POISON and its TREATMENT

POISON...

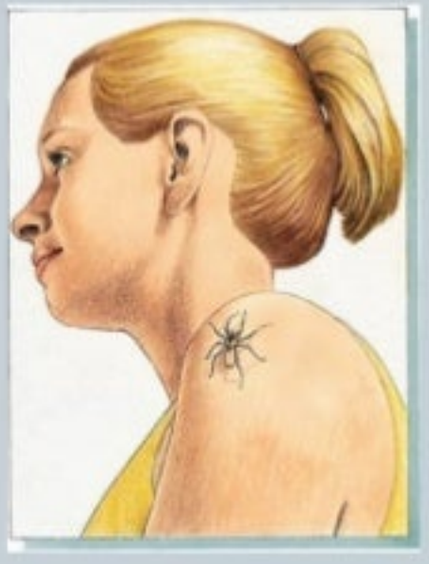
- Poison is defined as a substance which when administered, inhaled or swallowed is capable of acting deleteriously on the body i.e. produce ill health or death.

- All substances are poisons, there is none which is not a poison" ……. Paracelsus

- Poison may be of synthetic, mineral, vegetable or animal origin.

- WHO estimates over 30 lakh poisoning cases with 2,20,000 death per year worldwide.

- **How poisons enter the body:**
  - **Inhalation**
  - **Ingestion**
  - **Absorption**
  - **Injection**



Classification of Poison

- Poison can be classified based on type of action exerted or based on Medicolegal purpose.

- Classification based on action can be classified in 6 categories:

1. Corrosive
2. Irritants
3. Neurotoxic
4. Cardiac poison
5. Respiratory poison
6. Miscellaneous.

- Classification based on **medicolegeal** purpose:

1. Suicidal

2. Homicidal
3. Accidental
4. Stupefying (dose related adverse effects)
5. Abortificient ((chiefly of a drug) causing abortion.
6. Cattle poison (Young calves can get lead **poisoning** if they lick lead paint or batteries.)
7. Arrow poison (**poison arrow** heads)
8. Food poisoning
9. Drug dependence.

Classification based upon action

1. **Corrosive:**

a) **Acids**: inorganic, organic and vegetables

- ***Inorganic acid***: H2SO4, HCl, Nitric acid
- ***Organic acids***: Acetic acid, Salicylic acid, Oxallic acid , Carbolic acid.
- ***Vegetable***: Hydrocyanic acid, Potassium cyanide.

B) **Alkali**: Hydroxides and carbonates:

a) ***Hydroxides***: of sodium , potassium and ammonium

b) ***Carbonates*** of sodium , potassium and ammonium.

2. Irritants:

a) **Inorganic:** ***Non metals*** like Phosphorus, Boron, Fluorine, Chlorine, Bromine etc.

AND ***metals*** like Arsenic, Lead, Mercury, Iron, Zinc etc.

b) **Organic** : Vegetables and animals:

- ***Vegetables***: castor oil, croton oil and seed, calotropis, ergot, capsicum.
- ***Animals***: Snakes, Scorpion, Spiders and poisonous insects.

c) **Mechanical:** Powdered glass, Diamond rust, Hair pin, Needles, Nails etc.

3. Neurotoxins:

A: Cerebral neurotoxins:

1. ***Narcotic or somniferous***: Opium and its alkaloids like morphine, codeine, thebaine, Noscapine and Narcine etc.

2. ***Inebriants***: which capable of intoxicating, e.g. Alcohol, anesthetic agents (*ether, chloroform etc*), Fuels (*Kerosene, petrol*), Sedative and *hypnotics(Barbiturates, Chloral hydrate*), Insecticides(*DDT and endrine, organophosphorus compounds like parathion and malathione*), and coal derivatives *(Naphthalene*).

3. ***Deliriants:*** e.g. Dhatura, Cannabis, Atropa belladonna, Cocaine, Camphor etc.

B) Spinal Neurotoxins:

a) Excitants: Nux vomica and its alkaloid strychnine.

b) Depressant: Lathyrus sativus and jasmine.

C) Peripheral neurotoxins:

e.g. Conium and curare

4. Cardiac poison:

- Aconite
- Digitalis
- Oleander
- Tobacco

5. Respiratory Poison: also known as ASPHYIANT.

- Carbon monoxide
- Carbon dioxide
- Sulphur dioxide,
- War gas (tear gas, Chlorine) , Phosphine etc.

6. **Miscellaneous:**

- Analgesics & Antipyretics

- Antihistaminics
- Tranquilizers
- Stimulants e.g. Amphetamines
- Antidepressants
- Hallucinogens: LSD
- Food poisoning
- Drug dependance.

Classification based on Medicolegal purpose

1. **Suicidal:** the near ideal suicidal poison are opium and barbiturates.

   Commonly used suicidal poison in India are: Arsenic, Aconite, Cyanide, Phenols, CO etc.

2. **Homicidal: Homicide** is the act of a human being killing another human being.

   The near ideal homicidal are Thallium and Fluoride (in rodenticide).

   Commonly used homicidal are: Snake venom, Corrosive, Opium, Strychnine, Cyanide etc.

3. Accidental poisons: Accidental poisoning occurs due to:

- Keeping insecticide at home and field.
- At place of work or industry.
- Carelessness ion storing poisons.
- Quack remedies: fraud remedies
- Snake, scorpion and insect bite.

4. **Stupefying poisons:**

- Stupefying means to render stupid.

- These are the poisons which alter the consciousness of person and are commonly give for purpose of rape, robbery, dacoit, theft, etc.

- Dhatura, Cocaine, Alcohol, Arsenic, Cigarette, Cannabis preparation like charas, ganja and bhang.

5. **Abortificient**: these are the poison used to induce criminal abortion.

examples are: Lead, Arsenic, Ergot, Castor oil, Croton oil Calotropis etc.

6. **Cattle poison**: these are the poison used to kill cattle.

examples are: Abrus precatorious, Aconite, Arsenic, Snake venom, Insecticides etc.

7. **Arrow Poison**: The poison which commonly applied on arrow head. Examples are: Curare, Abrus, Calotropis, Snake venom, Aconite etc.

8. **Food Poisoning**:

9. **Drug dependence.**

General treatment of Poisoning

- The general treatment is given if the specific nature of poison is not known.

- The condition of patient is assessed by detail clinical examination based on degree of loss of consciousness and presence or absence of other medical complications.

- The patient should be remove from the source of exposure.

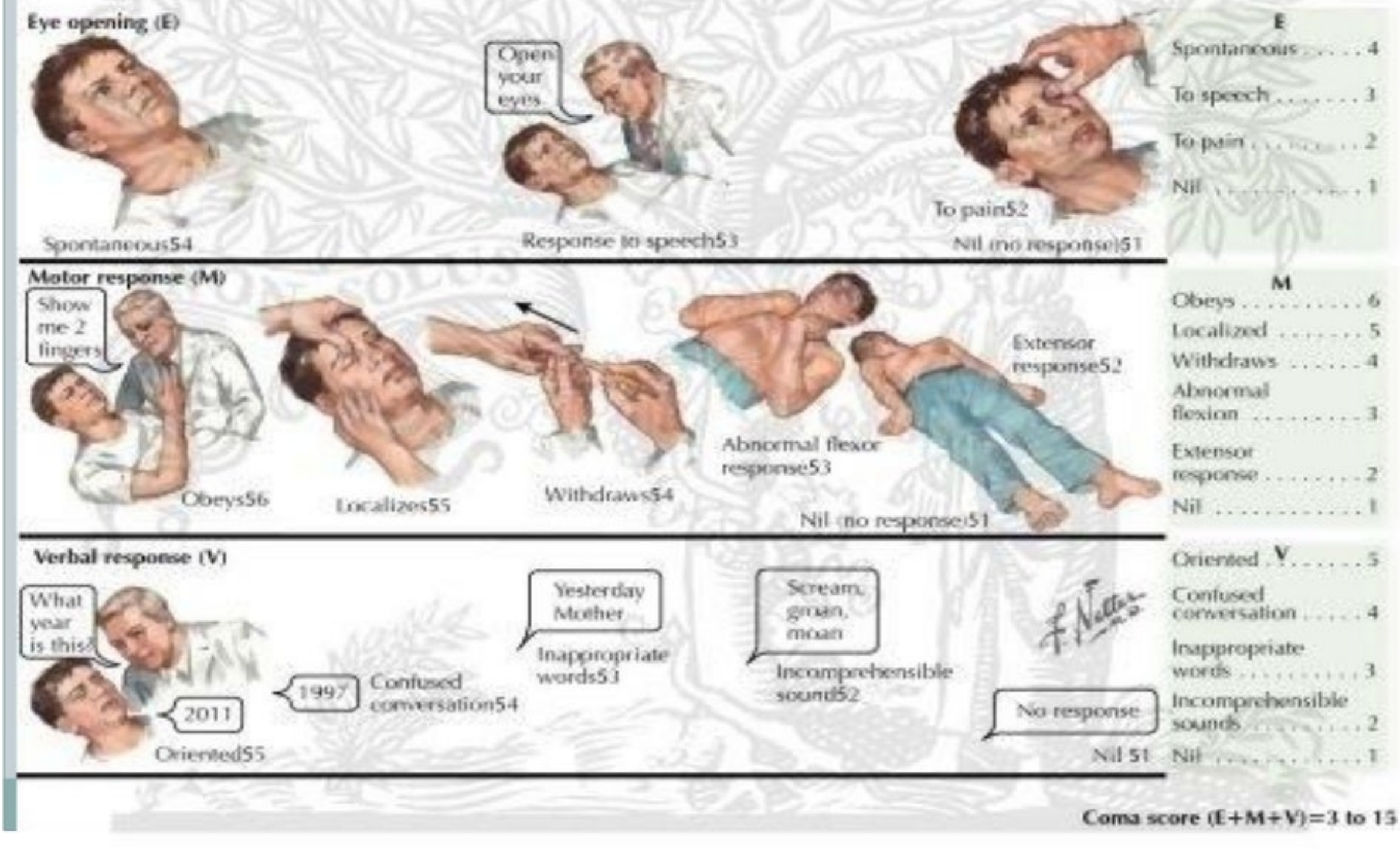
- The degree of loss of consciousness is graded by Reed's classification

- The degree of coma is assessed by Glasgow COMA scale.

