen, helium, methane etc.
4. **Volatile Asphyxiants-** These doesn,t affects respiratory system but when absorbed in blood produces toxic effect. E.g.- Aromatic Hydrocarbons, halogenated hydrocarbons etc.
5. **Systemic Asphyxiants-** Poisons that produces systemic effects. E.g.- Insecticide, arsine etc.

CARBON MONOXIDE

Characteristics

It is

- It produced due to partial combustion of carbon.

- Colourless,
- Tasteless,
- Non-irritant gas.
- Insoluble in water.
- Burns with a blue flame.
- Lighter in compare to air

Sign & Symptoms

50% COHb produces symptoms like alcoholic intoxication. 50%-60% produces syncope or coma with intermittent convulsions. Exposure of atmosphere containing 0.2% of gas will cause death in about 4 hours, 0.4% in one hour, and 10% in 20 to 30 minutes.

COHb%	Sign & Symptoms
0 to 10	No appreciable symptoms
10 to 20	Breathlessness on moderate exertion, mild headache
20 to 30	Throbbing headache, irritability, emotional unstability, judgment power disturbed, altered memory and rapid fatigue
30 to 40	Severe headache, nausea & vomiting, dimness of vision, confusion.
40 to 50	confusion increases, hallucinations, severe ataxia, tachypnoea and collapse on exertion
50 to 60	Syncope or coma with intermittent convulsions, tachypnoea, tachycardia with feeble pulse, pink or red discoloration of the skin.
60 to 70	coma deepens with loss of control on urine and faeces.
70 to 80	Deep coma with depressed or absent reflexes, week pulse.
Above 80	Death due to respiratory arrest.

Treatment

The primary aim is to protect airway.

i. Remove the victim from source of exposure to fresh air.

ii. Maintain patent airway, fresh air and orthobaric oxygen (100% oxygen at atmospheric pressure) by tight-fitting high-flow reservoir face mask or endotracheal tube. The immediate effect of oxygen is enhancement of the dissociation of COHb. It should be continued till COHb reduces to 15-20%.

iii. Hyperbaric Oxygen (HBO) is contraindicated due to adverse effect.

iv. Blood transfusion, if required.

v. Gastric lavage to prevent aspiration pneumonia.

vi. Cerebral oedema is treated by mannitol 500 ml IV as 20% solution over 15 min, followed by 500 ml of 5% dextrose over next 4 h.

vii. Hypotension is initially treated with IV fluids followed by inotropic agents. Standard ACLS protocols are followed to treat dysrhythmias.

viii. Antibiotics and symptomatic treatment.

POSTMORTEM APPEARANCE:

- Death by CO poisoning is called Conflagration.
- A cherry – red discoloration of the skin & mucous membranes.
- Froth may be seen at mouth and nose.

- Bilateral symmetrical necrosis of the lenticular nuclei and punctiform hemorrhages in the white matter of brain.
- Blisters may be seen on dependent parts or areas with bony prominence.
- Congestion of lungs with pulmonary oedema is present.

MLI

- Suicidal poisoning in the West is common by inhaling motor vehicle exhaust.
- Accidental poisoning may happen from cooking gas leakage and incomplete combustion of wood, charcoal or coal in closed spaces.

CHAPTER-26 ASPHYXIANTS- CARBON DIOXIDE

CARBON DI OXIDE

It is:

- Heavy,
- Colorless
- Odorless (slightly irritating) gas.
- Constituent of atmosphere air and concentration level is 0.4%.
- Mildly acidic in taste.

MOA

Pure CO_2 produces vagal inhibition with glottis spasm leading to asphyxia which may result into death. Decreased oxygen supply to tissues may cause death. \

SIGN & SYMPTOMS

Blood CO2 (%)	**Signs and symptoms**
0-2	No symptoms.
2-5	Increased respiration, throbbing headache
5-10	Hyperpnea, tinnitus, mental confusion, muscular tremors.
10-20	Slow respiration, fall in blood pressure

20-40	Dyspnea, muscular weakness, fall in blood pressure, loss of reflexes.
40-60	Dyspnea, feeling of tightness in chest, tinnitus, muscular weakness, drowsiness, unconsciousness, coma and death
60-80	Immediate unconsciousness, convulsions, death due to asphyxia (cerebral hypoxia).

