FORENSIC MEDICINE & TOXICOLOGY · MBBS

Common Poisons

Detailed notes on 26 poisons: names, class, fatal dose, active principle, mechanism, signs and symptoms, treatment and medicolegal aspects.

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Contents

01	Abrus precatorius	3
02	Dhatura	6
03	Croton	9
04	Castor	11
05	Marking nut	14
06	Nux vomica	17
07	Aconite	20
08	Poppy husk	23
09	Poppy capsule	25
10	Poppy seeds	28
11	Crude opium	30
12	Tobacco	33
13	Cannabis	36
14	Cerbera thevetia	39
15	Nerium oleander	42
16	Hydrochloric acid (HCl)	45
17	Nitric acid (HNO_3)	48
18	Sulphuric acid (H_2SO_4)	50
19	Malathion	53
20	Parathion	56
21	DDT	59
22	Lead tetroxide	61
23	Lead sulphide	64
24	Oxalic acid	66
25	Carbolic acid	68
26	Calomel	71
	Quick-reference table	74

Colour key

- Corrosive (5)
- Irritant: metallic (3)
- Cardiac (4)
- Irritant: vegetable (4)
- Neurotic (7)
- Agrochemical (3)

How these notes are built

- **Old book advice.** Treatments the books still print but current practice has dropped (emetics, acidifying the urine, bicarbonate for acids) are marked as cautions, not as steps to follow.
- **Law.** Sections are cited under the Bharatiya Nyaya Sanhita (BNS) and Bharatiya Nagarik Suraksha Sanhita (BNSS), with the old IPC and CrPC numbers in brackets.
- **Format.** Each point gives the medical or legal term first, then one short plain sentence explaining it.

Abrus precatorius

Common names: Rati, Gunchi, Gunja, Gulagunchi, Jequirity, Indian liquorice, Rosary pea, Rosary bead, Crab's eye



Abrus precatorius (rati, jequirity): scarlet seeds with a black spot at the hilum, in the opened pod

Photo: Wikimedia Commons, Abrus seed (2237059484).jpg



Abrus precatorius seeds, museum specimen with scale. Each seed is about 5 to 7 mm long

Photo: Didier Descouens (Muséum de Toulouse, MHNT series), CC BY-SA 4.0, via Wikimedia Commons

SCIENTIFIC NAME	Abrus precatorius Linn., family Fabaceae (Leguminosae)
CLASS OF POISON	Irritant: organic, vegetable
PART POISONOUS	All parts are poisonous. The seeds are the part used as poison: egg-shaped, bright scarlet with a large black spot at one pole, about 8 x 6 mm, weighing about 105 mg (Reddy) or 120 mg (Vij). White, black, yellow and blue varieties also occur.
FATAL DOSE	90–120 mg, 1–2 seeds, by injection (Reddy, Vij); 60–120 mg abrin (Rao)
FATAL PERIOD	3–5 days
ACTIVE PRINCIPLE	Abrin, a heat-labile toxalbumin (phytotoxin) that acts like viperine snake venom. The seeds also carry abrine (N-methyltryptophan, an amino acid), abralin (a glucoside), abric acid, a haemagglutinin, a lipolytic enzyme and urease.

Mechanism of action

- **Ribosome-inactivating toxalbumin.** Abrin stops protein synthesis inside the cell, so the cell dies.
- **Haemagglutination and haemolysis.** Abrin clumps and bursts red cells; the freed haemoglobin can block the renal tubules.
- **Local cytotoxic action.** At an injection site it causes inflammation, oedema, haemorrhage and necrosis, much as a viper bite does.
- **Route-dependent toxicity.** Given under the skin, abrin is about 100 times as toxic as by mouth (Reddy), because the gut digests part of it and the hard seed coat resists breakdown.

Signs and symptoms

- **Latent period.** Symptoms after ingestion may be delayed from a few hours to 2–3 days.

- **Seeds swallowed whole.** The hard coat passes through the gut unbroken, so whole or cooked seeds are harmless; chewed seeds poison (Vij).
- **Haemorrhagic gastritis.** Abdominal pain, nausea, vomiting and bloody diarrhoea follow chewed seeds; rectal bleeding may occur.
- **Circulatory collapse.** Cold sweat, trembling hands and a weak rapid pulse mark the slide into shock.
- **Miosis.** Reddy records constricted pupils.
- **Local reaction at an injection site.** Painful swelling and ecchymosis (bruising) appear, then inflammation and necrosis, with oozing of blood-stained fluid.
- **Systemic features after injection.** Faintness, vertigo, vomiting, dyspnoea and general prostration follow; convulsions may precede death from cardiac failure.
- **Delayed organ damage.** Cytotoxic injury to the brain, liver, kidneys and adrenals appears 2–5 days after exposure.
- **Picture in cattle.** The animal stops eating, drops down in 3–4 days, cannot rise, becomes comatose and dies; the owner usually takes it for a viper bite.

Treatment

- **Gastric lavage.** Rao advises the usual decontamination for patients who arrive within 4 hours of ingestion.
- **Activated charcoal.** It binds toxin still in the gut.
- **Purgative (cathartic).** It hurries seed fragments out; spontaneous diarrhoea may make it unnecessary (Rao).
- **Excision of the sui.** The needle and the surrounding tissue are dissected out of an injection wound so no more toxin is absorbed.
- **Antiabrin injection.** Reddy lists anti-abrin serum as the specific antidote.
- **Urinary alkalinisation.** Sodium bicarbonate 10 g orally per day keeps the urine alkaline and stops haemoglobin blocking the renal tubules (Reddy); Rao gives 10% sodium bicarbonate IV.
- **Supportive care.** IV fluids correct shock and dehydration; fits and arrhythmias are treated as they arise.

Medicolegal aspects

- **Cattle poison.** Leather workers kill cattle with 'suis' to get hides cheaply, or kill an enemy's animal for revenge.
- **Making the sui.** Decorticated seeds, alone or with datura, opium or onion, are pasted with spirit and water, rolled into sharp needles about 15 mm long weighing 90–120 mg, and sun-dried. Two needles are set in a wooden or bamboo handle and driven into the animal with a hard blow.
- **Mimics viperine snake bite.** Local swelling, haemorrhagic ooze and death in 3–4 days make the owner suspect snake bite, so poisoning goes unnoticed.
- **Homicide.** A sui held between two fingers is driven into the victim with a slap, often on the cheek.
- **Malingering.** Powdered seeds put in the eye produce conjunctivitis.
- **Abortifacient and contraceptive.** Seeds taken internally disturb uterine function; the juice is also used on an abortion stick.
- **Arrow poison.** Reddy and Vij both list abrus among Indian arrow poisons.
- **Law.** BNS 325 (formerly IPC 428/429): killing or maiming an animal. BNS 103 (formerly IPC 302): murder. BNS 123 (formerly IPC 328): causing hurt by poison. BNS 88 (formerly IPC 312): causing miscarriage.

AUTOPSY FINDINGS AND SAMPLES

- **Sui fragments.** Pieces of the needle may lie in the wound, with oedema and haemorrhage around the puncture.

- **Petechial haemorrhages.** Tiny bleeds lie under the skin, pleura, pericardium and peritoneum.
- **Visceral congestion.** Internal organs are congested and show haemorrhages; the gut is inflamed after oral poisoning.
- **Samples.** The injection site is excised with the needle fragments; stomach and contents, intestine, liver and kidney go in saturated common salt solution, and any seeds are kept dry for botanical identification.

> REMEMBER
>
> *Swallowed whole, rati seeds pass harmlessly; chewed or injected as a sui, abrin kills like a viper.*

Dhatura

Common names: Dhatura, Thorn apple, Safed dhatura (white), Kala dhatura (purple), Ummatta, Shivashekhara, Jimsonweed, Devil's trumpet, Mad apple



Datura stramonium (dhatura, thorn apple): spiny capsule split open to show the dark, kidney-shaped seeds
Photo: Wikimedia Commons, Datura stramonium fruit, doornappel vrucht (1).jpg

SCIENTIFIC NAME	Datura fastuosa (syn. D. metel) with varieties D. alba (white flower) and D. niger (deep-purple flower); Datura stramonium in the Himalayas. Family Solanaceae
CLASS OF POISON	Neurotic: cerebral, deliriant
PART POISONOUS	All parts, including the nectar (honey), are poisonous. Seeds and fruit are the most toxic. A spiny fruit (thorn apple) holds about 450 to 500 yellowish-brown seeds.
FATAL DOSE	0.6 to 1 g of seeds, about 100 to 125 seeds (Reddy); 50 to 100 seeds (Vij); 50 to 75 seeds, 0.6 to 1 g (Rao)
FATAL PERIOD	24 hours (Reddy, Vij); 3 to 24 hours (Rao)
ACTIVE PRINCIPLE	Belladonna alkaloids: hyoscine (scopolamine), hyoscyamine and traces of atropine, 0.2 to 1.4% in the plant. Hyoscine dominates, so drowsiness follows the delirium.

Mechanism of action

- **Antimuscarinic (anticholinergic, parasympatholytic) action.** The alkaloids competitively block acetylcholine at muscarinic receptors, so secretions dry up and the pupils dilate.
- **Central stimulation, then depression.** Higher centres are excited first, causing delirium; later the medullary centres are depressed, and respiration fails.
- **Loss of sweating with stimulation of the heat-regulating centre.** The skin turns hot and dry, and the temperature rises 2 to 3 degrees.
- **Rapid renal excretion.** The alkaloids appear unchanged in urine within hours and clear in 10 to 20 hours (Vij), which is why urine is the key sample.

Signs and symptoms

- **Onset.** Within half an hour of eating seeds, within minutes of a decoction. A bitter taste comes first.
- **Xerostomia with dysphagia.** Mouth and throat go dry, speech is difficult and the voice hoarse (dry as a bone).
- **Flushing (red as a beet).** Cutaneous vessels dilate, so the face and conjunctivae look red. A scarlatiniform rash may appear.
- **Mydriasis with cycloplegia.** Pupils dilate widely, light reflex fades, and near vision blurs (blind as a bat). Photophobia and diplopia occur.
- **Hyperpyrexia with dry, hot skin (hot as a hare).** Sweating stops while the heat centre is stimulated.
- **Tachycardia.** Pulse runs 120 to 140 per minute, full and bounding, later weak and irregular. Blood pressure rises early (Vij).
- **Urinary retention.** The bladder fills but the patient cannot pass urine.
- **Muttering delirium (mad as a wet hen).** Restless, purposeless excitement with visual and auditory hallucinations; he fails to recognise relatives.
- **Carphologia.** He picks at bedclothes, pulls imaginary threads from his fingertips and threads imaginary needles. Gait is drunken (ataxia).
- **Stage of coma.** Excitement passes in 1 to 2 hours into deep sleep or coma; death, though rare, is by respiratory paralysis. Mnemonic: 8 D's (Reddy), 10 D's (Vij).

Treatment

- Gastric lavage with weak tannic acid (Reddy, Vij) or potassium permanganate 1:5000 (Rao). Gut motility is slow, so lavage still helps late.
- **Emetics.** Contraindicated: a delirious patient may fit or slip into coma and aspirate.
- **Activated charcoal, repeated, with a cathartic.** Repeated bowel wash-outs and free purgation for 3 to 4 days clear lodged seeds.
- **Physostigmine, the physiological antidote.** Reddy gives 1 mg i.v. or i.m., hourly if needed; Rao gives 2 mg slow i.v., repeated, not over 4 mg in 30 minutes, kept for severe delirium. It crosses into the brain, so it reverses the central effects.
- **Peripheral cholinergics.** Neostigmine (prostigmine) 0.5 to 1 mg or pilocarpine 5 to 15 mg s.c. act on the periphery only and do not clear the delirium.
- **Sedation for delirium.** Benzodiazepines (diazepam or lorazepam i.v.) are the current choice. Phenothiazines and haloperidol are avoided, as they add anticholinergic effect and lower the seizure threshold. Morphine is avoided, because it depresses the respiratory centre.
- **Supportive care.** Cool the patient for hyperpyrexia, catheterise the full bladder, and support breathing when coma deepens.

Medicolegal aspects

- **Stupefying poison (road poison, railroad poison).** Crushed seeds go into tea, sweets, chapatis, paan or prasad given to travellers at railway stations and choultries, and the victim is robbed while stupefied.
- **Seeds float in liquids, so poisoners prefer an infusion.** Seeds mixed with incense give fumes that cause lethargy.
- **Secondary offences.** Rarely used to facilitate rape or kidnapping; children are lured with dhatura-laced sweets.
- **BNS 123 (formerly IPC 328):** causing hurt by poison with intent to commit an offence, which covers stupefying for robbery.

- **Involuntary intoxication.** A person in datura delirium given the drug without his knowledge is not criminally responsible: BNS 23 (formerly IPC 85).
- **Accidental poisoning.** Children eat the fruit; seeds are mistaken for capsicum seeds; quacks misuse them. Suicide and homicide are rare.
- **Other uses.** Added to liquor or toddy, or to majoon, to strengthen intoxication; smoked with tobacco or ganja; used as abortifacient, aphrodisiac and love philter.
- **Datura seed vs capsicum seed.** Datura: larger, kidney-shaped, brown, pitted, double-edged border, odourless, bitter. Capsicum: smaller, round, pale yellow, smooth, single sharp border, pungent.

AUTOPSY FINDINGS AND SAMPLES

- **Asphyxial picture.** Findings are not characteristic; the lungs may be oedematous and the stomach mildly inflamed.
- **Seeds or fragments in the stomach and intestines.** They resist putrefaction and are found even in a decomposed or exhumed body.
- **Search the vomit, stomach contents and faeces carefully for seeds.** The mydriatic alkaloid itself may be destroyed by putrefaction (Vij).
- **Preserve urine as well as routine viscera.** The alkaloid is excreted unchanged in urine, which may test positive when the stomach wash does not.
- **Mydriatic (cat's eye) test.** A drop of the suspected fluid or urine in a cat's eye dilates the pupil within half an hour if datura is present.

> REMEMBER
>
> *Blind as a bat, hot as a hare, dry as a bone, red as a beet, mad as a hen.*

Croton

Common names: Jamalgota, Jamal gota, Naepala, Nepala, Purging croton



Croton tiglium (jamalgota): green three-lobed capsules. Each holds three seeds, the toxic part
Photo: Wikimedia Commons, Croton tiglium 02.JPG

SCIENTIFIC NAME	Croton tiglium Linn., family Euphorbiaceae
CLASS OF POISON	Irritant: organic, vegetable
PART POISONOUS	Seeds and the oil pressed from them (croton oil). Root is also used. Seeds are oval, blackish-brown with longitudinal lines and a white oily kernel, odourless, about the size of small castor seeds but without mottling.
FATAL DOSE	4–5 seeds or 1–2 mL oil (Reddy); 4 seeds or 20 drops oil (Vij)
FATAL PERIOD	6 hours to 3 days (Reddy); few hours to few days (Vij)
ACTIVE PRINCIPLE	Crotin, a toxalbumin like ricin, in the seeds; crotonoside, a less toxic glycoside. The oil holds a vesicating resin of crotonoleic acid, methyl crotonic acid and other fatty acids.

Mechanism of action

- **Toxalbumin (phytotoxin).** Crotin acts like ricin: it agglutinates and lyses red cells and damages cells.
- **Vesicant resin.** Crotonoleic acid in the oil blisters skin and mucosa on contact.
- **Drastic purgative action.** The resin inflames the bowel wall, which drives violent watery purging and fluid loss.

Signs and symptoms

- **Burning pain.** A hot burning sensation runs from the mouth to the stomach.
- **Ptyalism.** Excess salivation follows the acrid, burning taste of the oil.
- **Vomiting and purging.** Powerful, repeated purging is the leading feature; stools may be bloody.
- **Dehydration.** Fluid loss brings thirst, cramps and a weak pulse.
- **Vertigo and prostration.** Giddiness and extreme weakness set in as fluid is lost.

- **Collapse.** In substantial doses circulatory collapse precedes death.
- **Vesication of skin.** Applied externally, the oil causes burning, redness and blisters.
- **Dust exposure.** Inhaled seed dust irritates the eyes and airways.