Reed's classification of grade of consciousness

- **Grade I**: Drowsy but responding to verbal command
- **Grade II**: Unconscious but responding to stimuli
- **Grade III:** Unconscious but responded only to very painful stimuli
- **Grade IV:** Unconscious and non responding.

Glasgow Coma Scale		
Response	Scale	Score
Eye Opening Response	Eyes open spontaneously	4 Points
	Eyes open to verbal command, speech, or shout	3 Points
	Eyes open to pain (not applied to face)	2 Points
	No eye opening	1 Point
Verbal Response	Oriented	5 Points
	Confused conversation, but able to answer questions	4 Points
	Inappropriate responses, words discernible	3 Points
	Incomprehensible sounds or speech	2 Points
	No verbal response	1 Point
Motor Response	Obeys commands for movement	6 Points
	Purposeful movement to painful stimulus	5 Points
	Withdraws from pain	4 Points
	Abnormal (spastic) flexion, decorticate posture	3 Points
	Extensor (rigid) response, decerebrate posture	2 Points
	No motor response	1 Point
Minor Brain Injury = 13-15 points; **Moderate Brain Injury** = 9-12 points; **Severe Brain Injury** = 3-8 points		


Eye opening (E)
Spontaneous54
Open your eyes
Response to speech53
To pain52
Nil (no response)51
E
Spontaneous 4
To speech 3
To pain 2
Nil 1
Motor response (M)
Show me 2 fingers
Obeys56
Localizes55
Withdraws54
Abnormal flexor response53
Extensor response52
Nil (no response)51
M
Obeys 6
Localized 5
Withdraws 4
Abnormal flexion 3
Extensor response 2
Nil 1
Verbal response (V)
What year is this?
2011
1997
Oriented55
Confused conversation54
Yesterday Mother
Inappropriate words53
Scream, groan, moan
Incomprehensible sound52
No response
Nil 51
V
Oriented 5
Confused conversation 4
Inappropriate words 3
Incomprehensible sounds 2
Nil 1
Coma score (E+M+V)=3 to 15

- **AIM of general treatment of poisoning:**

- TO remove the unabsorbed poison
- TO treats and excrete the absorbed poison.

- **Steps of general treatment :**

1. *Removal of unabsorbed poison*
2. *Use of antidotes*
3. *Elimination of absorbed poison*
4. *Symptomatic treatment*
5. *General care of patients.*

1. Removal of unabsorbed poison

- This depends on route of administration of poison.
- Induction of vomiting or gastric lavage is generally adopted method to remove poison.

a) **Inhaled poison:**

- Remove patient to open atmosphere.
- Ensure clear airway
- Artificial respiration and oxygenation

b) Injected poison or snake bite:

- Use of tourniquets
- Incision and suction
- Ice pack may be applied

c) Contact poisoning:

- Wash the area with water or soap water.
- Apply antiseptic creams in case of stings.

d) Ingestion of poisons:

- Induction of vomiting
- Gastric lavage.

Induction of vomiting

- Vomiting induction is used to remove poison within 4-6 hours of ingestion.

- ***This method is contraindicated in case of :***

- Ingestion of corrosive poison (risk of perforation in GI tract).
- COMA (chances of aspiration)
- Ingestion of petroleum distillate (fumes may be inhaled).
- In cardio-pulmonary disease (chances of heart failure).
- In case of pregnancy and children

The various methods are employed for **induction of vomiting** as follows:

1. Mechanical irritation: irritation produces in throat with finger or any linear object in it.

2. B y using EMETIC agents:

- Plane warm water
- Weak solution of copper sulphate
- Mustard powder 15-20 gram in glass full of water.
- Common salt 15-20 grams in glassful of water with mustard oil.
- Ammonium carbonate 1-2 grams in one glass of water.
- Ipeca-cuhana (contains emetine) 1-2 grams with one glass of water or 30 ml syrup.

Gastric lavage

- This method is employed in case of indigestion of poison within 4-6 hours.
- Even in case of indigestion of poison above 6 hours this method is useful.

- This method is **contraindicated** in following cases:
  - Absolute contraindicated in corrosive poison (except carbolic acid).
  - In case of convulsions.
  - In children.
  - In case of COMA
  - In case of petroleum distillate.

- The gastric lavage is done by using specific tube , referred as BOA'S tube or EWALD'S tube.

- **Gastric lavage tube:**

- It is 1-1.5 meter long, 12.7 cm broad rubber tube thick enough to pass through esophagus. The tube consist of following parts.

- Funnel end: to pour in fluid and to take fluid out after lavage.

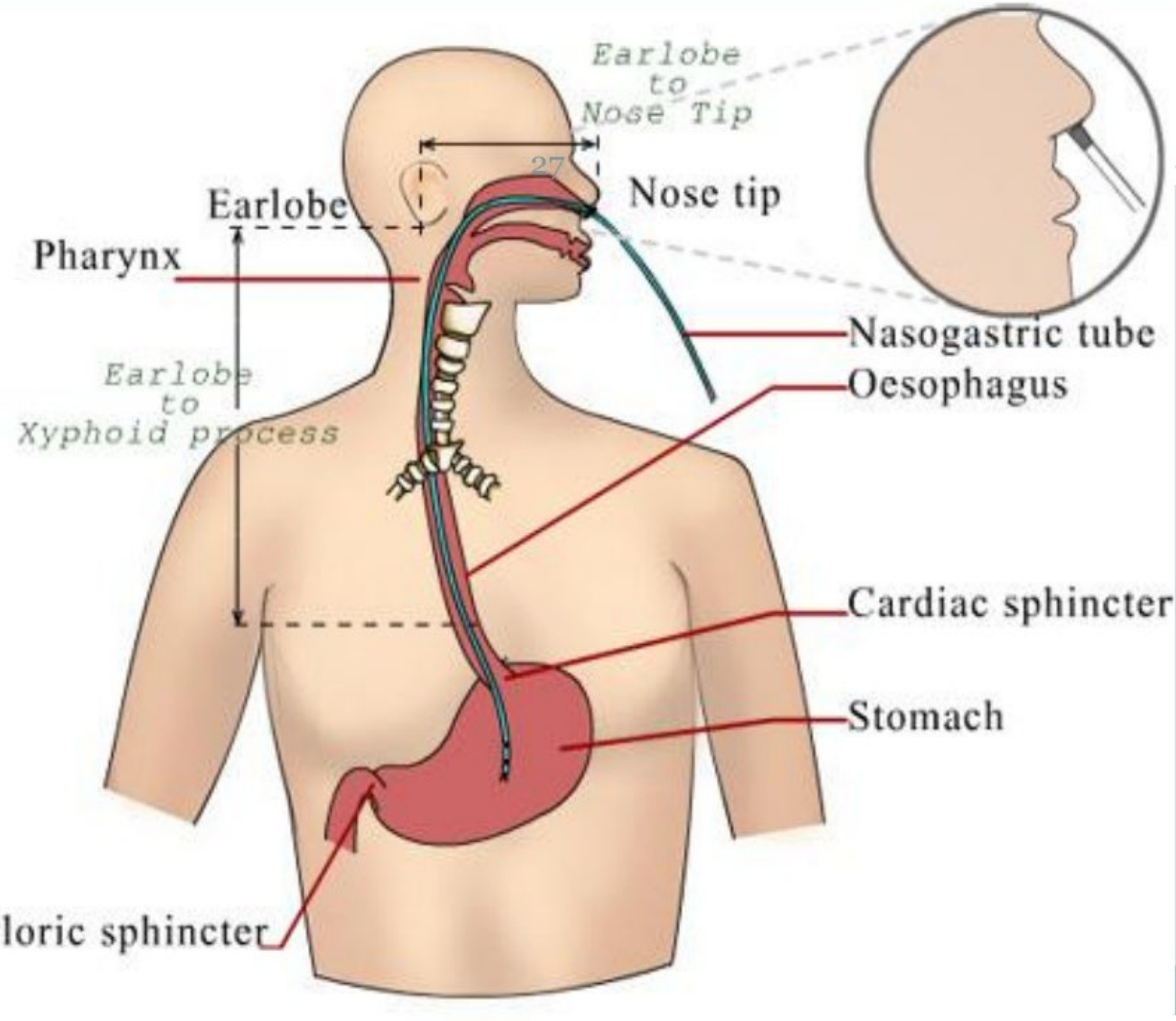
- Suction bulb: to take out fluid if syphon action failed or to force open perforation block by food particles.

- Lower rounded perforated end:

- Rubber tubing

- Mouth gag: its wooden structure (to avoid teeth damage) and have a central large hole to pass rubber tube.

- Ideal tube for gastric lavage is levacuator: clear plastic tube.


Earlobe to Nose Tip
Earlobe
Nose tip
Pharynx
Nasogastric tube
Oesophagus
Earlobe to Xyphoid process
Cardiac sphincter
Stomach
Pyloric sphincter

Precautions in gastric lavage

- The patient should be semi prone or prone to left side.
- Head should be lower than hip (for better drainage)
- Ensure clear airway
- Mouth gag should always use
- The first washing should be with plain water 100-200ml.
- Continue stomach wash till the color of ingoing fluid and outcoming fluid is same.
- Pinch the tube before removing (to avid aspiration)

Procedure of gastric lavage

- After positioning the patient, teeth are open and mouth gag is placed.
- The lower end of tube is lubricated with water, soap or milk and is passed through opening of mouth gag till the 50 cm mark , ensuring that tube is pass through stomach.
- If cough reflex , hissing sound heard at funnel or air bubble seen in dipping funnel end in water, it indicate that tube is enter into respiratory tract.
- The first washing is done with fresh water, small amount 100-200 ml used to avoid swapping of poison into intestine.
- Further lavage is done with plain warm water or $KMnO_4$ (1:1000).
- Each time 500ml of fluid is passed into funnel held at level higher than body.