TREATMENT:

i. Shift the patient to fresh atmosphere.
ii. Maintain airway.
iii. Oxygen therapy.
iv. Cardiac stimulants can be used.

POSTMORTEM APPEARANCE:

Features of asphyxia are found.

i. Cyanosis
ii. Dilatation of pupils.
iii. Capillary and venous congestion.
iv. Petechial hemorrhages.
v. Congestion of the viscera.

MLI

- Poisoning is mostly accidental.

CHAPTER- 27 ASPHYXIANTS- HYDROGEN SULPHIDE AND WAR GASES

HYDROGEN SULPHIDE

It is:

- Colorless
- Heavy
- Inflammable
- Smells like rotten eggs.
- Found in large quantity in sewer.

MOA

It acts as chemical asphyxiant and combines with methaemoglobin instead of haemoglobin and affects cellular respiration by inhibiting the action of cytochrome oxidase.

SIGN & SYMPTOMS

- Dullness and sleepiness
- Giddiness, nausea and feeling of oppression
- Headache, vertigo, nystagmus, weakness, coma.
- Arrhythmia, myocardial depression.
- Ocular Lacrimation, photophobia, conjunctivitis.
- Rhinitis, pneumonia, pulmonary oedema.
- Characteristic rotten egg like smell can be obsreved. However, very high concentrations > 150 ppm affects the smelling ability of the victim, so he may have no sense of smell

- Exposure of > 700-800 ppm is extremely lethal and can result in immediate cardiopulmonary arrest

TREATMENT

Primary aim of treatment is to stabilize respiration.

i. Remove the victim into fresh air.

ii. Artificial respiration and 100% O2 is given.

iii. Amyl nitrite and sodium nitrite (without thiosulphate) is used as antidote. It speeds up the process of methemoglobin conversion which gets spontaneously detoxified in the body.

iv. Correction of electrolyte imbalance and pulmonary oedema.

POSTMORTEM APPEARANCE

i. Signs of asphyxia (cyanosis, frothing at the mouth and nose, and petechial hemorrhages in respiratory mucosa) are seen.
ii. Rotten egg like odor is present.
iii. Greenish discoloration of viscera, gray matter of brain and bronchial secretions.
iv. Pulmonary oedema and congestion of viscera.

MLI

- Poisoning is mostly accidental, major cause of deaths in sewer workers.
- H_2S toxicity is also common in workers of petroleum industry in North America.
- Cases of suicide by H_2S are also found in Japan

WAR GASES

War Gases means any form of chemical whether it is solid or liquid or gas which are used to cause vast destruction and casualties during the period of war.

Different types of War Gases are-

(i) **VESICANT**—Mustard gas, sulphur etc.

(ii) **ASPHYXIANT**—Chlorine, phosgene.

(iii) **TEAR GASES**—Chlor acetophenone (CAP), ethyloidoaretate (K.S.K), bromobenzyl cyanide (B.B.C.).

(iv) **NERVE GASES**—They are easters of phosphoric acid. Their effect is similar to organophosphates.

CHAPTER-28 FOOD POISONING

WHO defines food poisoning as "diseases usually either infectious or toxic in nature, caused by agents that enter the body through the ingestion of food".

CAUSES:

(I) POISONING DUE TO BACTERIA AND TOXINS.
(II) POISONS OF VEGETABLE ORIGIN (natural food poisons)
(III) POISONS OF ANIMAL ORIGIN
(IV) CHEMICAL

BACTERIAL FOOD POISONING

It is of the following types:

(1) SALMONELLA FOOD POISONING: It is common food poisoning. Salmonellosis is primarily a disease of animals. Infection transmits into man through contaminated animals and their products e.g. meat, milk and milk products, sausages, custards, egg and egg products. Human carriers can also transmit the infection.