Treatment

- **Gastric lavage (stomach wash).** It removes oil and seed fragments still in the stomach.
- **Activated charcoal.** Rao treats croton as for castor, which includes charcoal.
- **Demulcent drinks.** Milk, egg white or barley water coat the inflamed gut and ease irritation.
- **No purgative.** The poison is itself a drastic purgative, so the bowel needs no further stimulus.
- **Fluid and electrolyte replacement.** IV fluids correct the losses from vomiting and purging.
- **Skin decontamination.** Oil on the skin is washed off with soap and water.
- **Symptomatic care.** Pain, cramps and shock are treated as they arise.

Medicolegal aspects

- **Accidental poisoning.** Croton oil is taken in excess as a purgative, swallowed by mistake for castor oil, or seeds are eaten in place of castor seeds.
- **Croton versus castor seed.** Croton seeds are blackish-brown with longitudinal lines; castor seeds are glossy brown and mottled.
- **Abortifacient.** The root and oil are taken internally to procure abortion.
- **Arrow poison.** Reddy lists the oil and Vij the soft parts of the plant.
- **Homicide and suicide.** Both are rare. Rao cites a homicide recorded by the Madras Chemical Examiner in 1936 and a quack jailed for croton-seed pills.
- **Mass poisoning.** Rao reports a Delhi wedding where ice cream contaminated with croton oil caused vomiting and purging among guests.
- **Law.** BNS 123 (formerly IPC 328): causing hurt by poison. BNS 88 (formerly IPC 312): causing miscarriage. BNS 103 (formerly IPC 302): murder.

AUTOPSY FINDINGS AND SAMPLES

- **Gastroenteritis.** The stomach and intestinal mucosa are congested, inflamed and eroded.
- **Visceral congestion.** Internal organs are congested.
- **Seed coat fragments.** Pieces of the striated outer covering may be found in the gut and identified under the microscope (Rao).
- **Samples.** Stomach and contents, intestine, liver and kidney go in saturated common salt solution; seed remnants are kept dry for identification.

> REMEMBER
>
> *Jamalgota: striped seed, drastic purge. Castor seed is mottled; croton seed has longitudinal lines.*

Castor

Common names: Arandi, Castor oil plant, Castor bean, Mole bean



Ricinus communis (castor, arandi): glossy mottled seeds with a small caruncle at one end
Photo: H. Zell, CC BY-SA 3.0, via Wikimedia Commons

SCIENTIFIC NAME	Ricinus communis Linn., family Euphorbiaceae
CLASS OF POISON	Irritant: organic, vegetable
PART POISONOUS	Entire plant is poisonous; the seeds are most toxic, as is the press cake left after the oil is extracted. Seeds are oval, glossy and mottled, in a big and a small variety; the small ones resemble croton seeds. Castor oil itself holds no ricin.
FATAL DOSE	5–10 seeds (Reddy); about 10 seeds, ricin 6 mg (Vij)
FATAL PERIOD	2–5 days (Reddy); 2 to several days (Vij)
ACTIVE PRINCIPLE	Ricin, a water-soluble toxalbumin (glycoprotein), highest in the seeds. The seeds also carry a powerful allergen (CBA). Castor oil contains ricinoleic acid, a purgative, but no ricin.

Mechanism of action

- **Ribosome-inactivating toxalbumin.** Ricin blocks protein synthesis, so cells die; its effects are delayed but widespread.
- **Two-chain structure.** One chain binds the cell surface and lets the toxin in; the other disrupts protein synthesis (Rao).
- **Gut mucosal injury.** Ricin enters the gastrointestinal lining cells and causes haemorrhagic inflammation and severe diarrhoea, even when injected under the skin.
- **Haemagglutination and haemolysis.** Ricin clumps and bursts red cells, which leads on to jaundice and uraemia.

Signs and symptoms

- **Latent period.** Toxic features start 2–24 hours after ingestion (Vij); full effect may take up to 5 days.

- **Seeds swallowed whole.** The hard seed coat prevents absorption, so unbroken or cooked seeds are harmless; seeds must be chewed to release ricin.
- **Local burning.** Burning in the mouth, throat and stomach; the oral burn looks like an alkali burn (Reddy).
- **Acute gastroenteritis.** Salivation, nausea, severe vomiting, colicky pain and bloody diarrhoea follow.
- **Hypovolaemic shock.** Fluid loss causes thirst, dehydration, cramps in the calves, hypotension and a weak rapid pulse.
- **Late features.** Haemolysis, jaundice, uraemia, drowsiness, delirium and convulsions appear; consciousness is often kept till near the end.
- **Seed dust.** Dust causes conjunctivitis, sneezing, rhinitis, asthmatic bronchitis and dermatitis; it is highly allergenic and may cause anaphylaxis.

Treatment

- **Gastric lavage.** It removes chewed seed fragments from the stomach.
- **Activated charcoal.** It binds ricin still in the gut.
- **No cathartic.** Severe bloody diarrhoea and fluid loss are the main threats, so a purgative only worsens dehydration.
- **Demulcents.** Bland coating drinks soothe the inflamed gut.
- **Observation of the asymptomatic.** A patient who has chewed seeds but has no symptoms is watched for 4–6 hours after decontamination and told to return if symptoms appear (Rao).
- **IV fluids and electrolytes.** Rehydration is the mainstay; most patients recover on fluids alone (Rao).
- **Monitoring.** Blood glucose, haemolysis and renal function are watched; blood transfusion may be needed.

Medicolegal aspects

- **Accidental poisoning.** Children eat the attractive seeds out of curiosity or by mistake.
- **Occupational poisoning.** Workmen separating oil from the seeds may be poisoned by contact with the dust and press cake.
- **Homicide.** Rarely, powdered seeds are given to kill.
- **Malingering.** Seed powder put in the eye produces conjunctivitis.
- **Georgi Markov case (London, 1978).** A pellet thought to carry ricin was fired into the thigh from an umbrella tip; he died within days.
- **Bioterrorism.** Reddy and Vij list ricin as a biological warfare agent; it is a Schedule 1 chemical under the Chemical Weapons Convention, enforced in India by the Chemical Weapons Convention Act 2000.
- **Law.** BNS 103 (formerly IPC 302): murder. BNS 123 (formerly IPC 328): causing hurt by poison.

AUTOPSY FINDINGS AND SAMPLES

- **Haemorrhagic gastroenteritis.** The gut mucosa is congested, softened and inflamed, with erosions and submucous haemorrhages.
- **Serosal haemorrhages and oedema.** Bleeds lie in the pleura and pericardium; the liver, kidneys, spleen and lungs are oedematous, and the heart is dilated.
- **Seed fragments.** Pieces may be found in the stomach or intestine; the prismatic outer cells of the seed coat help identify them under the microscope (Rao).
- **Samples.** Stomach and contents, intestine, liver and kidney go in saturated common salt solution; seed fragments are kept dry for botanical identification.

REMEMBER

Swallowed whole, castor seed passes harmless; chewed, it releases ricin. Castor oil carries no ricin.

Marking nut

Common names: Bhilawa, Bhilawan, Bhilwa, Bibva, Bhela, Oriental cashew, Marking nut



Semecarpus anacardium (bhilawa, marking nut): black nuts on a shrivelled orange-brown receptacle. The juice of the pericarp is the irritant

Photo: Wikimedia Commons, Semecarpus anacardium 01.jpg

SCIENTIFIC NAME	Semecarpus anacardium Linn. f., family Anacardiaceae
CLASS OF POISON	Irritant: organic, vegetable
PART POISONOUS	The nut (fruit). Its thick cellular pericarp holds a brownish, oily, acrid juice that turns black on exposure to air. The nuts are black and heart-shaped, with a rough projection at the base. Washermen (dhobis) use the juice as marking ink on clothes.
FATAL DOSE	5–10 g
FATAL PERIOD	12–24 hours
ACTIVE PRINCIPLE	Semecarpol (about 0.1%, a monohydroxy phenol) and bhilawanol (15–17%), the acrid irritant oils of the pericarp juice.

Mechanism of action

- **Contact irritant and vesicant.** The juice blisters skin and mucosa; the acrid blister fluid spreads the reaction to neighbouring skin.
- **Eczematous (allergic-type) dermatitis.** Itchy eruptions appear wherever the serum touches, including the fingertips after scratching.
- **Gastrointestinal irritant.** Swallowed in large dose, the juice blisters the throat and inflames the stomach and bowel.

Signs and symptoms

- **Vesication of skin.** A painful blister with acrid serum forms where the juice is applied; the lesion looks like a bruise.

- **Eczematous eruption.** Itching spreads to adjacent skin; scratching produces similar lesions on the fingertips and under the nails.
- **Ulceration.** The blister may later ulcerate and slough.
- **Constitutional symptoms.** Fever and painful micturition with brown urine may accompany skin lesions (Vij).
- **Ophthalmia.** Juice in the eye causes severe conjunctivitis.
- **Oral and gastric irritation.** Taken by mouth the juice irritates less; large doses blister the mouth and throat and cause severe gastroenteritis.
- **Systemic collapse.** Dyspnoea, tachycardia, hypotension, cyanosis, absent reflexes, delirium and coma may precede death.

Treatment

- **Skin decontamination.** The area is washed with lukewarm water and an antiseptic, or soap and water.
- **Eye irrigation.** Saline washes out juice from the conjunctiva.
- **Gastric lavage.** It removes swallowed juice or ground nut.
- **Demulcent drinks.** Milk or barley water soothe the blistered mouth and gut.
- **Symptomatic care.** Liniments for skin lesions, analgesia, and support of breathing and circulation as needed.

Medicolegal aspects

- **Artificial (fabricated) bruise.** Juice applied to the skin produces a bruise-like lesion to support a false charge of assault.
- **Telling it from a true bruise.** The marking-nut lesion is dark brown, on accessible parts, with a regular vesicular margin, red inflamed surrounding skin, itching and acrid serum; a true bruise shows extravasated blood and changes colour with age. See the table below.
- **Proof of fabrication.** Chemical analysis of the blister fluid detects the juice.
- **Punishment and torture.** The juice is put in the vagina or on the genitals for suspected infidelity, or on prisoners to extract a confession.
- **Criminal abortion.** The bruised nut or juice is applied to the cervical os, often on an abortion stick.
- **Vitriolage.** The juice is thrown on the face or body, like acid, to injure.
- **Malingering and quackery.** Malingerers produce ophthalmia with it; accidental deaths follow quack remedies given internally or rubbed on limbs.
- **Law.** BNS 248 (formerly IPC 211): false charge of an offence. BNS 118(2) (formerly IPC 326): grievous hurt by poison or corrosive means. BNS 124 (formerly IPC 326A/326B): acid attack, as the law's 'acid' includes any substance of burning nature. BNS 88 (formerly IPC 312): causing miscarriage.

AUTOPSY FINDINGS AND SAMPLES

- **Oral and gastric blisters.** Blisters lie in the mouth, throat and stomach, which are congested and inflamed.
- **Gastroenteritis.** The gut is inflamed; the viscera are congested.
- **Skin lesions.** Black vesicles with acrid serum may be seen on the skin (Rao).
- **Samples.** Stomach and contents, intestine, liver and kidney go in saturated common salt solution. Blister fluid from a skin lesion is sent for chemical analysis, since it proves the juice was applied.

ARTIFICIAL BRUISE (MARKING NUT) VERSUS TRUE BRUISE

FEATURE	ARTIFICIAL BRUISE	TRUE BRUISE
Cause	Juice of marking nut, calotropis or plumbago	Blunt trauma

Site	Exposed, accessible parts	Anywhere
Colour	Dark brown; no colour change with time	Typical colour changes with age
Shape	Irregular	Usually round
Margin	Well defined, regular, covered with small vesicles	Not well defined, diffuse; no vesicles
Surrounding skin	Red and inflamed	No inflammation
Contents	Acrid serum in blisters	Extravasated blood
Itching	Present	Absent
Fingertips	Vesicles from scratching	Absent
Chemical test	Juice detectable in blister fluid	Negative

> REMEMBER
>
> *Dhobi's marking ink fakes a bruise: blistered, itchy, dark brown, never changes colour, fingertips also blistered.*

Nux vomica

Common names: Kuchila, Kuchla, Nux vomica, Strychnine tree, Poison nut



Strychnos nux-vomica (kuchila): flat grey button-like seeds with a silky surface; they contain strychnine and brucine
Photo: Wikimedia Commons, Strychnos nux-vomica 001.JPG

SCIENTIFIC NAME	Strychnos nux-vomica, family Loganiaceae. Chief alkaloid strychnine, $C_{21}H_{22}N_2O_2$
CLASS OF POISON	Neurotic: spinal poison (convulsant)
PART POISONOUS	Seeds of the ripe fruit are the poisonous part, with strychnine and brucine about 1.5% each. Bark, wood and leaves carry brucine but no strychnine; fruit pulp has very little. Seeds are flat ash-grey discs about 2 to 2.5 cm wide, covered with silky radiating hairs.
FATAL DOSE	Strychnine 50 to 100 mg, one crushed seed (Reddy); 15 to 50 mg, the alkaloid of one seed (Vij); 1 to 2 crushed seeds (Rao)
FATAL PERIOD	1 to 2 hours (Reddy, Rao); a few hours (Vij)
ACTIVE PRINCIPLE	Strychnine and brucine, with a little loganin (a glycoside). Strychnine is 10 to 20 times more toxic than brucine and intensely bitter.

Mechanism of action

- **Glycine antagonism.** Strychnine competitively blocks glycine, the inhibitory transmitter, at receptors on anterior horn motor neurones of the cord and brainstem.
- **Loss of Renshaw cell inhibition.** With the brake removed, a slight noise, light or draught sets off a violent reflex contraction of all muscles together.
- **Spared consciousness.** The cerebrum is little affected, so the mind stays clear through each convulsion.
- **Asphyxial death.** Diaphragm and chest muscles lock in spasm, breathing stops, and hypoxia paralyses the medulla.

Signs and symptoms

- **Onset.** Swallowed alkaloid acts in 5 to 15 minutes; crushed seeds take an hour or more. Whole seeds pass out unchanged and cause no poisoning.
- **Prodrome.** Bitter taste, restlessness, fear, a sense of suffocation, muscle stiffness and twitching; perception is unusually sharp.
- **Generalised convulsions.** All muscles contract at once, first clonic, then tonic, lasting half to 2 minutes each.
- **Opisthotonos.** The body arches back in hyperextension and rests on the head and heels. Emprosthotonos (forward) and pleurosthotonos (sideways) occur less often.
- **Risus sardonicus.** Facial spasm draws the corners of the mouth back into a fixed grin.
- **Asphyxial signs during a fit.** Cyanosed, anxious face, staring prominent eyes, dilated pupils and bloodstained froth at the mouth.
- **Complete relaxation between fits.** The patient looks well though exhausted; another fit follows in 5 to 15 minutes or on the slightest stimulus.
- **Clear sensorium.** Consciousness is retained, so he suffers severe pain and knows death is near.
- **Fatal course.** Fits come faster and last longer; death usually follows 4 to 5 convulsions. Survivors stop fitting within 12 to 24 hours.

Treatment

- **Dark, quiet room.** Minimal handling, because noise, light or touch triggers convulsions.
- **Control convulsions first.** Diazepam 0.1 to 0.5 mg/kg i.v. slowly, then phenobarbital i.v.
- **Short-acting barbiturates, the classic antidote.** Pentobarbital sodium or sodium amytal 0.3 to 0.6 g i.v. (Reddy, Vij).
- **Refractory fits.** General anaesthesia with non-depolarising neuromuscular blockade (pancuronium, vecuronium) and mechanical ventilation. Succinylcholine is avoided, as muscle breakdown makes hyperkalaemia likely.
- Gastric lavage only after convulsions are controlled, with warm water; passing a tube earlier provokes fits.
- Activated charcoal suspension to adsorb strychnine, then remove it.
- **Airway and oxygen.** Tracheostomy may be needed when chest spasm prevents breathing (Vij).
- **Urine acidification.** Contraindicated: strychnine fits break down muscle (rhabdomyolysis), and acid urine favours myoglobin renal failure. IV fluids keep a good urine output instead.