- After 15-30 seconds funnel end is brought lower than body level when fluid from stomach came out by syphon action.
- The process is continued till the colour and nature of ingoing and outcoming fluid is same.
- About 20 liters of fluid is required for whole process (almost 20 washing).

- **Other fluids used in gastric lavage procedure are:**

- 5% sodium bicarbonate in acidic poisoning.
- 4% tannic acid in strychnine poisoning
- 25 % sodium thiosulphate in cyanide, iodine and arsenic poisoning.
- 1 % calcium gluconate in oxalate poisoning.
- 2 gm/L Desferrioxamine in iron poisoning.
- 1:2 castor oil in warm water in carbolic acid poisoning.

- The poison which stick to wall of stomach, in such case one of following can be left in stomach.

- $KMnO_4$ solution, Demulcent, Sodium and manganese sulphate as purgatives, sodium bicarbonate (in acidity) or Activated charcoal (as adsorbent).

- In children: gastric lavage is done with Ryle's tube or rubber catheter using 20ml or 50 ml syringe to push in or take out fluid from stomach. Usual length of tube is 30 cm .

- **Gastric lavage is contraindicated in :**
- Aspiration pneumonia,
- Vomiting
- Injury to mucosa of GIT, rarely perforation.

2. Use of antidotes

- ***Antidote is defines*** as the substance which counteract the deleterious effect of the poison without itself being harmful to the body.

- **Antidotes are used in case of :**

- Where emetics and gastric lavage is contraindicated.
- Poisoning by route other than oral.
- The poison that escapes gastric lavage or vomiting.
- In case of already absorbed poison.

- **Types of ANTIDOTES are:**

A) Physical antidotes, (B) Chemical Antidotes, (C) Pharmacological & Physiological antidote, (D) Chelating agent, (E) Universal antidote, (F) Household antidote.

1. Physical or Mechanical antidote

- These are defined as antidote which physically hinder the absorption of poison.

a) ***Demulcent:*** these substances form an oily layer on the gastric mucus membrane so that absorption in the stomach does not occurs.

- Examples are: *Oil, Butter, Ghee, Milk of magnesia, White of egg, Starch solution and Aluminium hydroxide gel.*
- Demulcents are contraindicated in: Phenol, Phosphorus, Organophosphorus& Kerosene Poisoning as these are soluble in oils.

b) ***Bulky Foods***: These substances engulf the poison and make it non available for causing effect and its absorption.

- *Examples are: Banana, Flour suspension, Boiled rice and Mashed potato.*

c) ***Activated Charcoal:*** it adsorb the poison and there by make it non available for absorption.

- It is given 1-2 gm /Kg in 8 ml diluents /gm .

2. Chemical Antidotes

- These substance chemically react with poison and thereby alter its chemical nature.
- **Certain substances which react with poison and make it inert are as:**

- Weak alkalies (Calcium oxide and magnesium oxide) for acid poisoning.
- Weak acids(Acetic acid, Vinegar, Lime juice) for alkali poisoning.
- Calcium carbonate for oxalic acid poisoning.
- Sodium & magnesium sulphate for lead poisoning.
- Copper sulphate for Phosphorus poisoning.
- Sodium thiosulphate for iodine and cyanide poisoning.
- Potassium ferrocyanide for copper poisoning.
- Hydrated ferric oxide for Arsenic poisoning.
- Sodium formaldehyde sulphoxylate in Mercury poisoning.
- Tannins: forms a complex with Metal, Alkaloid and Glucosides.
- $KMnO_4$ (1 in 1000) and weak solution of iodine (10-15 drops per 100 ml) oxidizes the poison

3. Pharmacological or Physiological antidotes

- These are the substances which have pharmacologically opposite action as compared to the poison. (its effect on physiology is exact opposite as that of poison).
- Certain substance which have opposite pharmacological action are:

- *Atropine* for Pilocarpine poisoning.
- *Atropine* for organophosphorus poisoning.
- *Naloxone* for opium and morphine poisoning
- *Chloroform* for strychnine poisoning.
- *Physostigmine* for Dhatura poisoning.
- *Ethyl alcohol* for Methanol poisoning
- *N-acetyl cystine* for Paracetamol poisoning.

- Immunotherapy: *Digoxine antibodies & snake antivenin.*

4. Chelating agents

- These are the substances which are used in metal poisoning. The form a non-ionized complex with metal ion so that it not available for absorption and there by cannot produce deleterious effect on body.
- Examples of chelating agents are:

- BAL: British Anti Lewisite
- EDTA
- Cuprimine
- DFM : Desferrioxamine

1. BAL: British Anti Lewisite:

- Chemically it is Dimercaprol or 2:3 Dimercaptopropanol.

- It is effective in Arsenic poisoning.
- Two SH group of BAL bind with metal .
- BAL is liquid and is given with 10 % solution in arachius oil or peanut oil with Benzyl Benzoate (3mg/Kg body weight).
- It is given in deep intramuscularly in gluteal region.
- It given 4 hourly in first 2 days, 6 hourly on 3rd day and 12 hourly till the 10th day.
- It cannot be given IV for danger of causing embolism due to arachius oil.
- ***DMSA succimers*** (2:3 dimercapto succininc acid) & ***DMPS Unithol*** (Dimercapto Propane Sulphonic Acid) are the derivative of Dimercaprol can be used Orally 30 mg/ Kg (NOT AVIALABLE YET)
- BAL uses contraindicated in Liver damage, Hypertension and Tachycardia.

2. EDTA:

- It is effective against Lead, Mercury and Copper poisoning.

- Specifically used against lead poisoning.

- As EDTA deplete serum calcium, CaNa2 is better option.

- It is given either orally 1 gm BD for 5 days.

- If given IV, 5 ml of 50% solution BD for 5 days.

- EDTA use is contraindicated in Renal damage cases.

3. Cuprimine: (Penicillamine, Dimethyl Cystein)

- It is degradation product of penicillin.

- It is effective in copper, lead and mercury poisoning.

- Specifically used in copper poisoning.

- Also usefull in Wilson's disease (Hepato-lenticular degeneration) and cysteinuria.

- It is given orally 30 mg/Kg in divided doses for 5-10 days.

- Its prolong use may causes thrombocytopenia, agranulocytosis, skin rashes and nephrotic symptoms.

4 Desferrioxamine (DFM):

- It is effective in iron poisoning.

- It is given as:

a) Orally 8-12 gm per day

b) IM or IV 2-3 gm per fay

- DFM is also usefull in Haemochromatosis and thalassemia.

Universal Antidote

- This antidote is called ***UNIVERSAL*** because it can be used in all cases of poisoning, especially when nature of poison is unknown.

- Universal antidote given 15-30 gm orally. It can be repeated 12-24 hourly.

- **The composition of universal antidote is:**

- ***Charcoal (50%, 2 parts):*** obtained from burnt bread or toast results in adsorption of poison.
- ***Mg oxide (25%, 1 part):*** Obtained from milk of magnesia, which results in neutralization of poison.
- ***Tannins (25%, 1 part):*** obtained from strong tea, which result in precipitate metal, alkaloid, glucosides and neutralization of alkali

Household Antidote

- These are substances which are available usually in the house and can be used in case of poisoning.

- Milk orally is effective in almost all ingested poison.

- Charcoal from burnt bread and toasts.

- Banana, Potato and Demulcent are physical antidotes.

- Common salt and mustard powder as emetic agent.

- Starch solution for iodine poisoning.

- Milk of magnesia for acid poisoning.

- Vinegar, Lemon Juice, Orange juice for alkali poisoning.

3. Elimination of Adsorbed poison

- **It is done by:**

- Purgation (catharasis): using 30 gm sodium or magnesium sulphate or 50 ml sorbitol.

- Forced diuresis using frusemide or mannitol infusion.

- Increased sweating(diaphoresis): hot pack applied or inj. Pilocarpine-5 mg S.C.

- Stimulating metabolism in liver (using vit B complex).

- Peritoneal and Hemo dialysis

- Whole bowel irrigation (Administration of nonabsorbable Polyethylene glycol to cause liquid stool to thus wash gut rapidly.

4.Symptomatic treatment

- Morphine , Pethidine or Fortwin for pain.
- I.V. fluids for Shock
- Blood for oligamic shock
- Glucose for hypoglycemia
- Artificial oxygenation
- Cardiac stimulants.
- Noradrenalin drip for peripheral circulatory failure.
- Atropine for abdominal pain
- Luminol or Diazepam for convulsion or restlessness.
- Sodium or Potassium for electrolyte imbalance.
- Anesthesia for convulsions.
- Adrenaline , Antihistaminic for anaphylactic reaction.

5. General care of Patient

- Warmth
- Good nursing care
- Prophylactic antibiotic
- Physiotherapy for rehabilitation
- Psychotherapy in attempted suicide.

BARBITURATE POISONING

- Acute barbiturate poisoning results from ingestion of an overdose either by accidental or by suicidal attempt.

- It is symptomatically characterised by: Depression of CNS, particularly respiratory and peripheral circulatory collapse.

- Patient show weak and rapid pulse. Cold and clumsy skin, slow, rapid and shallow breathing.

- Pupils may be constricted initially and respond to light, but later on develop paralytic dilation.

- Fatal complications are atelectasis, bronchoconstrictions, and acute renal shut down.

Management of Barbiturate poisoning

- In general, sedative-hypnotic drugs are nonselective in their effects. At lower doses, a reduction in restlessness and emotional tension occurs. At increasingly higher doses, sedation is followed by increasing levels of anesthesia and eventually death

- The severity of barbiturate poisoning is assessed by clinical examination prior to treatment and correlate with plasma level of barbiturate.
- Presence of reflexes, response to Painful stimuli, Maintenance of blood pressure & respiration without external assistance indicate fair prognosis.