The causative organisms are S. enteritidis of gaertner, S. typhimurium, S. cholerasuis, and less commonly paratyphoid bacilli

On ingestion, organism multiplies and develops acute enteritis and colitis. The onset is generally sudden with chills, fever, nausea, vomiting, and profuse watery diarrhea which usually last 2 to 3 days. Incubation period is 12 to 24 hours. Mortality is about 1 per cent.

(2) SAPHYLOCOCCAL FOOD POISONING: Certain strains of coagulase-positive Staphylococcus aureus are the causative agent. It releases Enterotoxins which is responsible for poisoning. It is found on the skin and in the nose and throat of men and animals. The contamination occurs from contaminated by staphylococci. The toxin is heat-resistant and remains in food even after the organisms dies. The toxin affects

intestine and CNS. The onset is sudden manifested by vomiting, abdominal cramps and diarrhoea. Fever is usually rare.

(3) CLOSTRIDIUM PERFRINGENS FOOD POISONING: This organism is found in faeces of humans and animals, and in soil, water and air. It transmits from ingestion of contaminated meat and poultry. Incubation period is 6 to 24 hours. The spores are heat resistant and multiply between 30° to 50° C and produces different toxins, so food needs to be cooled. Symptoms develops 8-24 hrs after consuming contaminated food. Symptoms are diarrhea, abdominal cramp. Fever may be absent or mild. Symptoms last upto 1 day or less.

(4) CEREUS FOOD POISONING: Bacillus cereus is present in soil and in uncooked, dried and processed foods. The spores are heat resistant and can survive even after cooking food. It produces 2 types of food poisoning.

(i) Emetic form- It has short incubation period. Symptoms are predominantly related to upper GIT.

(ii) Diarrheal form-It has comparatively longer incubation period Symptoms are predominantly related to lower GIT. Treatment is symptomatic.

Food poisoning is usually common in summer as the optimum temperature promotes the growth of infective organism. The organisms responsible for food poisoning are:

(1) The enteric group.

(2) Cholera.

(3) Bacillary dysentery.

(4) Staphylococcal and other bacterial infections.

(5) Amoebic dysentery and other protozoal infections.

(6) Acute infective hepatitis.

(7) Brucellosis.

(8) Various worm infestations.

(9) Schistosomiasis.

(10) Traveller's diarrhoea (E. coli).

Food poisoning should be differentiated from cholera, acute bacillary dysentery and arsenic poisoning by considering history, symptoms, food or vomitus or stool or blood examination for the causative agent.

TREATMENT:

1. Gastric lavage.
2. Glucose-saline infusion

BOTULISM

This food poisoning is caused by Clostridium botulinum. It is found in the soil, dust and the intestinal tract of animals. It produces a potent neurotoxin. It is not an infection but an intoxication.

ACTION: Cl. botulinum grows it's colony in the food and produces a powerful exotoxin. It acts on cholinergic fibres of autonomic nervous system. This results in paralysis of nerve endings, by blocking the nerve impulses at the myoneural junctions. It blocks the action of acetylcholine.

FATAL DOSE- 0.01 mg. or even less in adult.

SIGN & SYMPTOMS-

- Nausea, vomiting, abdominal distension, pain.
- Dry or sore mouth and throat

- Difficulty with visual accommodation, dysphonia, diplopia, ptosis, dysarthria, blurring of vision, muscle weakness.
- Descending bilaterally symmertrical motor paralysis initiated by abducent (VI) or oculomotor (III) nerve palsy.
- Dysphagia, constipation, respiratory insufficiency, and urinary retention.
- Coma, delirium.
- Consciousness persists until death.
- Subnormal temperature.
- Death may occur due to respiratory or cardiac failure.

TREATMENT:

1. Gastric lavage.
2. Emetics.
3. Activated charcoal.
4. Purgative (sorbitol).
5. Bowel wash.
6. Botulinum antitoxin.
7. Botulism immune globulin (BIG).
8. Guanidine.
9. Adequate respiration.
10. Alcohol precipitates toxin.

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