Medicolegal aspects

- **Accidental poisoning is most common.** Overdose of tonics or prescription errors (strychnine settles at the bottom of an alkaline mixture), quack remedies, and children eating seeds.
- **Suicide is rare because death is so painful.** Homicide is uncommon: the taste is intensely bitter and fits follow quickly (Vij), though Reddy records homicide with alkaloid or powdered seed.
- Rodenticide and for killing stray dogs.
- **Cattle poison and arrow poison.** BNS 325 (formerly IPC 428/429): killing or maiming an animal.
- **Aphrodisiac and love philter.** Misuse leads to accidental poisoning.
- **BNS 123 (formerly IPC 328):** causing hurt by poison; BNS 103 (formerly IPC 302) when used for homicide.
- **Strychnine poisoning vs tetanus.** Strychnine: no injury, sudden onset, all muscles at once, jaw involved late, full relaxation between fits, death in 1 to 2 hours, poison found. Tetanus: injury, gradual onset, lockjaw first, rigidity persists between spasms, fever, death after 24 hours or days.
- **A negative analysis does not exclude strychnine.** A minimal dose may be fully eliminated if the victim survives some hours (Vij).

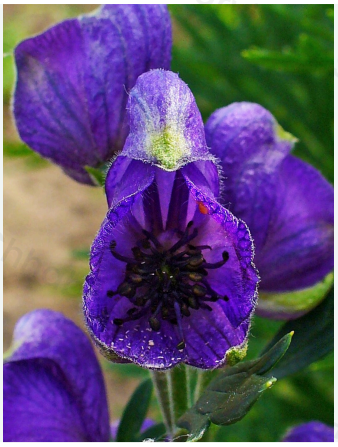
AUTOPSY FINDINGS AND SAMPLES

- **Asphyxial signs.** Findings are otherwise not characteristic.
- Early rigor mortis, not necessarily prolonged (Reddy, Vij); Rao adds that it passes off quickly and notes postmortem caloricity.
- Subperitoneal haemorrhages on the stomach, gastric and duodenal ecchymoses, and seed remains in the stomach. Stretched muscles may rupture.
- Preserve brain and spinal cord in addition to routine viscera, blood and stomach contents.
- **Strychnine resists putrefaction.** It has been recovered from a body exhumed 4 years 9 months after burial. Frog test: the extract injected into a frog's dorsal lymph sac causes tetanic convulsions.

> REMEMBER
>
> *Strychnine: mind clear, muscles fully relaxed between fits. Tetanus: lockjaw first, rigidity persists between spasms.*

Aconite

Common names: Mitha zahar, Mitha bish, Bish, Bikh, Mitha teliya (oil-soaked root), Vatsanabha, Monk's hood, Wolf's bane, Blue rocket



Aconitum napellus (monkshood, bachnag): the helmet-shaped violet flower that gives the plant its name; the root is the poisonous part
Photo: Wikimedia Commons, Aconitum napellus 010.JPG



Aconitum species (monkshood): a spike of hooded violet flowers
Photo: Wikimedia Commons, Torikabuto 01.jpg

SCIENTIFIC NAME	Aconitum ferox (Himalayan, most of the bazaar root) and Aconitum napellus, family Ranunculaceae. Chief alkaloid aconitine, $C_{34}H_{47}NO_{11}$
CLASS OF POISON	Cardiac poison (Reddy, Vij); neurotoxic in Rao
PART POISONOUS	All species and all parts are poisonous: least when young, more as seeds ripen, most in bloom. Root and seeds are the most potent. The dry root is conical, 5 to 10 cm long, dark brown outside; its white cut surface turns pink in air.
FATAL DOSE	Root 1 g; extract 250 mg; aconitine 2 to 5 mg (Reddy), 4 mg (Vij); tincture 2 to 30 mL (Reddy), 20 to 30 mL (Vij)
FATAL PERIOD	2 to 6 hours (Reddy); average 6 hours, range 45 minutes to 24 hours (Vij)
ACTIVE PRINCIPLE	Aconitine, one of the most virulent known poisons, with pseudoaconitine, indaconitine, bikhaconitine, picraconitine, aconine and others. Aconitine is about 1,000 times as toxic as quinine.

Mechanism of action

- **Sodium-channel opener.** Aconitine holds voltage-gated sodium channels open, so nerve and heart cells stay depolarised and fire abnormally.
- **Sensory nerve stimulation, then paralysis.** Endings tingle first and then go numb; this tingling-then-numbness is the hallmark of aconite.
- **Cardiotoxicity.** Vagal stimulation slows the heart, and the direct myocardial action causes ectopics, ventricular tachycardia or fibrillation and AV block.
- **Medullary depression.** The respiratory and cardiac centres are depressed, while the higher cerebral centres are little affected.

Signs and symptoms

- **Onset within minutes.** Burning from mouth to stomach is followed by tingling and numbness of mouth, tongue and pharynx.
- **Paraesthesia spreads over the whole body.** Skin tingles, then goes numb, with reduced sensation.
- **Gastrointestinal features.** Salivation, nausea, vomiting and diarrhoea; later thirst, a dry mouth and inability to swallow.
- **Hippus.** Pupils alternately contract and dilate, and stay dilated late. Vision dims; diplopia and colour patches occur.
- **Muscular weakness and ataxia.** The limbs give way, so he cannot stand or walk; speech slurs, muscles twitch and cramp.
- **Cardiovascular toxicity.** Early tachycardia, then bradycardia from AV block, irregular weak pulse, hypotension and arrhythmia.
- **Autonomic and general signs.** Pallor, profuse sweating, subnormal temperature and oppression in the chest.
- **Clear mind.** Consciousness usually stays intact until death, though hallucinations may occur.
- **Mode of death.** Cardiac arrhythmia or respiratory centre paralysis, or both; convulsions may precede death.

Treatment

- Gastric lavage with warm water, then weak iodine in potassium iodide or tannic acid to precipitate the alkaloid, or potassium permanganate (Vij).
- Activated charcoal in multiple doses, or milk, to delay absorption; then a cathartic.
- Atropine 0.5 to 1 mg (Reddy; 1 mg, Rao). It counters vagal slowing of the heart.
- **Cardiac monitoring with treatment of arrhythmia.** Rao cites 0.1% procaine (novocaine) 50 mL for arrhythmias.
- Artificial respiration and oxygen for respiratory failure.
- **Symptomatic care:** fluids for vomiting and hypotension, warmth for the low temperature.

Medicolegal aspects

- **Accidental poisoning.** Root eaten in mistake for horseradish, quack and ayurvedic remedies, liniments swallowed by mistake, and liquor laced to boost intoxication.
- **Suicide.** Reddy says suicidal cases are frequent; Vij finds accident more common than suicide or homicide.
- **Homicide.** Given with betel leaf (paan) or sweets to mask the taste. BNS 103 (formerly IPC 302); BNS 123 (formerly IPC 328): causing hurt by poison.
- **Ideal homicidal poison (Vij).** Cheap, small fatal dose, quick death, taste easily masked, signs mimic heart disease, and the alkaloid is hard to detect.
- **Arrow poison.** Tribals use the root extensively; the Lepchas of Sikkim are a classic example.
- **Cattle poison.** BNS 325 (formerly IPC 428/429): killing or maiming an animal.
- **Abortifacient.** The root is used to procure abortion. BNS 88 (formerly IPC 312): causing miscarriage.
- **Aconite root vs horseradish.** Aconite: conical, dark brown, cut surface turns pink, sweet then tingling taste. Horseradish: cylindrical, yellowish-white, stays white, pungent.

AUTOPSY FINDINGS AND SAMPLES

- **Asphyxial signs.** Findings are otherwise not characteristic.
- Gastroduodenal mucosa congested, with occasional ecchymoses; pallor of the mouth mucosa may be seen. Root fragments may remain in the stomach.

- Congestion of lungs, kidneys and brain.
- **Aconitine is very unstable and is destroyed by putrefaction.** It is rarely detected once absorbed, so vomit and stomach contents matter most.
- **Vomit mixed with wood ash (an alkali) destroys aconitine.** Hankin advised adding 2 parts rectified spirit to 1 part acetic acid to stop the decomposition (Vij).

> REMEMBER
>
> *Mitha zahar: sweet taste, then tingling and numbness. Cut root turns pink; horseradish stays white.*

Poppy husk

Common names: Doda, Bhukki, Post, Post ka doda, Poppy straw, Poppy husk, Poppy heads



Poppy straw (doda, post): dried, ripe poppy heads, the husk that is crushed and boiled into an opiate drink
Photo: Wikimedia Commons, Poppies-seeds-dry.png

SCIENTIFIC NAME	Papaver somniferum Linn., family Papaveraceae (opium poppy, white poppy)
CLASS OF POISON	Neurotic: cerebral, somniferous (narcotic)
PART POISONOUS	Ripe, dried capsule shells left after harvest, whole or crushed into husk. They contain only a trace of opium. The fresh latex of the unripe capsule is the most toxic part of the plant.
FATAL DOSE	Not fixed in the textbooks. Depends on morphine extracted; morphine 200 mg is fatal in a non-addict.
FATAL PERIOD	6 to 12 hours (Reddy); a few hours to 2 days (Vij), as for opium
ACTIVE PRINCIPLE	Traces of the opium alkaloids, chiefly morphine, with codeine, thebaine, narcotine (noscapine) and papaverine. Content is low and varies with harvest.

Mechanism of action

- **Opioid receptor agonist.** The small dose of morphine in the husk acts on mu receptors, so the user feels sedated and pain-free.
- **Tolerance and dependence.** Daily use of the decoction needs ever larger amounts, so the user slides into opioid dependence.
- **Respiratory centre depression.** A heavy dose, or husk mixed with alcohol or sedatives, slows breathing until asphyxia follows.

Signs and symptoms

- **Route of use.** Husk is boiled into a decoction and drunk, or chewed; addicts take it daily.
- **Acute intoxication.** Drowsiness, euphoria and itching, with constricted pupils and slow breathing.
- **Overdose.** Coma, pinpoint pupils and respiratory depression: the same triad as opium.

- **Chronic use (opioid dependence).** Constipation, anorexia, weight loss, disturbed sleep and loss of interest in work.
- **Reduced libido and impotence.** Opioids lower luteinising hormone and testosterone.
- **Opioid withdrawal syndrome (abstinence, 'cold turkey').** Yawning, lacrimation, rhinorrhoea, mydriasis, piloerection, sweating, cramps and diarrhoea. It peaks at 36 to 72 hours and settles in 5 to 10 days.
- **Behavioural change.** Poor attendance at work, irritability and borrowing or stealing money for the drug.

Treatment

- **Acute overdose.** Airway, oxygen and ventilation come first, then naloxone IV, titrated to breathing.
- **Naloxone dose.** Current practice: 0.4 mg IV (0.04 to 0.1 mg in an opioid-dependent patient), repeated every 2 to 3 minutes up to 10 mg. Vij gives 0.4 to 2 mg every 10 to 15 minutes. Titrate to breathing, not to full wakefulness, so withdrawal is not provoked.
- **Gastric decontamination.** Activated charcoal binds any alkaloid left in the stomach. Permanganate lavage is obsolete, as permanganate is caustic.
- **Supportive care.** IV fluids, warmth, and antibiotics if aspiration is suspected.
- **De-addiction.** Gradual withdrawal of the drug under supervision, with good nursing and nutrition.
- **Substitution therapy.** Methadone 30 to 40 mg a day, tapered gradually, covers the withdrawal state.
- **Symptom control.** Propranolol eases anxiety and craving; clonidine eases watering eyes, runny nose, sweating and diarrhoea (Rao).
- **Psychiatric counselling.** Relapse is common, so counselling continues after the acute phase.

Medicolegal aspects

- **Poppy straw under NDPS Act 1985.** All parts of the poppy plant after harvest, except the seeds, whether whole, cut, crushed or powdered, and whether or not juice has been extracted.
- **NDPS offences.** Producing, possessing, transporting, selling or using poppy straw without licence is punishable; the sentence rises with small, intermediate and commercial quantity.
- **Regional addiction.** An infusion of poppy capsules is drunk in certain districts of Punjab and parts of Rajasthan (Vij); doda-post use is widespread in North India.
- **Accidental poisoning.** Overdose occurs in addicts, especially when husk is combined with alcohol or sedatives.
- **Drugging of children.** Labour-class parents have given poppy decoctions to keep infants asleep; overdose can kill.
- **Cause of death.** Analysis alone does not settle it; tolerance cannot be measured at autopsy, so scene, history and autopsy are read together (Vij).

AUTOPSY FINDINGS AND SAMPLES

- **Asphyxial picture in fatal overdose.** Cyanosis, froth at mouth and nostrils, congested oedematous lungs.
- **Stomach contents.** Brownish decoction or husk fragments may be found; keep them for analysis.
- **Viscera for analysis.** Blood, urine, bile and brain with routine viscera; morphine is the marker detected.
- **Signs of chronic use.** Emaciation and poor nutrition; no needle marks, since husk is taken by mouth.

> REMEMBER
>
> *Poppy straw under NDPS means every part of the harvested poppy except the seeds.*

Poppy capsule

Common names: Green poppy capsule, Doda, Post, Poppy head



Unripe Papaver somniferum capsule, lanced: milky latex seeps from the cut and dries into raw opium
Photo: Wikimedia Commons, Opium pod cut to demonstrate fluid extraction1.jpg

SCIENTIFIC NAME	Papaver somniferum Linn., family Papaveraceae (opium poppy, white poppy)
CLASS OF POISON	Neurotic: cerebral, somniferous (narcotic)
PART POISONOUS	Unripe green capsule. Its milky latex, exuding on incision, is the source of opium and the most toxic part. Ripe dry capsules hold only a trace; seeds are harmless.
FATAL DOSE	As for opium: about 2 g opium, or morphine 200 mg, in a non-addict
FATAL PERIOD	6 to 12 hours (Reddy); a few hours to 2 days (Vij)
ACTIVE PRINCIPLE	About 25 alkaloids bound to meconic, lactic and sulphuric acids. Phenanthrene group (narcotic): morphine about 10%, codeine about 0.5%, thebaine about 0.3%. Benzylisoquinoline group (non-narcotic): narcotine (noscapine) about 6%, papaverine about 1%, narceine. The latex of the unripe capsule carries the full alkaloid load.

Mechanism of action

- **Latex as opium source.** Vertical cuts on the unripe capsule release milky latex; dried, it becomes opium.
- **Opioid receptor agonist.** Morphine in the latex acts on mu receptors, giving analgesia, sleep and euphoria.
- **Respiratory centre depression.** The brainstem loses its response to carbon dioxide, so breathing slows and fails.
- **Thebaine.** A minor alkaloid that acts as a convulsant, resembling strychnine (Rao).

Signs and symptoms

- **Route.** Juice of green capsules is eaten in a sweet halwa (Vij); capsules are also boiled and the decoction drunk.
- **Stage of excitement.** Brief euphoria and restlessness; children may convulse.
- **Stage of stupor.** Giddiness, heaviness of limbs, itching and drowsiness; the patient can still be roused.
- **Stage of narcosis.** Deep coma, flaccid muscles, absent reflexes.
- **Miosis.** Pinpoint pupils, fixed to light, dilating terminally with asphyxia.
- **Respiratory depression.** Slow, sighing breathing down to 2 to 4 per minute, often Cheyne-Stokes.
- **Cyanosis, cold sweat and hypothermia.** Face blue, skin cold and wet, temperature subnormal.
- **Bradycardia and hypotension.** Slow full pulse, low blood pressure.
- **Diagnostic triad.** Coma, pinpoint pupils and respiratory depression.