- Plasma barbiturate concentration of : for Short acting barbiturates (35 mg/L) and for Long acting barbiturates (90 mg/L) indicate unfavorable prognosis.

Treatment includes……

1. **Gastric lavage**:

- Gastric lavage may be performed if the patient presents obtunded within 1 hour of ingestion or rapidly deteriorates while in the emergency department.
- Vomiting can be induced by syrup of ipecac or concentrated salt solution.
- For prevention of absorption of poison, Activated charcoal (20 gm) with egg albumin can be given to patient through Ryle's tube and repeated 4 hourly.

2. **Endotracheal intubation:**

- It is performed when spontaneous respiration is inadequate and also to remove secretion is patient who show depressed cough and pharyngeal reflex.
- This reduces lung complication by providing adequate ventilation.

3. *Forced diuresis*:

- This is performed to increase urinary excretion of barbiturates.
- Forced diuresis is potentially dangerous procedure and should be consider to a patient who have take phenobarbitone in such dose that patient not survive only by supportive therapy.
- Diuretics like Mannitol and Furosemide have been employed for forced diuresis.
- Mannitol, an osmotic diuretics, given I.V. initially in a dose of 100-120 ml of 25% solution.
- Subsequently a sustained infusion of 5% Mannitol alternatively with saline or 5% dextrose is administered at the rate of 500ml per hour for next three hours.
- An average urine volume of 10-12 liters in 24 hours is considered as satisfactory diuresis.
- Furosemide is more powerful diuretics is used in a dose of 20mg along wit 500 ml of 1.2% sodium bicarbonate and one liter of 5% dextrose .

4. Alkalinisation:

- Mild systemic alkalosis reduces plasma concentration of non ionized and diffusible form of barbiturate.
- This lead to withdrawal of barbiturate from brain and CSF.
- In addition alkalinisation prevents reabsorption of barbiturate and enhances its elimination.
- This process significantly increases excretion of long acting barbiturate and not for short acting barbiturate.
- Sodium bicarbonate 3.5 gm per 50 ml may be added to every liters of fluid intended for I.V.
- The urinary pH should be checked hourly and maintained in between 7.5 -8.5 .
- Another substances employed are THAM (tris hydroxymethyl amonomethane) is administered I.V. as 1/3rd molar solution in .2% sodium chloride.

5. Dialysis:

- Peritoneal dialysis or hemodialysis is used to remove barbiturate from body.

- Both are more effecting in removing long acting barbiturate

- Peritoneal dialysis is not more effective that forced diuresis.

- But hemodialysis is forty time more effective than forced diuresis in promoting elimination of barbiturate.

- **Hemodialysis is spacially indicated in following cases:**

1. Shock and progressive lethal dose level.
2. Ingestion of lethal dose.
3. In patient with whom peritoneal dialysis is not effective or contraindicated.

ALCOHOL POISONING

- Alcohol is Neurotoxic cerebral inebriant poison.
- Alcohol is a generic term for **ethanol**, which is a particular type of alcohol produced by the fermentation of many foodstuffs - most commonly barley, hops, and grapes.
- Other types of alcohol commonly available such as:

- **Methanol** (common in glass cleaners)
- **Isopropyl alcohol** (rubbing alcohol).
- **Ethylene glycol** (automobile antifreeze solution) are highly poisonous when swallowed, even in small quantities.

- Ethanol is generally consume as alcoholic beverages like WINE, PORT, SHERRY, VODKA etc.
- Absolute alcohol is 99% Ethanol, Rectified spirit is 95% alcohol and Denatured alcohol is 95% ethanol and 5% methanol.

- Ethyl alcohol: MOA-

- Ethanol exerts its actions through several mechanisms. For instance, it binds directly to the gamma-aminobutyric acid (GABA) receptor in the CNS and causes sedative effects similar to those of benzodiazepines.

- ethanol is also an *N* -methyl-D-aspartate (NMDA) glutamate antagonist in the CNS.

- Ethanol also has direct effects on cardiac muscle, thyroid tissue, and hepatic tissue.

- Ethanol is rapidly absorbed, and peak serum concentrations typically occur 30-60 minutes after ingestion.

- Its absorption into the body starts in the oral mucosa and continues in the stomach and intestine

Acute poisoning of Alcohol….

- There are four stages which characterised by specific symptoms depends upon alcohol concentration:

1. ***Stage of Excitement:***

- In this blood alcohol level is 50-100 mg/dL (0.05-0.1%)
- Characterised by- Hesitation, Self criticism, and restraint loss.
- Feeling of well being and pleasure.
- Person may became Argumentative, sentimental, sad or depressed.
- Face is flushed and pupils are dilated.
- Smell of alcohol in breath.

2. Stage of In coordination:

- In this blood alcohol level is 100-30mg/dL (0.1-0.3%)
- In-coordination in thought, speech and action.
- Confusion, impaired memory, slurred speech,
- Staggering gait, impaired skilled movement, blurred vision.

3. Stage of narcosis:

- In this blood alcohol level is 30-50mg/dL (0.3-0.5%)
- Patient is in deep sleep, respond only to deep stimuli.
- Excessive salivation, pulse is increased, temperature decreases and pupil contracted.
- Mac Ewan's sign: pupil dilated on pinching of muscle of face and neck and again contracted on release of pinch.

4. State of medullary paralysis:

- In this blood alcohol level is more than 50 mg/dL (+ 0.5%)
- Respiration is slow and stertous,
- Skin is cold and clumsy and cyanotic
- Pupil are dilated and abolition of reflexes and pulse became imperceptible.

- Treatment of Alcohol poisoning:

- Gastric lavage using sodium bicarbonate
- Safeguard respiration, I.V. glucose, thiamine and fluids
- Avoid emetics
- Ethyl alcohol: 1mg/kg in methanol poisoning as 10% solution given I.V.
- 4-methyl pyrazole is better antidote for methanol poisoning.
- Symptomatic treatment.
- Withdrawal symptom may be treated with carbamazepine.
- Treatment of ethanol and isopropanol intoxication is largely supportive.
- Because of the hemorrhagic gastritis that can follow isopropanol ingestion, H2 blockade or proton-pump inhibitors may be helpful.
- The primary antidotal treatment of methanol or ethylene glycol involves blocking alcohol dehydrogenase. This enzyme can be inhibited by either ethanol or fomepizole

- Fomepizole should be administered as a loading dose of 15 mg/kg. Subsequent doses should be at 10 mg/kg every 12 hours for 4 doses.

- In addition to blocking alcohol dehydrogenase, significant metabolic acidosis should be treated with sodium bicarbonate infusions. If methanol is suspected.

- Folinic acid should be administered at a dose of 1 mg/kg, with a maximal dose of 50 mg. it should be repeated every 4 hours.
- If folinic acid is not immediately available, folic acid can be substituted at the same dose.

- if ethylene glycol overdose is suspected, the patient should also receive 100 mg of intravenous thiamine every 6 hours and 50 mg of pyridoxine every 6 hours.

Chronic Alcohol Toxicity

- Alcohol leads to physical, social , moral and mental deteoration.
- Serum amino glutamyl transferase level increases.
- **Physical Symptoms:**

-Gastritis, Pancreatitis, Fatty liver, Cirrhosis of liver,

- Malabsorption, altered gonadal function
- Loss of memory
- Baby born to mother who drink heavily in pregnancy are usually mentally retarded.

- **Mental symptoms as:**

- **Delirium tremens:** Psychotic conditions in alcoholics due to long continuous action on brain.
- Develops in habitual drinkers,
- Characterised by acute insanity, insomnia, Excitements,
- Disorientation of time and place, tremors of face , tongue and hands.
- Suicidal and homicidal tendencies.

- Korsakoff's psychosis:

- Syndrome characterisd by disorientation, multiple neuritis, loss of memory and hallucinations
- Acute Halucinosis.

Methanol Poisoning….

- Accidental ingestion of methanol or adulterated alcoholic beverages containing methanol can produce dangerous effect on health.
- Abdominal cramps
- Marked muscular weakness and dizziness
- Depressed cardiac and respiratory action.
- More marked effect on CNS than ethanol
- Blurring of vision or complete blindness
- Unconscious and COMA
- Convulsion and death.

- Treatment include gastric lavage with 5% NaHCo3
- Antidote: Ethyl alcohol 1ml/kg/day as 10% solution I.V.for 5 days.

Morphine Poisoning…

- Morphine, is a phenanthrene alkaloid obtained from the dried juice of unripe capsule of poppy fruit of Papaver somniferus. Is is present near about 10% in dried juice.
- Morphine is a drug classified as a narcotic_analgesic that is commonly used to treat moderate to severe pain.
- It acts by depressing the central nervous system ,particularly depresses cortex, respiratory and cough centers in medulla. But stimulates vagus and vomiting centers.
- Its analgesic effect exerted by binding with Mu (Mu1, Mu2, & Mu3), Kappa (k1,k2,k3), Delta and Sigma receptors.
- Morphine is a very potent drug, and when one has developed tolerance due to frequent use, there is a possibility of dependence and addiction.
- The symptoms of morphine toxicity have several stages.

Symptoms of morphine poisoning…..

- ***Stage I: Stage of excitement and euphoria:***

- Increased sense of well being, increased mental activity.
- Flushing of face, sometimes hallucination.
- in children, marked convulsion occurs.
- This is short lasting stage and may not be there if large dose is taken.

- ***Stage II: Stage of sopor (Stupor or depression):-***

- Headache, nausea, vomiting, lethargy, drowsiness.
- Contracted pupil, cyanosed face and itching all over body.

- ***Stage III: Stage of narcosis (coma):***

- Deep coma, muscle relaxed, hypotension, hypothermia, cyanosis .
- Pin –point pupil not responding to eye, Cheyen stroke breathing.

Diagnosis of opium poisoning…

- Coma
- Typical opium smell (raw flesh)
- Cyanosis, Pinpoint pupil
- Froth at nose and mouth, Cheyen stroke breathing
- Moist cold skin, slow pulse, and hypothermia.
- The triad of pinpoint pupil, coma and depressed respiration (4-5 per minute) strongly suggest opioid poisoning.