Treatment

- **Airway and breathing first.** Clear the airway, give oxygen and intubate if breathing is shallow; artificial ventilation is the life-saving step in deep coma.
- **Naloxone (specific opioid antagonist).** Current practice: 0.4 mg IV (0.04 to 0.1 mg in an opioid-dependent patient), repeated every 2 to 3 minutes up to 10 mg. Vij gives 0.4 to 2 mg every 10 to 15 minutes. Titrate to breathing, not to full wakefulness, so withdrawal is not provoked.
- **Naloxone infusion.** Its action is shorter than morphine's, so coma can return; Reddy advises 0.4 to 0.8 mg per hour for up to 48 hours.
- **Gastric decontamination.** Activated charcoal binds the alkaloid; morphine is re-excreted into the stomach, so it helps even late. Permanganate lavage is obsolete, as permanganate is caustic.
- **Purgation.** Sodium sulphate 30 g (Reddy) or magnesium sulphate 15 g (Vij) clears the bowel.
- **Coma cocktail.** When the poison is unknown, 50% glucose, thiamine 100 mg and naloxone are given IV; naloxone starts at 0.4 mg and is titrated (Reddy gives 2 mg).
- **Supportive care.** IV fluids, correction of electrolytes, warmth, and antibiotics against aspiration pneumonia.
- **Atropine is not recommended.** It adds to the paralysis and does not reverse opium coma (Reddy). Nalorphine and levallorphan are also avoided.

Medicolegal aspects

- **Licensed cultivation.** Poppy is grown in India only under government licence; NDPS Act 1985 controls cultivation.
- **Poppy straw under NDPS Act 1985.** Harvested capsules, whole or crushed, count as poppy straw; seeds are excluded.
- **Opium under NDPS Act 1985.** The coagulated juice of the opium poppy is itself opium, a narcotic drug.
- **Addiction.** Capsule infusion is drunk in parts of Punjab and Rajasthan; a halwa from green-capsule juice is also eaten (Vij).
- **Accidental poisoning.** Children given capsule decoctions to keep them quiet may die of overdose.
- **BNS 123 (formerly IPC 328).** Causing hurt by poison, used when the decoction is given to stupefy someone.

AUTOPSY FINDINGS AND SAMPLES

- **Asphyxial picture.** Cyanosed face and nails, froth at mouth and nose, dark intense lividity.
- **Lungs.** Congested and oedematous; trachea and bronchi covered with froth.
- **Stomach.** Capsule fragments, halwa or decoction may be present; smell of opium fades with putrefaction.

- **Viscera for analysis.** Blood, urine, bile and brain with routine viscera; liver and kidney if urine or bile are not available.

> REMEMBER
>
> *Unripe capsule gives the latex (opium); ripe dry capsule holds only a trace.*

Poppy seeds

Common names: Khus khus, Khaskhas, Posto, Poppy seed



Poppy seeds (khus khus): tiny kidney-shaped seeds with a pitted coat; they carry only traces of alkaloid
Photo: Wikimedia Commons, Poppy seeds.jpg

SCIENTIFIC NAME	Papaver somniferum Linn., family Papaveraceae (opium poppy, white poppy)
CLASS OF POISON	Non-poisonous food item from the opium poppy (Neurotic: somniferous plant)
PART POISONOUS	Seeds are not poisonous. They contain no opium (Vij) and are harmless and nutritive (Reddy). The latex of the unripe capsule is the toxic part of the plant.
FATAL DOSE	Not poisonous in ordinary use; no fatal dose recorded
FATAL PERIOD	Not applicable
ACTIVE PRINCIPLE	No narcotic alkaloid of practical importance. Seeds yield a bland edible oil (khas khas ka tel) used for cooking and lighting.

Mechanism of action

- **No opioid action.** The seeds lack the latex that carries morphine, so they produce no narcotic effect.
- **Demulcent and nutritive.** Reddy describes them as soothing and nourishing, which is why they are eaten.

Signs and symptoms

- **No toxic effect in ordinary use.** Seeds eaten in curries, sweets and breads cause no narcosis.
- **No miosis or respiratory depression.** Normal pupils and breathing are expected after eating seeds.
- **Differential diagnosis.** A patient who blames 'khus khus' but shows coma, pinpoint pupils and slow breathing has taken opium, capsule or husk.
- **Clinical history.** Ask about doda, post or afeem; the opioid triad points to those, not to the seeds.
- **Appearance.** White (Reddy) or creamy (Vij) kidney-shaped seeds, sprinkled over sweets.
- **Oil.** Poppy seed oil is bland and used in cooking; it causes no poisoning.

Treatment

- **No specific treatment.** Seeds alone need none.
- **Search for another cause.** Signs of opioid poisoning mean another source of opium, which must be found.
- **If the opioid triad is present.** Treat as opium poisoning: airway, oxygen, naloxone IV titrated to breathing, and activated charcoal.
- **Supportive observation.** Monitor breathing and pupils until the true cause is clear.
- **Sample retention.** Keep vomitus or gastric aspirate; analysis separates seeds from opium.

Medicolegal aspects

- **Excluded from poppy straw.** The NDPS Act 1985 defines poppy straw as all parts of the harvested poppy except the seeds.
- **Legal food.** Seeds are sold and eaten freely in India as a condiment (Rao).
- **False defence.** A claim of 'only khus khus' does not explain opioid poisoning; seeds contain no opium.
- **Screening caution.** Urine opiate screening tests may turn faintly positive after a heavy seed meal; confirm with history and laboratory methods.
- **Teaching point.** Of the whole poppy plant, only the seeds are harmless.

AUTOPSY FINDINGS AND SAMPLES

- **No findings attributable to seeds.** Seeds in the stomach are a food residue only.
- **Look further.** Asphyxial signs with opium lumps or capsule fragments point to opium, not seeds.
- **Viscera for analysis.** Preserve routine viscera with blood and bile when opium is suspected.

> REMEMBER
>
> *Khus khus is harmless food: the one poppy part outside NDPS 'poppy straw'.*

Crude opium

Common names: Afeem, Afim, Opium, Kasoomba (decoction), Madak, Chandu (smoking preparations)



Crude opium (afeem): dark brown, sticky cakes of dried poppy latex, here seized blocks beside a lighter for scale
Photo: Wikimedia Commons, Black tar (raw) opium seized in Afghanistan.jpg

SCIENTIFIC NAME	Papaver somniferum Linn., family Papaveraceae (opium poppy, white poppy). Crude opium is the air-dried latex of the unripe capsule.
CLASS OF POISON	Neurotic: cerebral, somniferous (narcotic)
PART POISONOUS	Dried latex (opium) collected from vertical incisions on the unripe capsule. This is the most toxic product of the plant. Ripe capsules hold a trace; seeds are harmless.
FATAL DOSE	Opium 2 g (Reddy, Vij); 200 to 900 mg (Rao). Morphine 200 mg
FATAL PERIOD	6 to 12 hours (Reddy); a few hours to 2 days (Vij)
ACTIVE PRINCIPLE	About 25 alkaloids bound to meconic, lactic and sulphuric acids. Phenanthrene group (narcotic): morphine about 10%, codeine about 0.5%, thebaine about 0.3%. Benzylisoquinoline group (non-narcotic): narcotine (noscapine) about 6%, papaverine about 1%, narceine. Indian opium is standardised to about 10% morphine at the Government Opium Factory, Ghazipur (Vij).

Mechanism of action

- **Opioid receptor agonist.** Morphine binds mu, kappa and delta receptors in the brain and spinal cord, the sites used by the body's own endorphins.
- **Central nervous system depression.** Opium depresses all centres except the oculomotor, vomiting and sweating centres, so pupils shrink and sweat pours while breathing fails.
- **Respiratory centre depression.** The brainstem stops responding to rising carbon dioxide, so breathing slows to 2 to 4 per minute and death comes from asphyxia.
- **Gut dysmotility.** Peristalsis slows and the stomach empties late; absorbed morphine is also re-excreted into the stomach.

Signs and symptoms

- **Onset.** Symptoms start within half an hour to one hour by mouth; within 3 to 4 minutes after injection.
- **Stage of excitement.** Short-lived euphoria, talkativeness and restlessness; it may be absent after a large dose. Children tend to convulse in this stage.
- **Stage of stupor.** Headache, giddiness, heaviness of the limbs, itching of the skin and an intense urge to sleep. The patient can still be roused.
- **Stage of narcosis (coma).** Deep coma from which the patient cannot be roused; muscles flaccid and reflexes lost.
- **Miosis.** Pinpoint pupils that do not react to light. They dilate terminally once asphyxia sets in.
- **Respiratory depression.** Slow, stertorous breathing, falling to 2 to 4 per minute, often Cheyne-Stokes in type.
- **Cyanosis with cold, sweating skin.** Face and lips turn blue; temperature is subnormal and the skin is bathed in sweat.
- **Bradycardia and hypotension.** Pulse slow and full, blood pressure low. Breath may smell of opium.
- **Non-cardiogenic pulmonary oedema.** Fluid fills the lungs; Rao notes it in virtually all fatal cases.
- **Diagnostic triad.** Coma, pinpoint pupils and respiratory depression together point to opioid poisoning.

Treatment

- **Airway and breathing first.** Clear the airway, give oxygen and intubate if breathing is shallow; artificial ventilation is the life-saving step in deep coma.
- **Naloxone (specific opioid antagonist).** Current practice: 0.4 mg IV (0.04 to 0.1 mg in an opioid-dependent patient), repeated every 2 to 3 minutes up to 10 mg. Vij gives 0.4 to 2 mg every 10 to 15 minutes. Titrate to breathing, not to full wakefulness, so withdrawal is not provoked.
- **Naloxone infusion.** Its action is shorter than morphine's, so coma can return; Reddy advises 0.4 to 0.8 mg per hour for up to 48 hours.
- **Gastric decontamination.** Activated charcoal binds the alkaloid; morphine is re-excreted into the stomach, so it helps even late. Permanganate lavage is obsolete, as permanganate is caustic.
- **Purgation.** Sodium sulphate 30 g (Reddy) or magnesium sulphate 15 g (Vij) clears the bowel.
- **Coma cocktail.** When the poison is unknown, 50% glucose, thiamine 100 mg and naloxone are given IV; naloxone starts at 0.4 mg and is titrated (Reddy gives 2 mg).
- **Supportive care.** IV fluids, correction of electrolytes, warmth, and antibiotics against aspiration pneumonia.
- **Atropine is not recommended.** It adds to the paralysis and does not reverse opium coma (Reddy). Nalorphine and levallorphan are also avoided.

Medicolegal aspects

- **Suicide.** Opium is chosen because death is painless; mixed with mustard or ginger oil it acts fast and resists lavage (Vij).
- **Homicide is rare.** The dark brown colour, characteristic smell and bitter taste give it away.
- **Infanticide.** Used to kill illegitimate infants; a mother has smeared tincture of opium on her nipple before breastfeeding. Charge under BNS 103 (formerly IPC 302): murder.
- **Accidental poisoning.** Children swallow opium pills meant for elders, or are drugged to sleep by parents; infants are poisoned through breast milk.
- **Addiction (morphinism).** Taken as a supposed aphrodisiac, to steady the nerves before a bold act, and as kasoomba at festivals; smoked as madak or chandu. Addicts tolerate 3 to 6 g a day.
- **NDPS Act 1985.** Opium is a narcotic drug; production, possession, sale, transport or use without licence is punishable by quantity.

- **Body packers.** Smugglers swallow sealed packets of heroin or morphine; a burst packet causes fatal overdose, an intact one bowel obstruction.
- **Other uses.** Doping of race horses; rarely a cattle poison (Reddy).

AUTOPSY FINDINGS AND SAMPLES

- **Asphyxial picture.** Not specific: deeply cyanosed face and nails, froth at mouth and nostrils, intense dark lividity.
- **Lungs.** Congested and oedematous with subpleural petechiae; trachea and bronchi full of froth. Vij calls lung oedema with near-black facial lividity the hallmark.
- **Stomach.** Small, soft brownish lumps of opium may be found; the smell of opium on opening the body fades once putrefaction starts.
- **Viscera for analysis.** Blood, urine, bile and brain with routine viscera; keep liver and kidney when urine or bile is not available. Morphine may be found in liver or bile when blood and urine are negative.
- **Field test.** Marquis reagent (sulphuric acid with formalin) turns purple-red, then violet, then blue with morphine.

> REMEMBER
>
> *Coma, pinpoint pupils and respiratory depression: the triad of opium poisoning.*

Tobacco

Common names: Tobacco, Tambaku, Khaini, Bidi, Snuff



Nicotiana tabacum (tobacco): large, broad, simple leaves and pink trumpet-shaped flowers; the leaves carry nicotine
Photo: Wikimedia Commons, P1000485 Nicotiana tabacum (tobacco) (Solanaceae) Plant.JPG

SCIENTIFIC NAME	Nicotiana tabacum, family Solanaceae. Chief alkaloid nicotine, $C_{10}H_{14}N_2$
CLASS OF POISON	Cardiac poison (Reddy, Vij)
PART POISONOUS	All parts are poisonous except the ripe seeds. Dried leaves (tambaku) carry 1 to 8% nicotine and are the usual source; Rao names dried leaves and stems.
FATAL DOSE	Nicotine 50 to 100 mg, or 15 to 30 g crude tobacco (Reddy); nicotine 40 to 60 mg, about 20 g tobacco (Vij); 60 mg nicotine or 2 g tobacco (Rao)
FATAL PERIOD	5 to 15 minutes (Reddy); a few minutes to a few hours (Rao, Vij)
ACTIVE PRINCIPLE	Nicotine and anabasine (equally toxic), with nornicotine (less toxic). Nicotine is a colourless, volatile, bitter liquid alkaloid that browns in air and light.

Mechanism of action

- **Nicotinic agonist.** Nicotine binds nicotinic acetylcholine receptors in autonomic ganglia, the neuromuscular junction, adrenal medulla and brain.
- **Biphasic action.** Ganglia and muscle end-plates are first stimulated, then blocked by persistent depolarisation, so early excitement gives way to paralysis.
- **Catecholamine release.** Adrenal and central stimulation release adrenaline, vasopressin and other hormones, which raise pulse and blood pressure early.
- **Rapid absorption from every surface.** Lungs, mucous membranes, gut and intact skin all take up nicotine, so a poultice or wet leaves can poison.

Signs and symptoms

- **Gastrointestinal stage.** Burning in mouth and throat, hypersalivation, nausea, vomiting and abdominal pain.

- **Early cholinergic excess.** Miosis, sweating, lacrimation, bronchorrhoea, fasciculation, urination and defecation.
- **Early cardiorespiratory stimulation.** Tachycardia, hypertension and tachypnoea (Reddy); Vij describes hypertension with bradycardia.
- **Early CNS excitement.** Headache, confusion, dizziness, ataxia, agitation and tremor.
- **Late ganglionic blockade.** Bradycardia, hypotension, arrhythmia and collapse.
- **Late neuromuscular paralysis.** Weakness spreads to the respiratory muscles, and breathing fails.
- **Late CNS depression.** Mydriasis, lethargy, convulsions and coma.
- **Green tobacco sickness.** Workers handling wet tobacco leaves absorb nicotine through the skin and develop nausea, vomiting, giddiness and weakness.
- **Chronic poisoning.** Cough, bronchitis, amblyopia, tobacco heart (extrasystoles, angina-like pain) and thromboangiitis obliterans (Rao).

Treatment

- **Skin decontamination.** Remove soaked clothing and leaves, and wash the skin, because absorption continues through intact skin.
- Gastric lavage quickly, as absorption is fast, with warm water and charcoal.
- Activated charcoal, then a purge and colonic wash-out (Reddy).
- Airway, oxygen and ventilation for respiratory muscle paralysis.
- Atropine 1 to 2 mg i.m. for muscarinic features (Reddy; 1.5 mg, Rao). It works best in the early or moderate stage.
- **Mecamylamine, the specific nicotinic antagonist.** It comes only as oral tablets, which is unsuitable in a vomiting, hypotensive or convulsing patient (Vij).
- **Supportive care.** Treat convulsions and hypotension. Nicotine clears in about 16 hours, so a patient kept alive that long usually recovers.

Medicolegal aspects

- **Accidental poisoning is the usual form.** Children eat cigarettes or bidis; leaves or juice applied to wounds or as an enema; nicotine gum overused.
- **Occupational poisoning.** Tobacco harvesters (green tobacco sickness), processing workers and insecticide sprayers absorb nicotine through the skin.
- **Nicotine as insecticide.** It is the oldest known insecticide and is used for fumigation and spraying; such products fall under the Insecticides Act 1968.
- **Malingering.** Leaves soaked in water are bandaged into the axillae overnight; vomiting and weakness appear by morning, to dodge duty.
- **Suicide and homicide are rare.** Vij records homicide with tobacco or snuff mixed in beer or other drinks. BNS 123 (formerly IPC 328): causing hurt by poison.
- **COTPA 2003 (Cigarettes and Other Tobacco Products Act).** Section 4 bans smoking in public places; Section 6 bans sale to persons under 18.

AUTOPSY FINDINGS AND SAMPLES

- Asphyxial signs, with brownish froth at the mouth and nostrils.
- Haemorrhagic congestion of the gastrointestinal tract and pulmonary oedema.
- Stomach may hold leaf fragments or smell of tobacco.
- Nicotine resists putrefaction (Vij), so late analysis can still succeed. Preserve stomach contents, liver, kidney, blood and urine.

REMEMBER

Nicotine rivals cyanide in speed: death in 5 to 15 minutes. Survive 16 hours and recovery is likely.

Cannabis

Common names: Indian hemp, Bhang, Siddhi, Sabji, Patti, Ganja, Charas, Hashish, Majoon (Majun), Marijuana, Pot, Grass, Weed



Cannabis sativa leaf: palmate, with 5 to 9 narrow, saw-edged leaflets radiating from one point
Photo: Wikimedia Commons, Marijuana.jpg



Charas (hashish): resin rubbed from the flowering tops, pressed into a dark, slightly glossy lump
Photo: Wikimedia Commons, Pure Manali hashish (1 tolla = 10 g) (4142310413).jpg

SCIENTIFIC NAME	Cannabis sativa (var. indica), family Cannabaceae. Main psychoactive principle delta-9-tetrahydrocannabinol (THC), $C_{21}H_{30}O_2$
CLASS OF POISON	Neurotic: cerebral, deliriant (Reddy, Rao); hallucinogen (Vij)
PART POISONOUS	Active resin occurs in all parts except stem, root and seeds (Reddy); the female flowering tops are richest. Bhang: dried leaves and fruiting shoots. Ganja: flowering tops of the female plant (Reddy), or its resinous leaves and bracts (Vij). Charas: resin exuding from leaves and stems (Reddy, Vij), or from the flower tops (Rao). Majoon: a sweet made from bhang. Marijuana: dried leaves, flowering tops, stems and seeds.
FATAL DOSE	Charas 2 g, ganja 8 g, bhang 10 g per kg body weight (Reddy, Vij); THC 30 mg/kg (Reddy)
FATAL PERIOD	Several days (Reddy); 12 to 24 hours (Vij)
ACTIVE PRINCIPLE	Cannabinoids in the resin, chiefly THC. Cannabinol is weakly active and cannabidiol non-intoxicating. Heating decarboxylates THC-acid in the resin into active THC.

Mechanism of action

- **Cannabinoid (CB1) receptor agonist.** THC acts on receptors in brain areas for pain, memory, reward and motor co-ordination.
- **High receptor density in basal ganglia and cerebellum.** This explains the impaired co-ordination and the risk of road accidents.
- **Intermediate density in cortex and hippocampus.** This explains loss of short-term memory and altered thought.

- **Low density in the brainstem.** Cardiac and respiratory centres are spared, which is why death from cannabis alone is so rare.

Signs and symptoms

- **Onset.** Effects start soon after smoking and last 1 to 2 hours; after swallowing they start in about half an hour and last 2 to 3 hours.
- **Euphoria.** Cheerfulness, laughter, talkativeness and disinhibition, often with a feeling of cleverness.
- **Perceptual distortion.** Altered sense of time and space, depersonalisation, illusions and hallucinations, often sexual.
- **Cognitive impairment.** Poor short-term memory, attention and judgement, with slowed reaction time.
- **Conjunctival injection and tachycardia.** These are the commonest physical signs.
- Increased appetite and thirst, dry mouth and urinary frequency.
- Ataxia, tremor, tingling and numbness of the limbs, with pupils dilated (Vij) or constricted at high dose (Rao).
- **Acute anxiety and paranoia.** Panic and fear of dying may occur, especially in those with prior mental illness.
- **Stage of narcosis.** Drowsiness and deep sleep follow; recovery is the rule, and death from respiratory failure is very rare.
- **Chronic use.** Hashish (hemp) insanity, amotivational syndrome, gynaecomastia, low testosterone and sperm count, and moral deterioration.

Treatment

- **Reassurance in a quiet room.** Most patients settle once told they will recover.
- **Activated charcoal if a large amount was swallowed recently.** Lavage is rarely needed; emetics are contraindicated, as a drowsy patient may aspirate.
- **Coma regimen if unconscious.** 100 mL of 50% glucose, naloxone 2 mg and thiamine 100 mg i.v. (Reddy).
- Diazepam 5 to 10 mg i.v. for violence or aggression.
- Anxiolytics, and haloperidol if needed, for flashbacks or psychosis.
- Supportive care after intravenous injection of crude extract, which causes vomiting, diarrhoea and hypotension.
- Psychotherapy and de-addiction care for chronic use.

Medicolegal aspects

- **Overindulgence causes most poisoning.** Accidental ingestion or inhalation also occurs.
- **Stupefying poison.** Road poisoners use majoon and charas to stupefy victims before robbery; dhatura is sometimes mixed into majoon. BNS 123 (formerly IPC 328).
- **Steadying the nerves.** Criminals take it before a violent act, a practice also noted among soldiers before British rule.
- **Run amok.** A cannabis psychosis in which the person, after brooding, kills an enemy (real or imagined) and then anyone in his way, finally killing himself or surrendering.
- **Criminal responsibility.** Voluntary intoxication is no defence; intent and knowledge are judged as if sober: BNS 24 (formerly IPC 86).
- **Driving impairment.** Cannabis damages motor skills and judgement and causes road accidents.
- **NDPS Act 1985.** Cannabis (hemp) is a narcotic drug; Section 2(iii) covers charas and ganja but not bhang (leaves). Section 20 punishes cultivating, possessing or selling cannabis. Cultivation needs a licence.

- **Social use.** Sadhus and fakirs take it to dull hunger and thirst and aid meditation; it is used as an aphrodisiac.

AUTOPSY FINDINGS AND SAMPLES

- **Asphyxial picture.** Findings are not characteristic.
- **Death is usually indirect:** inhaled vomit, road accidents, drowning or injury under intoxication.
- **Preserve blood, urine and stomach contents.** Urine is screened for 11-nor-carboxy-THC.
- Urinary metabolites persist 2 to 3 days after casual use and up to 4 weeks in heavy users. A positive urine shows exposure at an uncertain time, not intoxication at death.

> REMEMBER
>
> *Bhang from leaves, ganja from female flowering tops, charas from resin; potency rises in that order.*

Cerbera thevetia

Common names: Yellow oleander, Pila kaner, Peela kaner, Exile, Bastard oleander, Lucky nut, Manjal arali



Yellow oleander (Thevetia peruviana, pila kaner) fruit and seed: the green drupe holds a hard nut; the kernel carries the cardiac glycosides thevetin and thevetoxin

Photo: Wikimedia Commons, Thevetia peruviana - Fruits.jpg



Yellow oleander nuts: triangular, ridged stones split along one seam, each with up to four seeds; one or two crushed kernels can kill

Photo: Wikimedia Commons, Thevetia peruviana MHNT.BOT.2007.27.24.jpg

SCIENTIFIC NAME	Cerbera thevetia, also called Thevetia peruviana (current name Cascabela thevetia); family Apocynaceae
CLASS OF POISON	Cardiac poison (vegetable cardiac glycoside)
PART POISONOUS	All parts are poisonous, and a milky juice exudes from every part. The seeds (kernels) are the most toxic: one triangular, deeply grooved nut holds 2 to 4 pale yellow seeds (Reddy says five). Root and leaves are also used.
FATAL DOSE	8 to 10 seeds; 15 to 20 g of root; 5 to 10 leaves (Reddy). 8 to 10 seeds or 15 to 20 g of root (Vij).
FATAL PERIOD	2 to 3 hours (Reddy, Vij); death often comes later, over 1 to 3 days, from arrhythmia.
ACTIVE PRINCIPLE	Cardiac glycosides: thevetin (thevetin A and B), thevetoxin, nerifolin, peruvoside, ruvoside and cerberin. Thevetin is about one-eighth as potent as ouabain and acts like digitalis. Seeds hold about 4 percent glycoside (Reddy).

Mechanism of action

- **Na+/K+-ATPase inhibition.** The glycosides block the sodium pump on the heart muscle cell membrane.
- **Raised intracellular calcium.** Sodium builds up inside the cell, so less calcium leaves it; the heart becomes irritable and ectopic beats appear.
- **Vagal (parasympathetic) stimulation and slowed AV conduction.** The heart slows and impulses fail to pass from atria to ventricles, which gives heart block.
- **Hyperkalaemia.** With the pump blocked, potassium stays outside the cells and the serum potassium climbs; a high level marks severe poisoning.

Signs and symptoms

- **Local irritation.** Chewing the seed or bark gives burning, numbness and a feeling of heat in the mouth, with tingling of the tongue and a dry throat.
- **Gastro-intestinal upset.** Vomiting, abdominal pain and diarrhoea (purging) start early.
- **Neurological effects.** Headache, giddiness, dilated pupils, loss of muscle power and fainting.
- **Sinus bradycardia.** The pulse becomes slow; later it is weak and irregular.
- **Heart block (AV block).** First, second or complete block is the typical yellow oleander rhythm on ECG.
- **Other digitalis-like arrhythmias.** Sinoatrial block, atrial and ventricular ectopics, ventricular tachycardia or fibrillation.
- **Hypotension and hyperkalaemia.** Blood pressure falls and serum potassium rises.
- **Terminal collapse.** Death from peripheral circulatory failure or a fatal arrhythmia.

Treatment

- **Gastric lavage (stomach wash).** Both Reddy and Vij advise it early, while seed fragments are still in the stomach.
- **Activated charcoal in multiple doses.** Repeated doses bind glycoside that recirculates through the bile and gut.
- Atropine sulphate 0.6 to 1 mg i.v., repeated as needed. It counters the vagal slowing; the aim is a heart rate above 50 per minute.
- **Old regimen in the books.** Sodium molar lactate with glucose, 1 mg atropine, 2 mL adrenaline and 2 mg noradrenaline as an infusion (Reddy; Vij gives lactate, glucose and atropine).
- **Anti-digoxin Fab fragments (digoxin-specific antibody).** These bind the glycoside; Rao records 200 to 480 mg i.v. reversing life-threatening oleander toxicity.
- **Temporary cardiac pacing.** A transvenous pacemaker is used for complete heart block or bradycardia that atropine does not reverse; isoprenaline helps meanwhile (Rao).
- **Correction of hyperkalaemia and continuous ECG monitoring.** Calcium salts are avoided, because calcium adds to the glycoside effect on the heart.
- **Supportive care.** Fluids, correction of electrolytes and antiarrhythmics such as lignocaine for ventricular irritability.

Medicolegal aspects

- **Suicidal poison.** Yellow oleander seeds are a very common means of suicide in South India and Sri Lanka; Vij notes pockets of rural West Bengal.
- **Homicide uncommon.** The bitter taste and the need for a large dose make it a poor homicidal poison.
- **Accidental poisoning.** Children eat the seeds or fruit, and adults mistake the fruit for unripe mango (Rao).
- **Abortifacient.** Root and seeds are taken for criminal abortion; a twig is also used as an abortion stick. BNS 88 (formerly IPC 312): causing miscarriage.
- **Cattle poison.** Crushed seeds are fed to the animal with corn or bread. BNS 325 (formerly IPC 428/429): killing or maiming an animal.
- **BNS 123 (formerly IPC 328):** causing hurt by means of poison; BNS 103 (formerly IPC 302) if death results by homicide.
- **Exhumation value.** As the poison resists putrefaction, a late exhumation can still yield it.

AUTOPSY FINDINGS AND SAMPLES

- **Non-specific findings.** Congestion of the viscera and generalised venous engorgement.

- **Gastro-intestinal irritation.** Stomach and duodenum are congested and may hold fragments of seeds.
- **Subendocardial haemorrhage (ecchymoses).** Bleeding under the inner lining of the heart (Vij, Rao).
- **Resists putrefaction.** Yellow oleander can be detected long after death and even on exhumation.
- **Viscera preservation.** Stomach with contents, small intestine, liver, kidney and blood go in saturated saline for chemical analysis; seed fragments are kept separately.

> REMEMBER
>
> *Seed inside a triangular grooved nut. Thevetin is one-eighth as potent as ouabain.*

Nerium oleander

Common names: White oleander, Pink oleander, True oleander, Kaner, Safed kaner, Lal kaner, Karabi, Karavira, Arali



Nerium oleander (white or pink kaner): five-petalled pink flowers in clusters with long, leathery, lance-shaped leaves; all parts, root included, hold oleandrin

Photo: Wikimedia Commons, Nerium oleander flowers leaves.jpg

SCIENTIFIC NAME	Nerium oleander, synonyms Nerium odorum and Nerium indicum; family Apocynaceae
CLASS OF POISON	Cardiac poison (vegetable cardiac glycoside)
PART POISONOUS	Vij and Rao call all parts poisonous, including the nectar, which yields poisonous honey. The root is the part used for suicide, abortion and cattle poisoning; leaves are also toxic. Smoke from the burning plant is toxic.
FATAL DOSE	15 to 20 g of root; 5 to 15 leaves (Reddy). 15 to 20 g of root (Vij).
FATAL PERIOD	20 to 36 hours (Reddy); 24 to 36 hours (Vij, Rao).
ACTIVE PRINCIPLE	Cardiac glycosides. Reddy names oleandrin (oleandroside), nerin (nerioside), folinerin and rosagenin; Vij names neriodorin, neriodorein and karabin.

Mechanism of action

- **Na+/K+-ATPase inhibition.** Oleandrin and neriodorin act like digitalis on the heart muscle and cause death from cardiac failure.
- **Digitalis-like conduction block.** Slowed AV conduction and vagal effect give bradycardia, then AV block and fibrillation.
- **Picrotoxin-like (strychnine-like) spinal action.** Neriodorein and karabin cause muscle twitching and tetanic spasms (Vij).
- **Hyperkalaemia.** Blocking the sodium pump raises serum potassium in severe poisoning.

Signs and symptoms

- **Contact dermatitis.** The sap occasionally inflames the skin; scent of fading flowers can give headache, dizziness and nausea.
- **Dysphagia and dysarthria.** Difficulty in swallowing and speaking after ingestion.
- **Gastro-intestinal irritation.** Abdominal pain, vomiting, profuse frothy salivation and diarrhoea.
- **Bradycardia, then tachycardia.** Pulse first slow, later rapid and weak; blood pressure falls.
- **Heart block and fibrillation.** AV block and other digitalis-type arrhythmias appear on ECG.
- **Tachypnoea and mydriasis.** Breathing is hurried and the pupils dilate.
- **Neuromuscular signs.** Muscle twitching, tetanic spasms and trismus (lockjaw).
- **Terminal stage.** Drowsiness, coma, respiratory paralysis; death usually from cardiac failure.

Treatment

- **Gastric lavage (stomach wash).** Reddy and Vij both advise it, followed by symptomatic care.
- **Activated charcoal in multiple doses.** Repeated doses interrupt reabsorption of the glycoside from the gut.
- Atropine sulphate 0.6 mg i.v., repeated as needed. It treats bradycardia and AV block of vagal origin.
- **Anti-digoxin Fab fragments.** They cross-react with oleandrin; Rao records 200 to 480 mg i.v. as life-saving.
- **Temporary cardiac pacing.** Needed for heart block that does not respond to atropine or isoprenaline.
- **Correction of hyperkalaemia with ECG monitoring.** Calcium is avoided because it worsens glycoside cardiotoxicity.
- **Supportive care.** Fluids, antiarrhythmics such as lignocaine or phenytoin for ventricular ectopy, and control of spasms.