- **MARQUIS TEST:** 1 drop of mixture of H2SO4 (3ml) and 3 drop of Formalin, dropped on blotting paper soaked in material: purple, then violet and finally blue colour appear.

- **Fatal dose:** Opium 2 gm, ***Morphine 200mg***, Codeine 0.5 gm and Pethidine 1.0 gm

Treatment of morphine poisoning…

- Gastric lavage (even injected morphine is excreted in stomach). KmNO4 converts morphine into oxymorphine .
- Activated charcoal is the GI decontamination method of choice for patients with opiate intoxication following ingestion.
- Enema and purgatives
- Airway control and adequate oxygenation, Endotracheal intubation is indicated in patients who cannot protect their airway.
- Symptomatic treatment.

- ANTIDOTES OF MORPHINE POISONING:

- Naloxone 0.4 -2 mg. I.V. every 5 minute till the patient became conscious and pupil dilates .

- 0.1 mg/kg in the child or infant

- Nalmefene and Naltrexone are newer opioid antagonists that have longer half-lives than Naloxone (4-8 h and 8-12 h vs. 1 h).

- Methadone, a long-acting narcotic often used to attenuate withdrawal symptoms and used in narcotics recovery programs, also has extensive potential for abuse.

ORGANOPHOSPHORUS POISONING

- Organophosphate (OP) compounds are a diverse group of chemicals used in both domestic and industrial settings.
- Examples of organophosphates include **insecticides** (*malathion, parathion, diazinon, fenthion, dichlorvos, chlorpyrifos, ethion*), **nerve gases** (*soman, sarin, tabun, VX*), **ophthalmic agents** (*echothiophate, isoflurophate*), and **antihelmintics** (*trichlorfon*). **Herbicides** (*tribufos [DEF], merphos*) are tricresyl phosphate–containing **industrial chemicals.**
- The primary mechanism of action of organophosphate pesticides is inhibition of acetylcholinesterase (AChE).
- Organophosphates inactivate AChE by phosphorylating the serine hydroxyl group located at the active site of AChE.
- Once AChE has been inactivated, ACh accumulates throughout the nervous system, resulting in overstimulation of muscarinic and nicotinic receptors

- Accumulation of acetylcholine leading to hyperexcitation of muscle.
- A 30% drop in activity of cholinestrase result in toxicity.
- Clinical manifestation of OP poisoning according to receptor

Muscarinic	Nicotinic	Central
Miosis Blurred vision Nausea Vomiting Diarrhoea Salivation Lacrimation Bradycardia Abdominal pain Diaphoresis Wheezing Urinary Incontinence Fecal Incontinence	Muscle Fasciculations Paralysis Pallor Muscle weakness Hypertension Tachycardia Mydriasis (rare)	Unconsciousness Confusion Toxic psychosis Seizures Fatigue Respiratory Depression Dysarthria Ataxia Anxiety

Signs & Symptoms of organophosphorus poisoning…

- Poisoning may occurs by ingestion , inhalation, or absorption through skin.
- Initial symptoms: headache, malaise, constrictio of chest, photophobia.
- In 1-8 hours: rest all feature mentioned above will appear.
- In severe cases: Pulmonary and cerebral oedema, cyanosis, pin point pupil, convulsion , coma and death.
- **Fatal dose: Varies in between 25 mg to 25 gm.**
  - TEPP: 5gm
  - Parathion: 150 mg
  - Malathone: 25 gm
  - Diazinon: 10-20 gm.

Treatment of OPP poisoning…

- The goal of treatment is: to reduced absorption of toxins, enhance elimination and neutralization of toxins.

1. **Reduce Absorption :**

- Removal from surface of skin, eyes and hair
- Emesis induction
- Gastric lavage with KMnO4 or with Na2SO4
- Activated charcoal administration and cathartics
- Dilution—milk/other drinks for corrosives
- Whole bowel irrigation & Endoscopic or surgical removal of ingested chemical
- Skin decontamination—important aspect—not to be neglected:

– Remove contaminated clothing

– Wash with soap and water (soaps containing 30% ethanol advocated).

2. Antidotes:

- Atropine blocks Muscarinic effect.
- Atropine given initially 2-4 mg in moderate poisoning and 406 mg in severe poisoning I.V. or I.M. and repeated 2-4 mg at 3-10 minute interval.
- Atropine is given till the sign of atropinization in the form of dry mouth, dilated pupil, tachycardia and warmth appears.
- Dose of atropine must be regulated cautiously so that no cyanosis and ventricular fibrillation occure.

3. Cholinesterase reactivators:

- Oxime compounds are used.
- For e.g. Diacetyl Monoxime (D.A.M.), Protopam (Palidoxime chloride), Palidoxime iodide, P2S (Pyridine aldoxime methane sulphonate), P2AM (Pyridine aldoxy methiodate) etc.
- These are given 1-2 gm I.V. as 5% solution in isotonic saline slowly in 10-20 minutes. Repeated 12-24 hourly.

4. Symptomatic treatment:

- Intravenous MgSO4 (4 g) given in the first day after admission have been shown to decrease hospitalization period and improve outcomes in patients with OP poisoning .
- Magnesium sulfate blocks calcium channels and thus reduces acetylcholine release.
- Benzodiazepines are widely used in human OP poisonings to control agitation, provide sedation in ventilated patients and control of seizures
- For intermediate syndrome, which is resistant to the standard treatment, supportive therapy and consideration of artificial respiration are recommended.
- For organophosphate-induced delayed neuropathy, standard therapy should be accompanied with neuroprotective drugs like corticosteroids.

SNAKE BITE TREATMENT…

- In all over word about 2500 species of snakes are found.
- In India, about 250 species of snake are found, out of which only 50 species are poisonous.
- Snakes are found all over the world, except, Greenland, Ireland, New Zealand,& Jamaica.
- Poisonous snake have elongated body and short tail. There is no limb, eyelids to be fused appear to be absent.
- Body is covered with scales, have 2 eyes and 2 nostrils, no external ear.
- Tongue is forked and help as sense organ.

Features	Poisonous snake	Non- poisonous snake
Body Scale	On anterior side and large covered the whole width	Small and moderate
Head scales	small	large
Fangs	Fangs are present and are canalized (in vipers) and grooved (in colubrine)	Usually fangs are absent, if present, they are short and solid.
Tail	Are compressed	Not compressed
Appearance	Generally nocturnal	Not nocturnal
Bite mark	Shows 2 fang mark and there may be present the marks due to other small teeth (in colubrine)	No fang mark
saliva	Toxic	Non toxic

Classification of poisonous snakes…

1. **Colubrine**: Elapidae: Cobra and Krait (pupils are round)

   -Hydrophidae: Sea snake (pupil are round).

2. **Viperiaedae:** Vipers (pupils are vertical)

- **On the basis of poisons snakes are classified as:**

1. ***Elapids: Neurotoxic***: fangs are 4-6 mm
2. ***Vipers: Vasculotoxic***: fans are 12-15 mm mobile
3. ***Sea snakes: Myotoxic***: fangs are 2-4 mm

Poisonous snakes in INDIA…

1. **Cobra (Nag)** : ***Naja naja*** : it is usually black, 5-6 feet and is proffered in populated area.
2. **King Cobra** :6-18 feet, preferred in forests.
3. **Common krait:** Kawariya, Chitti: ***Bungarus caeruleus.*** usually steel black, 3-5 feets , preferred area is near the houses. There are single or double white arches on back.
4. **Russel viper**: Ghonas, (***Dabiola russelii***), is 4-5 feet long, head is flat , triangular and has white V shape mark pointing forwards. It is produced larger hassling sound when about to attack.
5. **Pit-vipers:** 1-3 feet, has a heat sensing green pit between each eye and nostrils.
6. **Saw-scaled vipers**: Fursa: 1-1.5 feet, usually brown in colour, the rough scales on body produce rustling sound when snake moves.
7. **Sea snake**: have a snout and tail is fin like.

Snake venom…

- Snake venom (toxalbumin) is heterogenous mixture of proteins produced and stored in specialized salivary gland.
- Snake venoms contain more than 20 different constituents, mainly proteins, including enzymes and polypeptide toxins. The following venom constituents cause important clinical effects:

1. **Procoagulant enzymes :(Viperidae)** that stimulate blood clotting but result in incoagulable blood.

-Russell's viper venom contain several different procoagulants which activate different steps of the clotting cascade. The result is formation of fibrin in the blood stream

- Eventually, and sometimes within 30 minutes of the bite, the levels of clotting factors have been so depleted ("consumption coagulopathy") that the blood will not clot.

2. ***Haemorrhagins***: (zinc metalloproteinases) that damage the endothelial lining of blood vessel walls causing spontaneous systemic haemorrhage.

3. ***Cytolytic or necrotic toxins*** - these digestive hydrolases (*proteolytic enzyme and phospholipases A*) polypeptide toxins and other factors increase permeability resulting in local swelling. They may also destroy cell membranes and tissues.
4. ***Haemolytic and myolytic phospholipase A2*** - these enzymes damage cell membranes, endothelium, skeletal muscle, nerve and red blood cells.
5. ***Pre-synaptic neurotoxins (Elapidae and some Viperidae)*** - these are phospholipases A2 that damage nerve endings, initially releasing acetylcholine transmitter, then interfering with release.
6. ***Post-synaptic neurotoxins (Elapidae***) - these polypeptides compete with acetylcholine for receptors in the neuromuscular junction and lead to curare-like paralysis

Fatal dose of snake venom…

Snake	Fatal dose	Amount injected per bite
Cobra	12 mg of dried venom	200-350 mg
Kraits	6 mg of dried venom	20-22 mg
Russels viper	15 mg of dried venom	150-200 mg
Saw scaled viper	8 mg of dried venom	25 mg

- **Fatal periods: In colubrine: 20 min to 6 hours and in viperine 2-4 days.**

Signs & Symptoms of snake bite....