Medicolegal aspects

- **Suicidal poison.** Root, leaves or fruit taken as paste or decoction; common among villagers in parts of India.
- **Abortifacient.** Root is taken internally or used locally; a root or twig of white oleander serves as an abortion stick. BNS 88 (formerly IPC 312): causing miscarriage.
- **Cattle poison.** Root juice on a rag is pushed into the animal's anus, or mixed with fodder. BNS 325 (formerly IPC 428/429).
- **Homicide uncommon.** The bitter taste and large dose make it a poor homicidal choice.
- **Accidental poisoning.** Use as a love philter, folk remedy for venereal disease or swelling, poisonous honey, and food roasted on oleander sticks.
- **BNS 123 (formerly IPC 328):** causing hurt by means of poison.

AUTOPSY FINDINGS AND SAMPLES

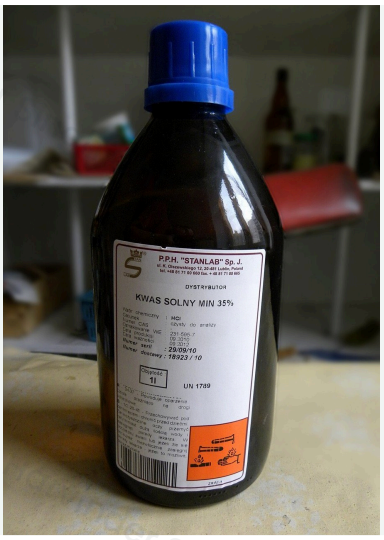
- **Non-specific findings.** General congestion of organs.
- **Petechial haemorrhages on the heart.** Small bleeds on the heart surface are the noted finding (Vij, Rao).
- **Stomach contents.** Fragments of root may be found in the stomach.
- **Heat resistant.** Nerium can be detected even from a burnt body or its ashes; arsenic is the other such poison.
- **Detectable long after death (Reddy).** Viscera go in saturated saline, with root or leaf fragments kept for botanical identification.

REMEMBER

Nerium and arsenic resist heat: both detectable in the ashes of a burnt body.

Hydrochloric acid (HCl)

Common names: Muriatic acid, Spirit of salt, Spirit of salts, Namak ka tezab



Hydrochloric acid (muriatic acid, spirit of salt), concentrated 35%: a reagent bottle with the corrosive warning; the strong acid fumes in moist air

Photo: Wikimedia Commons, Hydrochloric acid 02.jpg

SCIENTIFIC NAME	Hydrogen chloride in water; HCl
CLASS OF POISON	Corrosive: mineral (inorganic) acid
FATAL DOSE	15–20 mL of concentrated acid (Reddy, Vij)
FATAL PERIOD	18–36 hours (Reddy, Vij); 15–24 hours (Rao)
ACTIVE PRINCIPLE	Hydrochloric acid itself. A colourless, pungent, fuming liquid. A weak (about 0.2%) solution is normally present in gastric juice.

Mechanism of action

- **Coagulative necrosis (coagulation necrosis).** The acid draws water out of the tissue and coagulates cell proteins, so a firm crust (eschar) forms.
- **Conversion of haemoglobin to acid haematin.** This turns corroded mucosa brown or black after it first looks grey.
- **Fume injury.** HCl gives off fumes that inflame the larynx, trachea and lungs, so pulmonary oedema can kill even without much gut damage.
- **No true remote action.** Death comes from local damage, shock from pain, laryngeal spasm or oedema of the glottis.

Signs and symptoms

- **Immediate burning pain.** It runs from mouth and throat to the stomach and spreads over the abdomen.
- **Corrosion of mucous membrane.** The lining of mouth and tongue looks grey or greyish-white at first, later brown or black from acid haematin.
- **Skin usually spared.** HCl seldom corrodes the skin badly; the epidermis may peel after a few days (Vij).

- **Teeth.** No significant change, unlike the chalky-white teeth of sulphuric acid (Vij).
- **Dysphagia and odynophagia.** Swallowing is painful; the mouth fills with saliva and mucus.
- **Retching and vomiting.** Vomit is strongly acid and contains altered blood; every attempt to drink brings on fresh vomiting.
- **Respiratory irritation from fumes.** Coughing, choking, dyspnoea and cyanosis follow inhalation.
- **Chronic exposure (industrial).** Coryza, conjunctivitis, corneal ulcer, bronchitis, inflamed gums and loose teeth.
- **Clear sensorium.** The mind usually stays clear until death.
- **Late sequelae in survivors.** Oesophageal stricture and pyloric stenosis from scar tissue.

Treatment

- **Airway first.** Oedema of the glottis may need intubation or tracheostomy; give oxygen for fume inhalation.
- **Emetics contraindicated.** Vomiting brings the acid back over the oesophagus and risks perforation and aspiration.
- **Gastric lavage contraindicated.** The softened stomach or oesophagus may perforate under the tube (Reddy, Vij, Rao).
- **Dilution with water or milk.** About 250 mL given within 30 minutes of ingestion dilutes the acid in situ.
- **No bicarbonate or carbonates.** Neutralisation with them releases carbon dioxide, which distends and may rupture the stomach.
- **Demulcents.** Milk, egg white, olive oil or starch water coat and soothe the mucosa.
- **Supportive care.** Nil by mouth for 2–3 days, IV fluids, analgesia (morphine or pethidine), antibiotics; steroids are controversial.
- **Endoscopy at 12–24 hours.** It shows the site and depth of injury and guides feeding and follow-up.
- **Late care.** Bougie dilatation for stricture; surgery for perforation or pyloric stenosis. Skin and eyes are irrigated with copious water.

Medicolegal aspects

- **Suicide.** Most cases are suicidal (Reddy); a few are accidental or homicidal.
- **Accident.** Cheap toilet and tile cleaners in the home contain HCl, so accidental ingestion occurs.
- **Abortifacient.** Rarely injected into the vagina to procure abortion. BNS 88 (formerly IPC 312): causing miscarriage.
- **Acid attack (vitriolage).** HCl may be used instead of sulphuric acid. BNS 124(1) (formerly IPC 326A): grievous hurt by acid. BNS 124(2) (formerly IPC 326B): throwing or attempting to throw acid.
- **BNSS 397 (formerly CrPC 357C).** Every hospital must give free first aid and treatment to an acid attack victim and inform the police.
- **Laxmi v Union of India (Supreme Court, 2013).** Retail sale of acid was regulated: photo ID, stated purpose, a sale register and no sale to minors.
- **Industrial poisoning.** Fumes in factories cause acute respiratory deaths and chronic poisoning.

AUTOPSY FINDINGS AND SAMPLES

- **Corrosion less severe than with sulphuric acid.** Mucosa brownish, often without much skin damage.
- **Stomach.** Contains brownish fluid; mucosal folds of the whole stomach are brownish; perforation is rare.
- **Respiratory tract.** Acute inflammation and oedema of the air passages and lungs are common because of the fumes.
- **Free acid matters.** HCl occurs normally in gastric juice, so the analyst must show free acid in excess of normal. Little or none may remain if the victim survived 2 days or more.

- Preserve stomach with contents, a loop of upper small intestine, half liver and half of each kidney; corroded skin and stained clothes separately (Vij). Test: silver nitrate gives a curdy white precipitate of silver chloride.

> REMEMBER
>
> *Muriatic acid: grey mucosa, spared skin, fumes in the lungs, and free HCl must exceed normal gastric HCl.*

Nitric acid (HNO_3)

Common names: Aqua fortis, Red spirit of nitre, Spirit of nitre, Shora ka tezab



Fuming nitric acid (aqua fortis): the concentrated acid gives off choking fumes as soon as the bottle is opened
Photo: Wikimedia Commons, Nitric acid fuming.jpg



Fuming nitric acid: yellow from dissolved nitrogen dioxide. The yellow skin stain is a separate reaction with skin protein (xanthoproteic reaction)
Photo: Wikimedia Commons, Fuming nitric acid 40ml.jpg

SCIENTIFIC NAME	Nitric acid; HNO_3
CLASS OF POISON	Corrosive: mineral (inorganic) acid
FATAL DOSE	10–15 mL of concentrated acid (Reddy, Vij, Rao)
FATAL PERIOD	12–24 hours (Reddy, Vij); 24–30 hours (Rao)
ACTIVE PRINCIPLE	Nitric acid itself. A clear, colourless, heavy, fuming liquid with a choking odour; yellow fumes in air.

Mechanism of action

- **Coagulative necrosis.** Like other mineral acids it dehydrates tissue and coagulates protein, leaving an eschar.
- **Xanthoproteic reaction.** Nitric acid nitrates the aromatic amino acids (tyrosine, tryptophan) of tissue protein, forming yellow xanthoproteins that stain skin, teeth and mucosa.
- **Gas formation.** More eructation and greater abdominal distension than with sulphuric acid.
- **Fume injury.** Nitrous fumes irritate eyes and air passages and can cause asphyxia and pulmonary oedema.

Signs and symptoms

- **Burning pain.** Mouth, throat, oesophagus and stomach burn at once; pain spreads over the abdomen.
- **Yellow staining (xanthoproteic reaction).** Lips, angles of the mouth, fingers and clothing show yellow stains.
- **Teeth.** Crowns show a yellowish coating.
- **Eructation and distension.** Belching and a swollen abdomen are more marked than with sulphuric acid.
- **Vomiting.** Vomit is strongly acid with altered blood; thirst is intense but drinking provokes vomiting.

- **Fume inhalation.** Lacrimation, photophobia, sneezing, coughing, dyspnoea and asphyxia.
- **Tenesmus and suppressed urine.** Urine may be brown (Reddy).
- **Mind clear.** Consciousness is usually kept until close to death.
- **Late sequelae.** Oesophageal and gastric strictures and pyloric stenosis in survivors.

Treatment

- **Airway and oxygen.** Fumes and glottic oedema may need intubation or tracheostomy.
- **Emetics contraindicated.** Vomiting re-exposes the oesophagus and invites aspiration.
- **Gastric lavage contraindicated.** The tube may perforate the corroded stomach or oesophagus.
- **Dilution with water or milk.** About 250 mL within 30 minutes (Reddy).
- **No bicarbonate or carbonates.** Carbon dioxide adds to an already gas-distended stomach and may rupture it.
- **Demulcents.** Milk, egg white, olive oil or starch water.
- **Supportive care.** Nil by mouth, IV fluids, analgesics, antibiotics; steroids controversial.
- **Endoscopy at 12–24 hours to grade injury.** Later dilatation for stricture or surgery for perforation and pyloric stenosis.
- **Skin and eye burns.** Copious water irrigation for 15–30 minutes; refer eye burns to an ophthalmologist.

Medicolegal aspects

- **Accident or suicide.** Most cases are one of these; homicide is rare.
- **Accidental drinking unlikely.** Its yellow colour and irritating fumes warn the taker (Vij).
- **Diagnostic sign.** Yellow xanthoproteic staining on skin, teeth or clothes points to nitric acid.
- **Industrial exposure.** Fume inhalation in workshops and factories causes respiratory deaths.
- **Vitriolage.** Nitric acid is sometimes thrown to disfigure. BNS 124(1) (formerly IPC 326A) and BNS 124(2) (formerly IPC 326B).
- **Grievous hurt.** Permanent disfiguration of head or face or loss of sight falls under BNS 116 (formerly IPC 320).
- **BNSS 397 (formerly CrPC 357C).** Free treatment for acid attack victims and intimation to police.

AUTOPSY FINDINGS AND SAMPLES

- **Yellow corrosion of skin and mucosa.** Lips, mouth, tongue and oesophagus are stained yellow by the xanthoproteic reaction.
- **Stomach.** Wall soft, friable, ulcerated; yellow may be masked by brown-black acid haematin. Perforation is less common than with sulphuric acid.
- **Fume deaths.** Larynx, trachea and bronchi congested; lungs oedematous.
- Preserve stomach with contents, small intestine loop, half liver and half of each kidney; corroded skin and yellow-stained clothes separately (Vij). Test: brown ring with ferrous sulphate and sulphuric acid.

> REMEMBER
>
> *Aqua fortis turns tissue yellow: xanthoproteic reaction. Brown ring test confirms nitrates.*

Sulphuric acid (H_2SO_4)

Common names: Oil of vitriol, Vitriol, Battery acid, Gandhak ka tezab



Sulphuric acid (oil of vitriol) charring cloth: the acid pulls water out of cellulose and leaves black carbon, as it does to clothing and skin in vitriolage

Photo: Wikimedia Commons, Sulphuric acid on a piece of towel.JPG



Dehydration of sugar by concentrated sulphuric acid: white sucrose turns brown, then a black, spongy carbon mass, showing the acid's dehydrating, charring action

Photo: Wikimedia Commons, Dehydration of sugar by sulfuric acid.png

SCIENTIFIC NAME	Sulphuric acid; H_2SO_4
CLASS OF POISON	Corrosive: mineral (inorganic) acid
FATAL DOSE	5–10 mL of concentrated acid (Reddy, Vij, Rao)
FATAL PERIOD	12–24 hours (Reddy, Vij, Rao)
ACTIVE PRINCIPLE	Sulphuric acid itself. Heavy, oily, colourless, odourless, non-fuming and hygroscopic; it chars organic matter. Commercial acid is brown or dark.

Mechanism of action

- **Coagulative necrosis.** The acid dehydrates tissue and coagulates protein; the eschar limits deeper spread.
- **Exothermic reaction and charring.** Heat produced on contact with water adds a thermal burn; tissue is carbonised black.
- **Acid haematin.** Haemoglobin turns to acid haematin, so corroded areas go from grey-white to brown or black.
- **Site of worst damage.** Columnar epithelium of the stomach, especially the pylorus, suffers most; squamous oesophageal lining resists acid better.

Signs and symptoms

- **Corrosion of lips and chin.** Lips swollen and excoriated, with brown or black streaks running from the angles of the mouth to the chin and neck.
- **Teeth chalky-white and brittle.** Tongue swollen, sodden and black.
- **Immediate burning pain.** Pharyngeal pain is the commonest complaint; epigastric pain spreads over abdomen and chest.

- **Stridor, drooling, odynophagia and dysphagia.** Swallowing and speech become painful; the voice turns hoarse.
- **Vomiting.** Vomit is brown or black, mucoid, strongly acid and may hold shreds of charred stomach wall.
- **Intense thirst.** Every attempt to drink is followed by vomiting.
- **Shock and asphyxia.** Circulatory collapse or oedema of the glottis may kill early.
- **Abdomen distended and tender.** Severe constipation and tenesmus; chemical peritonitis follows perforation.
- **Mind clear till death.** Pupils usually dilated, eyes sunken.
- **Late sequelae.** Oesophageal stricture, pyloric stenosis and skin scars in survivors.

Treatment

- **Airway first.** Tracheostomy or intubation for oedema of the glottis.
- **Emetics contraindicated.** Vomiting re-exposes the mucosa and may rupture the stomach.
- **Gastric lavage contraindicated.** Perforation risk is highest with sulphuric acid.
- **Dilution in situ.** About 250 mL of water or milk within 30 minutes (Reddy).
- **No bicarbonate or carbonates.** They release carbon dioxide, distend the stomach and may rupture it.
- **Demulcents.** Olive oil, milk, egg white, starch water, melted butter.
- **Supportive care.** Nil by mouth about a week with IV nutrition, analgesia, antibiotics; prednisolone 60 mg/day (Reddy), though Vij and Rao call steroids controversial.
- **Endoscopy at 12–24 hours.** Grades the burn (Zargar grade I to IIIb) and predicts stricture; later bougie dilatation or surgery for pyloric stenosis.
- **Skin and eye burns.** Wash with large volumes of water; irrigate eyes 15–30 minutes.

Medicolegal aspects

- **Accident.** Mistaken for castor oil or glycerine; battery fluid in the home and workshop.
- **Suicide.** Most cases are suicidal (Reddy); Vij calls it rare today.
- **Homicide rare.** Sour taste, immediate pain and visible change in food give it away; infants and drunk victims are the exceptions.
- **Vitriolage.** Throwing acid, classically oil of vitriol, at the face to disfigure. The burns are deep and very painful, heal slowly with contracture and may blind. BNS 124(1) (formerly IPC 326A) and BNS 124(2) (formerly IPC 326B).
- **Grievous hurt.** Permanent disfiguration of head or face or loss of sight: BNS 116 (formerly IPC 320). Death makes it culpable homicide or murder.
- **Laxmi v Union of India (Supreme Court, 2013).** Regulated over-the-counter acid sale and fixed minimum compensation for victims. BNSS 397 (formerly CrPC 357C) mandates free treatment.
- **Abortifacient.** Taken by mouth or injected into the vagina. BNS 88 (formerly IPC 312).
- **Disposal of bodies.** The acid bath murders (John George Haigh). BNS 238 (formerly IPC 201): causing disappearance of evidence.

AUTOPSY FINDINGS AND SAMPLES

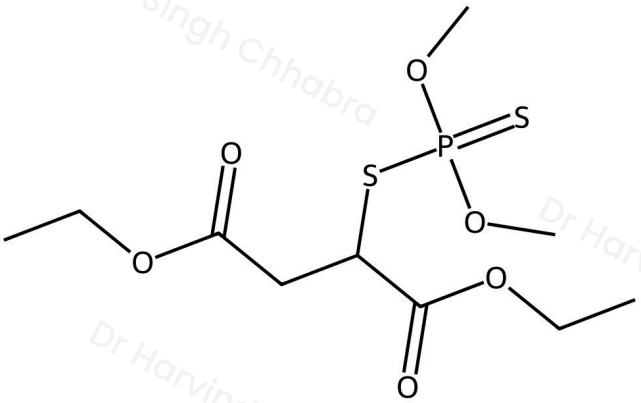
- **External corrosion.** Lips, angles of mouth, chin, neck, chest and fingers show grey-white areas that become brown or black and leathery; acid tracks run down from the mouth.
- **Stomach.** May become a soft, spongy black mass that breaks on touch; mucosal ridges worse than furrows; pylorus often worst. Perforation is common with chemical peritonitis.
- **Clothing.** Charred holes and brown-black stains; examine and preserve separately.

- **Time course.** Inflammation days 1–7, granulation days 4–7, perforation most likely days 7–21, cicatrisation and stricture from week 3.
- Preserve stomach with contents, small intestine loop, half liver and half of each kidney; corroded skin separately (Vij). Test: barium chloride gives white barium sulphate. Free acid may vanish if the victim lived 2 days.

> REMEMBER
>
> *Oil of vitriol: black char, chalky teeth, stomach a black spongy mass; perforation commonest of the three acids.*

Malathion

Common names: Kill bug, Bugsoline, Bugsolin-20, Cimexol, Jyothi oil (household insecticide brands)



Malathion: structural formula of the organophosphorus ester ($C_{10}H_{19}O_6PS_2$). The P=S group is converted in the liver to the P=O form, malaoxon

Structural formula drawn for these notes

SCIENTIFIC NAME	S-1,2-bis(ethoxycarbonyl)ethyl O,O-dimethyl phosphorodithioate, $C_{10}H_{19}O_6PS_2$. An alkyl organophosphate.
CLASS OF POISON	Agrochemical: organophosphorus insecticide (alkyl phosphate)
FATAL DOSE	About 1 g orally (Reddy; Vij; Rao)
FATAL PERIOD	Death usually within 24 hours if untreated (Reddy; Vij). Vij's table gives 0–6 hours. Up to 10 days in treated cases that fail (Reddy).
ACTIVE PRINCIPLE	Malathion itself. The liver converts part of it to malaoxon, the active anticholinesterase. Sold dissolved in a petroleum solvent such as aromax.

Mechanism of action

- **Irreversible acetylcholinesterase inhibition (phosphorylation).** Malaoxon, formed from malathion in the liver, binds the enzyme that breaks down acetylcholine, so acetylcholine piles up at nerve endings and keeps firing.
- **Ageing.** The bound enzyme loses an alkyl group and can no longer be reactivated. Malathion is a dimethyl compound, and these age within hours, so pralidoxime should start early. Reddy's 24–36 hours is a general figure.
- **Overstimulation, then paralysis.** Excess acetylcholine first drives muscarinic, nicotinic and brain receptors, and later blocks them.
- **Low mammalian toxicity.** Carboxylesterases in human liver break malathion down quickly; insects lack this pathway, which is why it is used as a household insecticide.

Signs and symptoms

- **Muscarinic features (SLUDGE / DUMBELS).** Salivation, lacrimation, urination, defaecation, gut cramps and emesis.
- **Bronchorrhoea and bronchospasm.** The airways flood with secretions and narrow, so breathing sounds wet and wheezy.
- **Miosis.** Pinpoint pupils, often with blurred vision; Vij notes it is not present in every case.
- **Bradycardia and hypotension.** A slow pulse and falling blood pressure from vagal overactivity.
- **Nicotinic features (MTWHF).** Mydriasis, tachycardia, weakness, hypertension and fasciculations from sympathetic ganglia and motor end-plate stimulation.
- **Fasciculations.** Fine twitching of eyelids, tongue, face and calves, followed by weakness and areflexia.
- **CNS features.** Headache, restlessness, anxiety, confusion, slurred speech, ataxia, convulsions and coma.
- Kerosene-like smell of breath, vomit and clothes from the solvent; Vij also describes a garlic odour.
- **Intermediate syndrome.** At 1–4 days, weakness of neck flexors, proximal limb muscles, cranial nerves and breathing muscles; it does not respond to atropine or oximes.
- **Organophosphate-induced delayed polyneuropathy (OPIDN).** At 1–5 weeks, calf pain and tingling, then foot drop and ascending flaccid weakness.

Treatment

- **Airway, breathing, circulation.** Suction secretions, give oxygen and intubate early; hypoxia should be corrected before atropine to reduce arrhythmia risk.
- **Skin and eye decontamination.** Clothes come off and skin is washed with soap and water; staff wear gloves because the poison passes through skin.
- **Gastric decontamination.** Lavage with water or saline only for a large ingestion within about 1 hour, airway protected, then activated charcoal 1 g/kg. Permanganate is not used: it is caustic and may oxidise malathion to the more toxic malaoxon. Ipecac and emetics are avoided.
- **Atropine sulphate (muscarinic antagonist).** Adult 2–4 mg IV, doubled every 5–10 minutes until atropinisation, then an infusion keeps that state for 24–48 hours.
- **Atropinisation end points.** Clear chest on auscultation, heart rate above 80/min, systolic BP above 80 mmHg, dry axillae and pupils no longer pinpoint. Full pupil dilatation is not the target.
- **Pralidoxime (2-PAM, oxime).** Reactivates enzyme before ageing and reverses nicotinic weakness. Books: 1–2 g IV over 10–15 min, repeated 6–12 hourly; WHO: 30 mg/kg bolus then 8 mg/kg/hour infusion.
- **Diazepam (benzodiazepine).** 5–10 mg IV for convulsions, agitation and fasciculations.
- **Contraindicated drugs.** Morphine, aminophylline, phenothiazines, reserpine, physostigmine and succinylcholine worsen cholinergic toxicity or prolong paralysis.
- **Observation for 3–4 days.** Intermediate syndrome can stop breathing after the patient seems well, so ventilator support must be ready.

Medicolegal aspects

- **Suicide.** Organophosphates are the commonest suicidal poison in rural India because they sit in every farm store.
- **Accidental poisoning.** Seen in manufacturers, packers, sprayers who work without protection, and children of users.
- **Homicide is rare.** The kerosene-like smell and early alarming signs give it away; alcohol has been used to mask the odour.
- Malathion is the least toxic common organophosphate (toxicity rating 4) and is sold as a household insecticide, so accidental home exposure is frequent.

- **Plasma and RBC cholinesterase.** RBC level below 50% of normal supports poisoning; plasma level falls faster and recovers faster.
- **BNS 123 (formerly IPC 328):** causing hurt by poison. BNS 103 (formerly IPC 302) applies if homicidal.
- **BNS 286 (formerly IPC 284):** negligent conduct with a poisonous substance, relevant to unsafe storage or spraying.
- Insecticides Act 1968 and Insecticides Rules 1971 control registration, sale, labelling and safe use of pesticides.

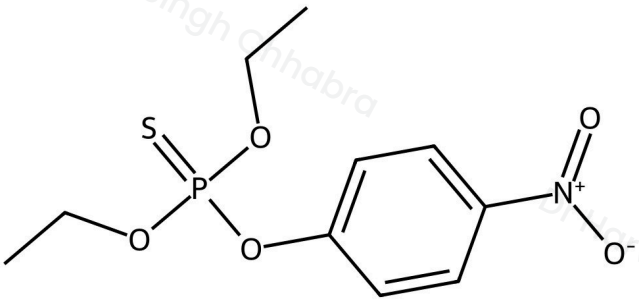
AUTOPSY FINDINGS AND SAMPLES

- **Asphyxial picture.** Cyanosis, deep lividity and fine froth, sometimes bloodstained, at the mouth and nostrils.
- **Kerosene-like smell from the mouth and stomach contents.** Gastric mucosa congested with submucosal petechiae.
- Lungs heavy, congested and oedematous with subpleural petechiae; brain congested and oedematous.
- Low RBC and end-plate cholinesterase can be shown; the poison resists putrefaction and is detectable in decomposed bodies.
- Viscera preserved in saturated sodium chloride solution, not rectified spirit, because organophosphates dissolve in alcohol. Heparinised blood is sent for cholinesterase.

> REMEMBER
>
> *Malathion is least toxic: human carboxylesterases detoxify it, insects cannot. Atropinise to a dry chest, not wide pupils.*

Parathion

Common names: Folidol (Follidol), E-605, Kill phos, Ekato, Nitrostigmine



Parathion: structural formula ($C_{10}H_{14}NO_5PS$). The P=S group is converted in the body to paraoxon, the active cholinesterase inhibitor
Structural formula drawn for these notes

SCIENTIFIC NAME	O,O-diethyl O-(4-nitrophenyl) phosphorothioate (diethyl nitrophenyl thiophosphate), $C_{10}H_{14}NO_5PS$. An aryl organophosphate.
CLASS OF POISON	Agrochemical: organophosphorus insecticide (aryl phosphate)
FATAL DOSE	175 mg orally, 80 mg i.m. (Reddy); 100 mg (Vij; Rao)
FATAL PERIOD	Death usually within 24 hours if untreated (Reddy; Vij). Vij's table gives 0–6 hours.
ACTIVE PRINCIPLE	Parathion, converted in the liver to paraoxon, the active toxin. Excreted as p-nitrophenol in urine.

Mechanism of action

- **Lethal synthesis to paraoxon.** Parathion itself is a weak inhibitor; liver cytochrome P450 turns it into paraoxon, which does the damage.
- **Irreversible acetylcholinesterase inhibition (phosphorylation).** Paraoxon disables the enzyme, so acetylcholine floods muscarinic, nicotinic and CNS synapses.
- **Ageing.** The phosphorylated enzyme loses an alkyl group and can no longer be reactivated, which is why oximes work best early.
- **Fat storage.** Parathion is stored in body fat and released slowly, so toxicity can be prolonged for about a week.

Signs and symptoms

- **Rapid onset.** Symptoms can start within 5 minutes of massive ingestion or inhalation and peak at 2–8 hours; skin absorption is slowest.
- **Muscarinic features (SLUDGE / DUMBELS).** Salivation, lacrimation, urination, diarrhoea, gut cramps and vomiting.

- **Bronchorrhoea and bronchospasm.** Wet, wheezy breathing that can mimic acute asthma and lead to pulmonary oedema.
- **Miosis.** Pinpoint pupils with blurred vision.
- Bradycardia, AV block and hypotension, or tachycardia and hypertension when nicotinic effects dominate.
- **Nicotinic features (MTWHF).** Mydriasis, tachycardia, weakness, hypertension and fasciculations.
- Muscle fasciculations and cramps, then flaccid paralysis of breathing muscles.
- **CNS features.** Restlessness, tremors, confusion, convulsions, coma and depression of the respiratory centre.
- **Chromolacryorrhoea.** Red tears from porphyrin in the lacrimal glands; rare.
- Intermediate syndrome (1–4 days) and delayed polyneuropathy (OPIDN, 1–5 weeks); Reddy names parathion among the causes of OPIDN.

Treatment

- **Airway and oxygen first.** Secretions are suctioned and the patient intubated early if breathing fails.
- **Decontamination.** Clothes removed and skin washed with soap and water; Reddy records death after one drop left on the forearm for two minutes, so speed matters.
- Gastric lavage with water or saline only for a large ingestion within about 1 hour, airway protected, then activated charcoal 1 g/kg. Permanganate is not used: it is caustic and may oxidise parathion to paraoxon.
- **Atropine sulphate.** Adult 2–4 mg IV, doubled every 5–10 minutes until atropinisation: clear chest, heart rate above 80/min, systolic BP above 80 mmHg, dry axillae, pupils no longer pinpoint.
- **Maintenance atropine.** An infusion holds atropinisation for 24–48 hours; Reddy notes an average need of 40 mg/day and up to 1000 mg/day.
- **Pralidoxime (2-PAM).** Books: 1–2 g IV over 10–15 min, repeated 6–12 hourly; WHO: 30 mg/kg bolus then 8 mg/kg/hour. Given early, before ageing.
- Diazepam 5–10 mg IV for seizures and fasciculations.
- **Contraindicated drugs.** Morphine, aminophylline, phenothiazines, reserpine, physostigmine and succinylcholine.
- **Watch for 3–4 days.** Fat stores keep releasing parathion, and intermediate syndrome can cause late respiratory arrest.

Medicolegal aspects

- **Highly toxic (toxicity rating 6, Vij).** A few drops on the skin or in a child have killed.
- **Suicide.** Organophosphates are the commonest suicidal poison in rural India.
- **Accidental poisoning.** Spraying without protection, spillage, and contamination of food; Vij cites the Kerala food poisoning cases.
- Homicide is rare because of the smell and early dramatic signs; alcohol has been used to hide the odour. Reddy records a 9-week infant killed with two drops.
- Parathion is banned in India for manufacture, import and use under the Insecticides Act 1968 (not stated in Reddy or Vij; see source notes).
- **BNS 123 (formerly IPC 328):** causing hurt by poison. BNS 103 (formerly IPC 302) if death is homicidal.
- **BNS 286 (formerly IPC 284):** negligent conduct with a poisonous substance.

AUTOPSY FINDINGS AND SAMPLES

- **Asphyxial signs.** Cyanosed face, bloodstained froth at the mouth and nostrils.
- Kerosene-like smell of stomach contents; congested gastric mucosa with submucosal petechiae.

- Lungs congested and oedematous with subpleural petechiae; frothy haemorrhagic exudate in the airways; brain congested.
- **Low RBC and end-plate cholinesterase.** Fatal blood parathion ranges from 0.5 to 34 mg/L (Vij). The poison survives putrefaction.
- Viscera preserved in saturated sodium chloride solution, not rectified spirit. Urine is sent too: p-nitrophenol turns strongly yellow with sodium hydroxide.

> REMEMBER
>
> *Parathion is a prodrug: liver makes paraoxon. Urine p-nitrophenol turns yellow with NaOH.*

DDT

Common names: DDT, Chlorophenothane



DDT (dichlorodiphenyltrichloroethane): delousing with a hand duster during the Second World War

Photo: CDC Public Health Image Library (US government), Public domain, via Wikimedia Commons



DDT sprayed over swamp water for mosquito (malaria) control, 1958

Photo: CDC Public Health Image Library (US government), Public domain, via Wikimedia Commons

SCIENTIFIC NAME	1,1,1-trichloro-2,2-bis(4-chlorophenyl)ethane, $C_{14}H_9Cl_5$. A chlorinated hydrocarbon (organochlorine).
CLASS OF POISON	Agrochemical: organochlorine (chlorinated hydrocarbon) insecticide
FATAL DOSE	30 g (Reddy; 150 mg/kg in his classification); 15–30 g (Vij; Rao)
FATAL PERIOD	Up to 6 hours (Rao); Reddy and Vij give none for DDT
ACTIVE PRINCIPLE	DDT itself, often dissolved in kerosene or other petroleum solvents that add their own toxicity.