- ***General signs and symptoms of Elapid envenomation...***

- Swelling and local pain, Local necrosis and/or blistering Cobra snake bite).
- Descending paralysis, initially of muscles innervated by the cranial nerves, commencing with ***ptosis, diplopia, or opthalmoplegia***.
- Paralysis of jaw and tongue may lead to upper airway obstruction and aspiration of pooled secretions because of the patient inability to swallow.
- Numbness around the lips and mouth, progressing to pooling of secretions, bulbar paralysis and respiratory failure.
- Hypoxia due to inadequate ventilation can cause cyanosis, altered sensorium and coma. This is a life threatening situation and need urgent intervention.
- Stomach pain which may suggest submucosal hemorrhages in the stomach (Krait).

- ***General signs and symptoms of Viperine envenomation…***

- Swelling and local pain. Tender enlargement of local lymph nodes as large molecular weight Viper venom molecules enter the system via the lymphatics.
- Bleeding from the gingival sulci and other orifices such as epistaxis.
- The skin and mucous membranes may show evidence of petechiae, purpura ecchymoses.
- Vomiting and Acute abdominal tenderness which may suggest gastro-intestinal or retro peritoneal bleeding.
- Low back pain, indicative of an early renal failure or retroperitoneal bleeding,.
- Hypotension resulting from hypovolaemia or direct vasodilatation.
- Lateralising neurological symptoms and asymmetrical pupils may be indicative of intra-cranial bleeding.
- Muscle pain indicating rhabdomyolysis, Parotid sr,velling, conjunctival oedema, sub-conjunctival haemorrhage.

- ***Late-onset envenoming:***

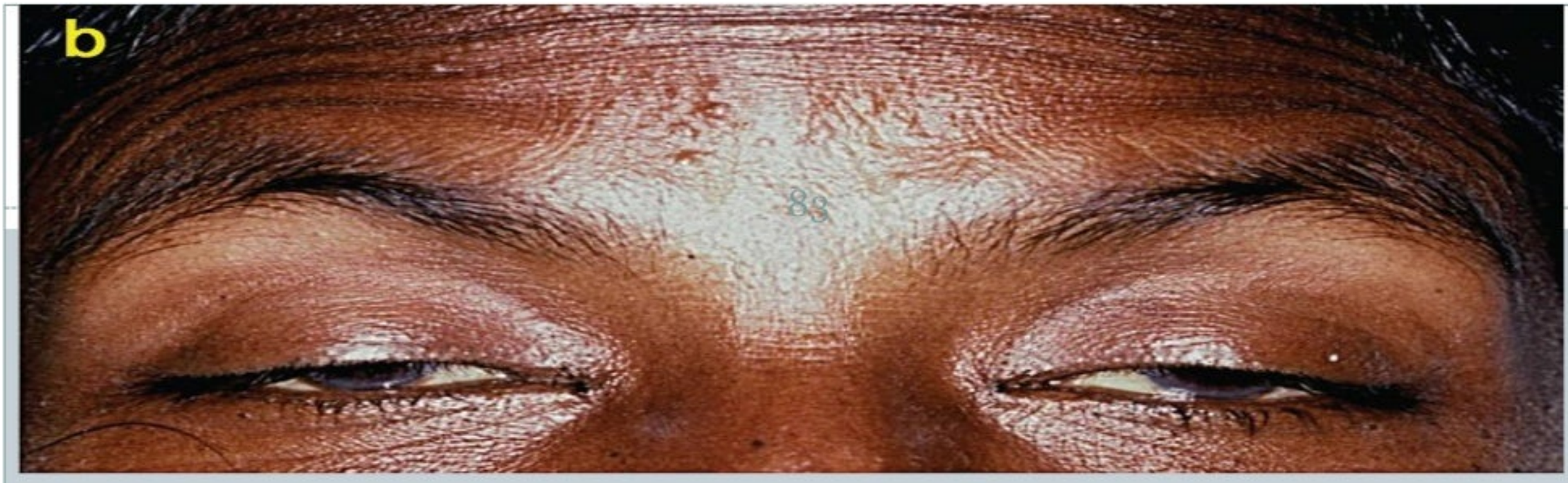
- The patient should be kept under close observation for at least 24 hours.

- Many species, particularly the Krait and the Hump-nosed pit viper are known for the length of time it can take for symptoms to manifest.

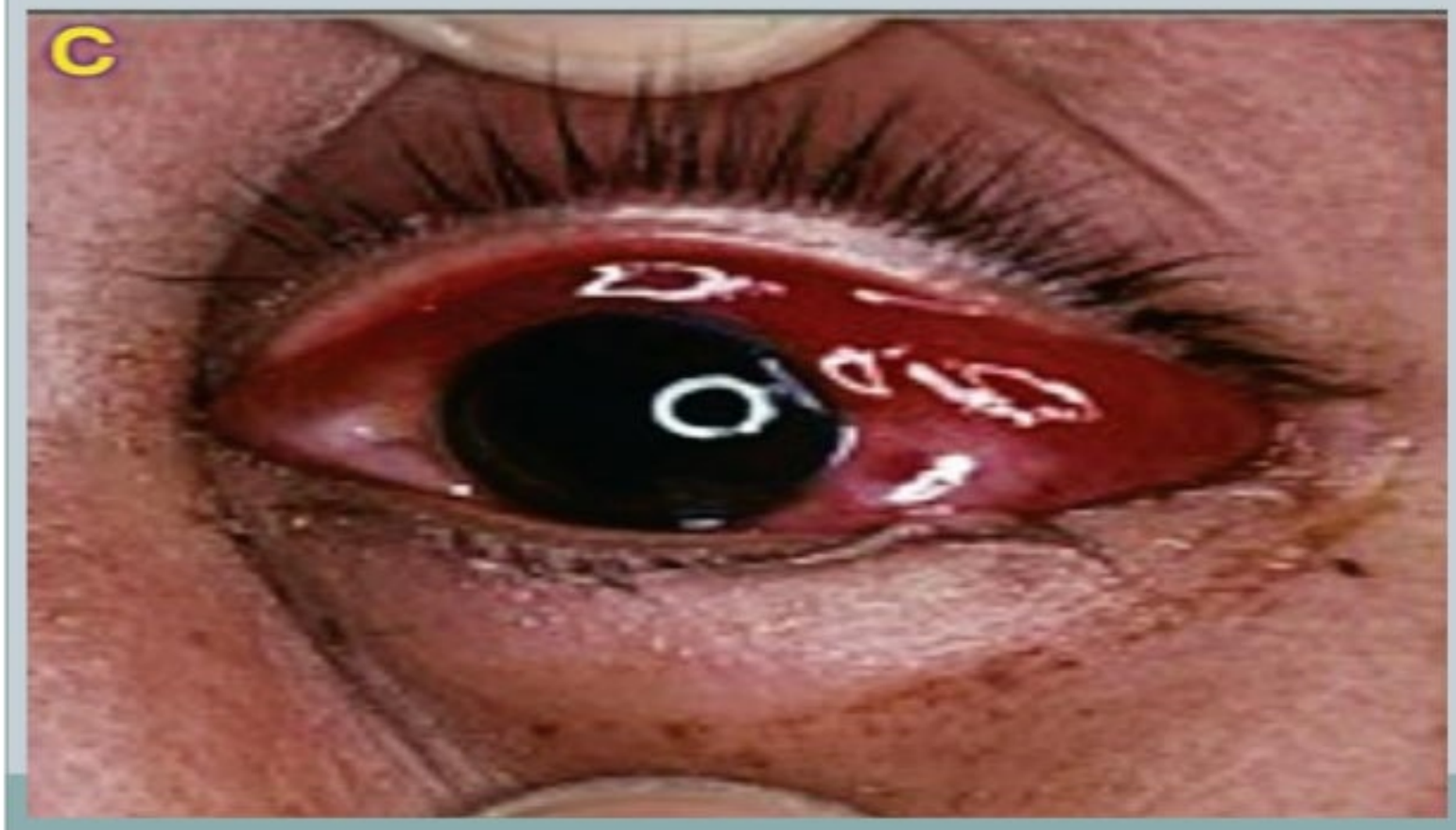
- Often this can take between 6 to 12 hours.

- This is also particularly pertinent at the start of the rainy season when snakes generally give birth to their young.

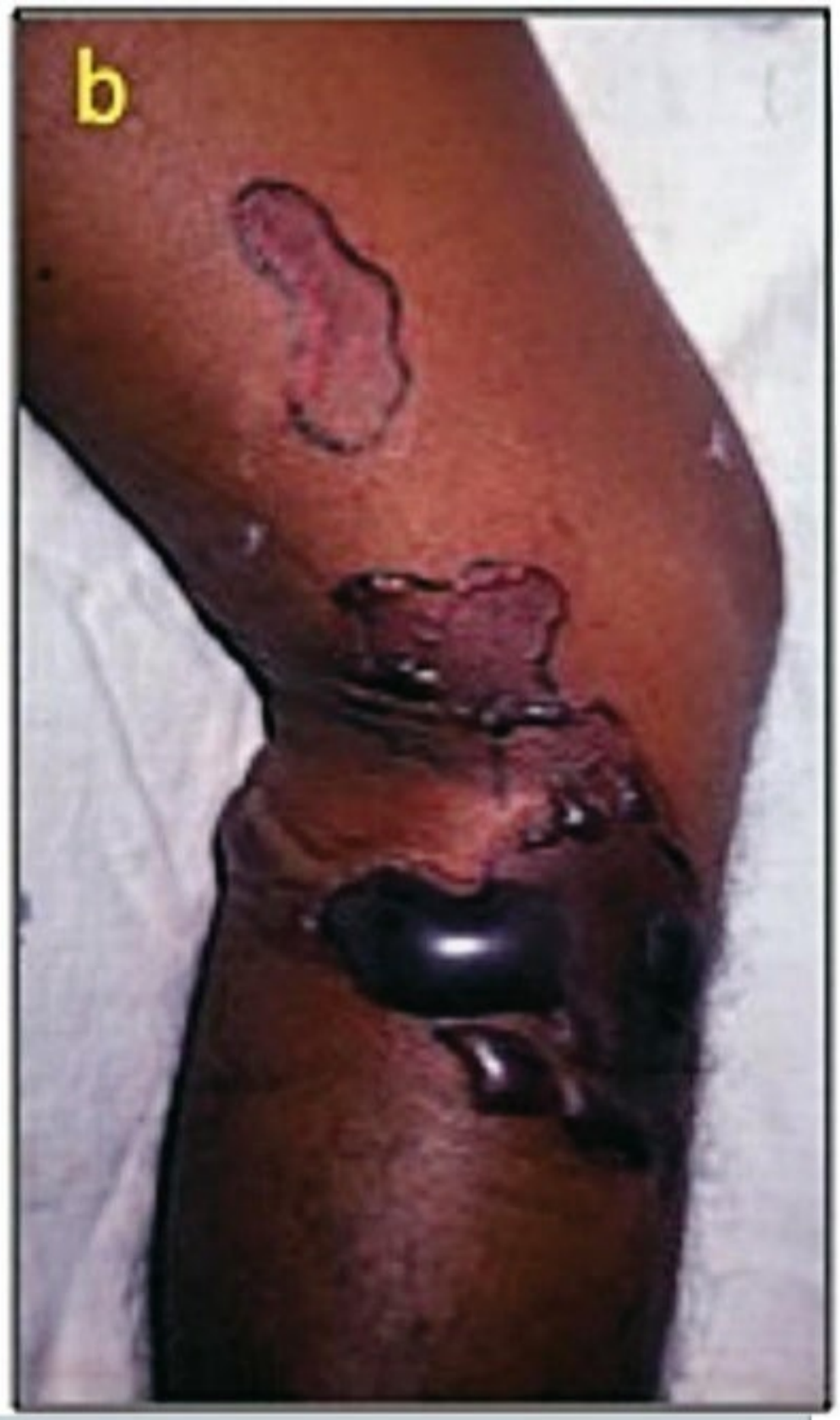
- Juvenile snakes, 8-10 inches long, tend to bite the victim lower down on the foot in the hard tissue area, and, thus any signs of envenomation can take much longer to appear.



Bilateral ptosis (Krait bite)



Chemosis, a sign of generalized increase in capillary permeability (Russell Viper bite)



local swelling, blistering and demarcated areas of dermonecrosis in a patient. (Cobra bite)

Treatment of snake bite…..

- The first aid treatment of snake bite in India is currently based on mnemonics: RIGHT

R. = Reassure the patient. 70% of all snakebites are from non- venomous species. Only 50% of bites by venomous species actually envenomate the patient.

I = Immobilize the bitten limb in the same way as a with fractured limb. Use bandages or cloth to hold the splints, not to block the blood supply or apply pressure. Do not apply any kind of compression in the form of tight ligatures, they don't work and can be dangerous.

G. H. = Get to Hospital Immediately. Traditional remedies have NO PROVEN benefit in treating snakebite.

T= Tell the doctor of any systemic symptoms of the patient such as ptosis that manifest on the way to hospital.

- The treatment step includes:

1. To allay fear of anxiety
2. Prevention of spread of venom
3. Use of antivenom serum
4. Treatment of absorbed poison
5. Symptomatic and general treatment.

- ***To allay fear and anxiety of patient:***

- Fear and tension of snake bite is commonest cause of death.
- Every patient should be assured that all snakes are not poisonous and that poisonous snake bite can be treatable.

- ***Prevention of spread of venom…***

- Apply tourniquets proximal to site of bite, though it is not recommended now, if it need to apply, preferably two tourniquets applied , out of which one should be on single bone.
- Tourniquets should be release for 20-30 second in every 10-15 minute.
- Do not apply too tight tourniquets as after reaching of hospital, when tourniquets removed, strong surge of venom may produces paralysis.
- Immobilize the parts since it prevent the spread of venom and relieve pain.

- Snakebite can often cause severe pain at the bite site. This can be treated with painkillers such as ***Paracetamol.*** Adult dose of 500-1000m9 4-6 hourly. Pediatric dose 10mg/kg every 4-6 hourly orally.
- **Aspirin should not be used** due to its adverse impact on coagulation. Do not use non steroidal anti-inflammatory drugs (\lSAIDs) as they can cause bleeding. This can be particularly dangerous in a patient already having coagulopathy.

Use of ANTIVENOM:

- The ASV available in India is polyvalent i.e. it is effective against all the four common species; Russell's Viper, Common Cobra Common Krait and Saw scaled Viper.

- There are no currently available monovalent ASVs in India .

- ASV is produced in both liquid and lyophilized forms and is available as granular powder.

- It is reconstituted by adding 10 ml of distilled water (be sure that it is a clear solution) and this solution is potent for 10 years.

- As it is a serum, hence time and patient condition permitted then sensitivity test should be done.

- If the person is sensitive to serum, then he is de-sensitized by injecting multiple small doses.

- Treatment of absorbed poison:
- in ELAPIDS:

- Alternate 0.6 mg atropine and 0.5 mg neostigmine.
- In paralytic case, S.C. adrenaline and I.M. calcium chloride.

- In Vipers:
- 30,000-40,000 units heparin.
- 300-600 gm of fibrinogen

- Symptomatic and General treatment:

- Use of tetanus toxoid

- Antibiotics
- Antihistaminic
- Analgesic, Barbiturates
- Blood transfusion, artificial oxygenation
- IgG is said to improve coagulopathy.

LEAD POISONING…

- Lead poisoning is a medical condition that occurs when people are exposed to lead compounds through inhalation, swallowing, and rarely, through the skin.
- Lead is 10 times more poisonous when get inhaled.
- ***Common source for exposure of lead is:***

-Paint - Many homes built before 1978 have paint with lead in it.

- Soil - In soil around a home.
- Dust - In household dust.
- Water - In drinking water. Your home might have plumbing that contains lead.
- Common poisonous preparation of lead is: Lead tetraoxide (Sindoor), Lead sulphide (Surmaa), Lead nitrate etc.
- On old painted toys and furniture.
- In food and liquids stored in lead crystal or lead-glazed pottery or porcelain.
- In industries that release lead into the air such as battery manufacturers

- Mechanism of action of lead:

- In inhibit enzyme –sulphyhydril group, ATPases, aminolevulinic acid dehydrase, and ferrochelates.

- Lead mimics biologically helpful minerals such as calcium, iron and zinc. Most lead settles in the bone, interfering with the production of red blood cells (leading to anemia).

- It also interferes with the absorption of calcium, which is required for strong bones, muscles, healthy muscle contraction and blood vessel function.

- Lead may fixes into brain in chronic condition.

Signs and Symptoms of acute lead poisoning….

- Acute poisoning is very rare.
- Constipation, stools are black and foul smelling.
- A sweet metallic, astringent taste.
- Burning of throat and vomiting (white or bloody).
- Colicky pain: relive by pressure.
- Oligouria, albuminuria
- Presence of lead and copro-pophyrin 3 in urine sample.
- Drowsiness, insomnia, headache, muscular cramps etc.
- IN sub-acute poisoning:

- Blue line on gum.
- Face is pale and look anxious, decreased body secretions.
- **Fatal dose of lead is uncertain : 0.5 -20 gm**
- **Fatal period is uncertain: 2-3 days to few month.**

Treatment of lead poisoning…

- Use of emetics and gastric lavage
- Fore pain in abdomen: Morphine and atropine.
- Calcium gluconate and CaCl2.
- Vit. C and Vit. D

- **Antidotes used:**

- Demulcent (as physical antidote)
- Sodium sulphate and magnesium sulphate (as chemical antidote)
- Pharmacological antidote: Calcium disodium editate, 1 gm per day as slo0w I.V. drip as 3% solution in saline.

Chronic lead poisoning ….

- Also known as plumbinism or saturnism
- It occurs due to occupational exposure , to thosw working in industries used lead.
- The clinical manifestations as:

- Hypochromic anemia
- Defective hemoglobin formation
- Increased fragility and reduced life span of RBC
- Arthralgia involving larger joint
- Increased B.P., arteriosclerotic changes
- Delirium
- Paralysis (lead palsy), affectign figners, wrist and foots
- Facial palor
- Infertility.
- Alopecia.

ARSENIC POISONING…

- Metallic arsenic is non-poisonous but arsenic salt produces toxicity.
- The inorganic forms consisting mostly of arsenite and arsenate compounds are toxic to human health.
- Humans are exposed to arsenic primarily from air, food and water.
- Drinking water may be contaminated with arsenic from arsenical pesticide, natural mineral deposits or improperly disposed arsenical chemicals.

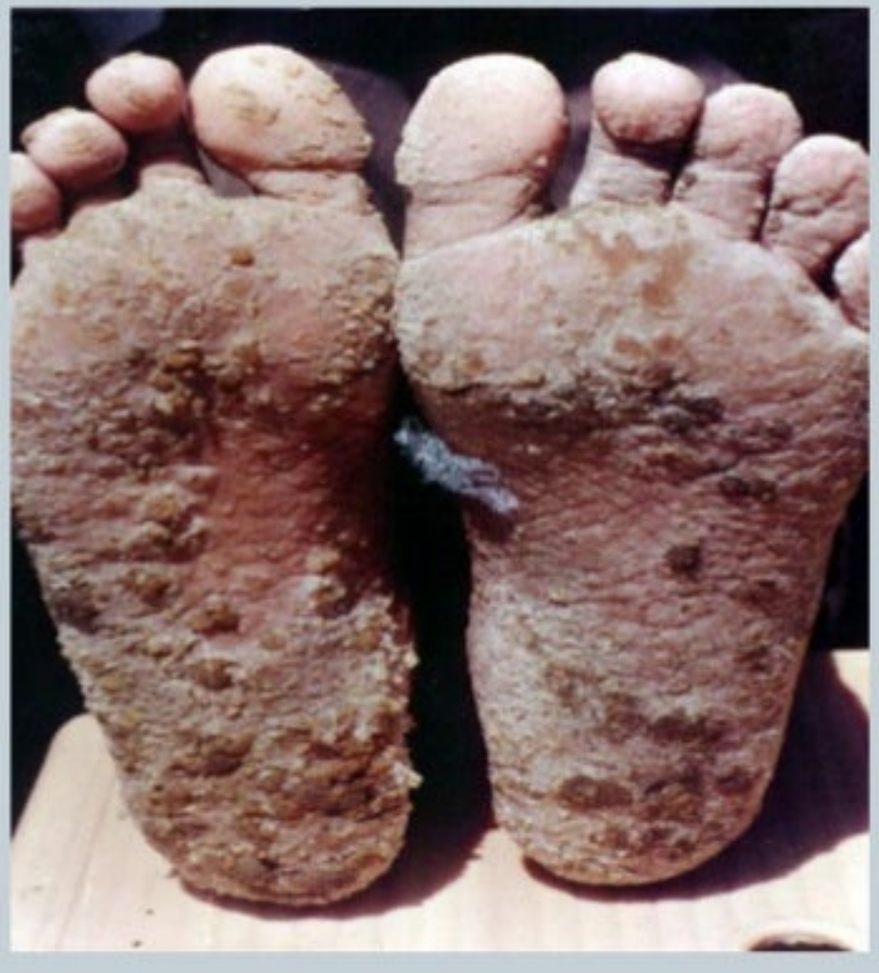
- ***Mechanism of poisonous action:***

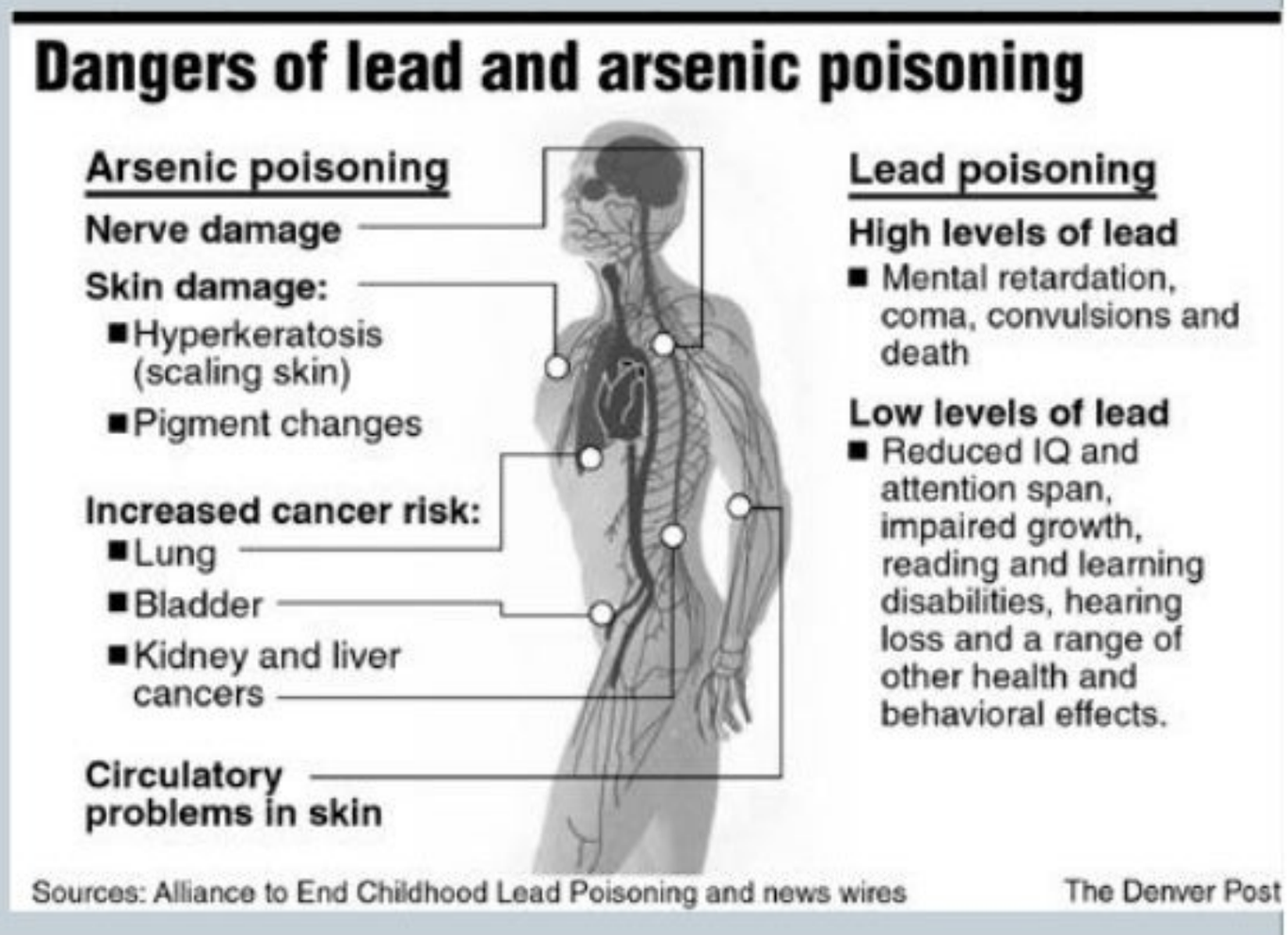
- Trivalent compound interfere with cell metabolism and oxidation by combining with SH enzymes (pyruvic dehydrogenase). This block the kreb cycle and result in depletion of ATP
- Pentavalant compound uncouple the mitochondrial oxidative Phosphorylation

Sign & symptoms of acute arsenic poisoning…

- The usual manifestation consists of nausea, vomiting, abdominal pain, watery diarrhoea (may be like "rice water") and possibly GI haemorrhage.
- Subsequent dehydration and hypotension, if severe, may lead to circulatory collapse.
- An acute encephalopathy may ensue with headache, confusion, delirium, seizures and coma.
- In less severe cases, irritability, personality change, and hallucinations may occur.
- A range of dysrhythmias may be seen including prolonged QT interval and torsade de pointes.
- Oral ingestion of high doses of arsenic may cause an acute lung injury, acute respiratory distress syndrome, or pulmonary oedema.
- acute hepatitis, haemolytic anemia, pancytopenia, acute renal failure, and rhabdomyolysis
- Fever may be present in acute poisoning. Fatigue, anorexia, or weight loss may occur in subacute poisoning.

- skin disease:
  - keratosis of palms and soles, and hyperpigmentation





- Fatal dose: 180-20 mg, trivalant are more toxic.

- Fatal Period: In narcotic form: sudden death occurs in 2-3 hours and in GI form 12-48 hours.

- **Treatment of Arsenic poisoning:**

- Use of emetics and gastric lavage with ferrickl oxide.

- Remove the patient from the source of arsenic; if there is skin contamination, wash with copious water; seal contaminated clothing.
- Resuscitate ('ABC' principles). Gastric lavage: consider if a significant amount has been ingested.

- Whole bowel irrigation with polyethylene glycol may be used to prevent arsenic absorption.
- Surgical and endoscopic removal of arsenic has been used successfully.
- Supportive treatment:

- Oxygen; bronchodilators if there is bronchospasm; positive end-expiratory pressure (PEEP) for pulmonary oedema.
- Intravenous fluids for hypovolaemia; blood transfusion for GI haemorrhage.
- Inotropes for myocardial depression.
- Torsades de pointes may be treated with magnesium sulfate, or with pacing or isoprenaline (if bradycardia-dependent).
- Treat seizures (diazepam, lorazepam +/- phenytoin).

Mercury poisoning

- There are 3 different forms of mercury

  - elemental

  - inorganic

  - organic

- Each has a different toxicological profile

Sources of mercury

- Elemental mercury:
  - Sphygmomanometers, thermometers, barometers
  - Liquid at room temp – volatilises easily
- Inorganic mercury:
  - Traditional remedies (ayurvedic, chinese)
  - Used in gold extraction, caustic soda manufacturing
  - Rodenticides
- Organic mercury:
  - Fungicides, seed dressings
  - Methylmercury in fish …

Organic mercury poisoning: Rare … but severe

- Exposure: ingestion, topical or inhalation
- CNS Toxicity:
  - poor concentration, fatigue, ataxia, tremor, constricted visual fields,
  - coma & convulsions
- BM suppression
- Renal toxicity - dealkylation to inorganic form
- Poorer response to treatment

Inorganic mercury poisoning

- Gastrointestinal phase: Hg^{2+} is a potent GI irritant
  - – gingivitis, stomatitis
  - - oesophageal, gastric, small and large bowel erosions
  - - haematemesis, bloody diarrhoea, CVS collapse
- Systemic toxicity: Hg^{2+} inhibits sulphydryl enzymes
  - – hypotension, lactic acidosis
- Nephrotoxicity: Hg^{2+} deposits in the tubules → ATN
  - – acute renal failure
  - - potentially leads to CRF in survivors

Diagnosis of Mercury poisoning

- Blood mercury:
  - only really useful acutely
  - normal <10µg/l
  - symptoms with blood mercury >150-200µg/l
- Urine mercury
  - probably the most reliable indicator
  - normal <10µg/l
  - symptoms with urine mercury >100-150µg/l
- U&E
- Radiology: for elemental ingestion/aspiration/injection

Treatment of Mercury poisoning

- Remove from source
- Supportive care
  - particularly important with inhalation
- **DMPS** Chelation (2,3-Dimercapto-1-propanesulphonate)
  - Chelation therapy of choice for mercury
  - For both acute and chronic mercury poisoning
  - For all forms of Hg (inorganic > metallic >> organic)
  - Indications:
    - symptomatic patients
    - blood/urine mercury persistently > 100 - 150µg/l