Mechanism of action

- **Sodium channel opener.** DDT keeps axonal sodium channels open too long, so nerves fire repeatedly from a single stimulus.
- **CNS stimulant.** It acts chiefly on the cerebellum and motor cortex, causing tremors and convulsions, followed later by depression.
- **Myocardial sensitisation.** The heart becomes oversensitive to catecholamines, so ventricular arrhythmias can follow adrenaline.
- **Lipophilic storage.** DDT dissolves in fat, lodges in adipose tissue and is released slowly; it is also excreted in milk.

Signs and symptoms

- **Gastrointestinal irritation.** Salivation, nausea, vomiting and abdominal pain within an hour of ingestion.
- **Tremors.** Muscular tremor and myoclonic jerks, which may come before or without seizures.
- **Convulsions.** The commonest serious feature; seizures can come suddenly without any gut symptoms first.

- **Neurological prodrome.** Dizziness, weakness of the legs, agitation, apprehension and confusion.
- **Respiratory failure.** Death follows hypoxia from seizures, respiratory depression or aspiration of vomit.
- **Hepatotoxicity and nephrotoxicity.** Liver and kidney damage occur in some acute cases and more in chronic exposure.
- **Solvent effects.** Kerosene causes coughing, aspiration pneumonitis and CNS depression of its own.
- **Chronic poisoning.** Weight loss, weakness, tremor, ataxia and mental changes. DDT is a probable human carcinogen (IARC Group 2A).

Treatment

- **Airway and ventilation take priority.** Hypoxia from seizures and aspiration is the main danger.
- **Skin decontamination.** Clothes off and skin washed with soap and water, since solvent-borne DDT crosses the skin.
- **Gastric lavage with a protected airway, then activated charcoal.** The kerosene vehicle carries an aspiration risk.
- **No specific antidote.** Atropine and oximes have no role (Rao).
- Diazepam (benzodiazepine) for convulsions, followed by phenobarbitone or phenytoin if seizures persist.
- **Cholestyramine.** A bile-acid binding resin, 16 g/day in divided doses for several days, increases faecal excretion of organochlorines.
- **Adrenaline is avoided.** A sensitised myocardium can go into ventricular fibrillation with catecholamines.
- **Oily cathartics, milk and fats are avoided.** Lipids dissolve DDT and increase its absorption from the gut.

Medicolegal aspects

- **Persistent environmental pollutant.** DDT resists breakdown, accumulates in food chains (biomagnification) and appears in breast milk and dairy milk.
- **Accidental poisoning.** Commoner in children and in people who handle or spray the compound.
- Suicide occurs, more in rural areas; homicide is very rare because of the smell and the large dose needed.
- **India:** DDT is banned for agricultural use but permitted for public-health vector control, such as indoor spraying against malaria and kala-azar (not stated in Reddy or Vij; see source notes).
- Insecticides Act 1968 governs its registration, restriction and use.
- **BNS 123 (formerly IPC 328):** causing hurt by poison. BNS 286 (formerly IPC 284): negligent conduct with a poisonous substance.

AUTOPSY FINDINGS AND SAMPLES

- Asphyxial signs with froth, sometimes bloodstained, in the airways and pulmonary oedema.
- Congested gastrointestinal tract; stomach contents smell of kerosene from the solvent.
- Chronic cases show necrosis and degenerative change in the liver and kidneys.
- Adipose tissue, blood and urine are sent along with routine viscera; organochlorines are detected by gas chromatography. Viscera go in saturated saline.

> REMEMBER
>
> *DDT: sodium channel opener causing tremor and fits. No adrenaline, no milk or oils.*

Lead tetroxide

Common names: Red lead, Minium, Sindoor, Sindhur, Mitia sindhur, Vermilion (loosely), Lal sindoor



Lead tetroxide (Pb_3O_4, red lead, sindoor adulterant, minium pigment): bright orange-red powder
Photo: Wikimedia Commons, Red lead.jpg

SCIENTIFIC NAME	Lead tetroxide (triplumbic tetroxide), Pb_3O_4
CLASS OF POISON	Irritant: inorganic, metallic
FATAL DOSE	No separate figure for red lead. Lead salts: 20 g lead acetate, 40 g lead carbonate (Reddy); about 10 g/70 kg for most lead salts (Vij). Chronic: 1-2 mg lead daily (Reddy).
FATAL PERIOD	1-2 days (Reddy, Rao); within 24 h in severe cases, usually 2-3 days (Vij).
ACTIVE PRINCIPLE	Lead (Pb). Red lead is a scarlet crystalline powder, insoluble in water, used as a pigment and as sindoor.

Mechanism of action

- **Sulphydryl (-SH) binding.** Lead locks onto -SH groups of enzymes, so many cell enzymes stop working.
- **Inhibition of delta-aminolaevulinic acid dehydratase (ALA dehydratase).** The first steps of haem synthesis stall, so ALA rises in blood and urine.
- **Inhibition of ferrochelatase.** Iron cannot enter protoporphyrin, so free erythrocyte protoporphyrin (zinc protoporphyrin) rises and anaemia follows.
- **Mitochondrial and ATPase injury.** Lead upsets oxidative phosphorylation and Na-K and Ca ATPases; red cells die early (haemolysis).

Signs and symptoms

- **Acute (rare):** metallic, astringent taste with dry throat, thirst and burning abdominal pain. Vomiting and colic follow.
- **Constipation with black, offensive stools.** Lead sulphide forms in the gut and blackens the faeces; urine output falls (Vij).

- **Acute collapse.** Peripheral circulatory failure, paraesthesiae, cramps in the legs, then coma.
- **Chronic (plumbism, saturnism):** facial pallor around the mouth. It is one of the earliest signs and comes from capillary spasm.
- **Anaemia with basophilic stippling (punctate basophilia).** Red cells show many blue pinpoint dots; the anaemia is microcytic or normocytic and hypochromic.
- **Burtonian line (lead line).** A stippled blue-black line on the gum margin near carious teeth, from lead sulphide granules; seen in 50-70% (Reddy), up to 50% (Vij).
- **Lead colic and constipation.** Severe spasmodic pain, often at night, eased by pressure; the 'dry belly ache'.
- **Lead palsy.** Extensor weakness gives wrist drop (radial nerve) and foot drop; it is late and seen in under 10%.
- **Lead encephalopathy.** Headache, irritability, delirium, convulsions and coma; commonest in children, with permanent brain damage in about 85% of survivors (Reddy).
- **Reproductive effects.** Menstrual upset, sterility in both sexes and abortion between 3 and 6 months (Reddy).

Treatment

- **Remove the source.** Stop further exposure first; in chronic plumbism this matters more than any drug.
- **Gastric lavage with 1% magnesium or sodium sulphate.** Lead turns into insoluble lead sulphate and is not absorbed; 8-12 g magnesium or sodium sulphate orally clears the bowel (Reddy).
- **Calcium gluconate IV for colic.** About 2 g IV (Vij) relieves colic and pushes lead into bone.
- **$CaNa_2EDTA$ (calcium disodium versenate).** About 25–50 mg/kg/day, not more than 75 mg/kg/day, by IV infusion in 250–500 mL glucose or saline in divided doses for 5 days. The course may be repeated after a rest.
- **BAL (dimercaprol) with EDTA in severe cases.** BAL 4 mg/kg deep IM 4-hourly, started at least 4 h before EDTA, because EDTA alone pulls lead out of bone into the brain (Reddy). BAL is the chelator of choice when the kidneys are failing, as it leaves through bile.
- **Encephalopathy (children).** BAL 4 mg/kg 4-hourly plus $CaNa_2EDTA$ 75 mg/kg/day IV infusion until blood lead falls below 40 µg/dL, then oral chelation (Reddy).
- **Succimer (DMSA) orally.** 10 mg/kg 8-hourly for 5 days, then 12-hourly for 14 days (Reddy); 30 mg/kg (Vij). It is less toxic and suits outpatients.
- **D-penicillamine orally.** 0.3-0.5 g one to five times daily (Reddy); 0.5-1.5 g daily in adults (Vij). It is an alternative where DMSA is not available.
- **Supportive care.** Thiamine for nerve damage, fluids, anticonvulsants, and dialysis if the kidneys fail.

Medicolegal aspects

- **Sindoor.** Married Hindu women apply red lead in the hair parting; long use on the scalp causes chronic poisoning (Reddy, Vij, Rao).
- **Abortifacient.** Red lead paste on the abortion stick of the dai causes uterine contraction and degeneration of chorionic cells (Vij). BNS 88 to 90 (formerly IPC 312 to 314): causing miscarriage, and death from it.
- **Cattle poison.** Red lead is used alone or mixed with arsenic (Reddy). BNS 325 (formerly IPC 428/429): killing or maiming animals.
- **Homicide and suicide are rare.** The taste and the large fatal dose put off the poisoner, and the death is slow and painful (Vij).
- **Industrial disease.** Paint and pigment workers are at risk; chronic poisoning is far commoner than acute (Reddy).

- **Mineral stain.** Red lead stains on clothing can mimic bloodstains, so a red stain needs testing before it is called blood (Reddy).
- **BNS 123 (formerly IPC 328):** causing hurt by poison, where red lead is given with intent.

AUTOPSY FINDINGS AND SAMPLES

- **Acute:** gastroenteritis. Gastric mucosa thickened, softened and eroded, with a greyish-white deposit.
- **Chronic:** blue line on the gums, contracted liver and kidneys, pale swollen brain, fatty change in paralysed muscles.
- **Abortion stick use.** Excoriation, bruising or perforation of the upper vagina, cervix or uterus, with sepsis.
- **Preservation:** usual viscera in saturated saline. For chronic poisoning add 10 cm of long bone shaft, 15-20 plucked scalp hairs and nails (Vij); lead is stored in bone, hair and nails.

> REMEMBER
>
> *Red lead is the sindoor and the abortion-stick paste; its stain can be mistaken for blood.*

Lead sulphide

Common names: Galena, Surma, Kohl, Kajal (when lead-based), Collyrium



Lead sulphide (PbS, galena): heavy metallic grey crystals, the main ore of lead
Photo: Rob Lavinsky, iRocks.com, CC BY-SA 3.0, via Wikimedia Commons

SCIENTIFIC NAME	Lead(II) sulphide, PbS
CLASS OF POISON	Irritant: inorganic, metallic
FATAL DOSE	No separate figure; Reddy calls lead sulphide the least toxic lead salt. Lead salts generally about 10 g/70 kg (Vij). Chronic: 1-2 mg lead daily (Reddy).
FATAL PERIOD	Chronic course over weeks to months; acute lead poisoning 1-2 days (Reddy, Rao), 2-3 days (Vij).
ACTIVE PRINCIPLE	Lead (Pb). Galena forms cubic crystals; it is sold powdered as surma in place of antimony sulphide (Rao).

Mechanism of action

- **Cumulative poison.** Daily traces from eye cosmetics add up, because lead leaves the body far slower than it enters; bone half-life is over 25 years (Vij).
- **Greater absorption in children.** A child absorbs about 50% of swallowed lead against 10% in an adult (Reddy); hand-to-mouth habits and pica add to the dose.
- **Haem synthesis block.** Lead inhibits ALA dehydratase and ferrochelatase, so ALA, coproporphyrin and protoporphyrin pile up and anaemia follows.
- **Neurotoxicity.** Lead damages nerve cells and myelin and causes cerebral oedema (Rao); the growing brain suffers most.

Signs and symptoms

- **Pica.** The child craves non-food items, licks lead paint or plaster, and the exposure grows (Vij, Rao).
- **Pallor and anaemia.** Hypochromic microcytic anaemia with basophilic stippling of red cells.
- **Lead encephalopathy.** Vomiting, ataxia, lethargy, irritability, convulsions and coma; seen mostly in infants and young children (Rao).

- **Cerebellar ataxia.** Unsteady gait is common in children with lead poisoning (Reddy).
- **Developmental harm.** Even low blood levels are linked with lower IQ, behaviour problems, slowed growth and impaired hearing (Rao).
- **Abdominal colic and constipation.** Spasmodic pain, eased by pressure.
- **Burtonian line.** A blue gum line is less often seen in infants without teeth, as it needs carious teeth and food debris.
- **Lead lines on X-ray (lead osteopathy).** Dense transverse bands at the metaphyses of growing long bones and the iliac crest; they need about 4 weeks of heavy exposure (Reddy).

Treatment

- **Stop the source.** Discard lead-based surma or kajal and look for other sources in the home; this is the first step of treatment.
- **Screen the family.** Siblings and the mother share the cosmetic; Vij notes infants may also take lead from breast milk of an exposed mother.
- **Check blood lead and haem markers.** Blood lead, zinc protoporphyrin, urinary ALA and coproporphyrin, and X-ray for lead lines guide treatment.
- **Encephalopathy.** BAL 4 mg/kg deep IM 4-hourly, with $CaNa_2EDTA$ 75 mg/kg/day IV started 4 h after the first BAL dose, until blood lead is below 40 µg/dL (Reddy).
- **Severe without encephalopathy (blood lead over 70 µg/dL).** BAL 12 mg/kg/day plus EDTA 50 mg/kg/day, then oral chelation (Reddy).
- **Mild to moderate.** Oral succimer (DMSA) 10 mg/kg 8-hourly for 5 days, then 12-hourly for 14 days; or D-penicillamine 30 mg/kg/day in 3 doses, built up over 2 weeks (Reddy).
- **Supportive care.** Iron and calcium-rich diet, anticonvulsants, and follow-up blood lead for rebound after chelation.

Medicolegal aspects

- **Accidental chronic poisoning.** Surma used as a collyrium on the eyes, mainly by Muslims (Vij), and long use of lead cosmetics cause plumbism (Rao).
- **Infants and children at most risk.** Kohl applied to babies' eyes and swallowed from fingers adds to pica and lead paint exposure.
- **Adulteration.** True surma is antimony trisulphide; lead sulphide is sold in its place (Rao).
- Drugs and Cosmetics Act 1940 governs cosmetics such as kajal; a lead-containing product is a matter for the drug control authority.
- Homicide and suicide are not expected; lead sulphide is the least toxic lead salt (Reddy).
- **BNS 106 (formerly IPC 304A):** causing death by a rash or negligent act, which may apply where a seller negligently supplies a lead cosmetic that kills.

AUTOPSY FINDINGS AND SAMPLES

- **Brain pale and greatly swollen (cerebral oedema).** PAS-positive material may lie in the perivascular spaces (Reddy).
- Eosinophilic intranuclear inclusions in liver cells and proximal renal tubules (Reddy).
- Lead lines at metaphyses on radiographs of the long bones in children.
- **Preservation:** blood, liver and kidney, plus 10 cm of long bone, 15-20 plucked scalp hairs and nails for chronic lead (Vij). The residual surma or kajal should be seized and sent for analysis.

> REMEMBER
>
> *Real surma is antimony sulphide; lead sulphide (galena) passes as it and poisons children.*

Oxalic acid

Common names: Acid of sugar, Salt of sorrel, Essential salt of lemons (potassium binoxalate and quadroxalate), Ink-stain remover



Oxalic acid (acid of sugar) crystals: small colourless crystals that look much like Epsom salt (magnesium sulphate), a classic cause of accidental poisoning
Photo: Wikimedia Commons, Oxalic acid dihydrate.jpg

SCIENTIFIC NAME	Ethanedioic acid; $C_2H_2O_4$ (hydrated form $C_2H_2O_4.2H2O$)
CLASS OF POISON	Corrosive: organic acid, with remote (systemic) action
FATAL DOSE	15–20 g (Reddy, Vij)
FATAL PERIOD	1–2 hours in fulminant cases; within 12 hours from hypocalcaemia; 2–14 days from uraemia
ACTIVE PRINCIPLE	Oxalic acid and soluble oxalates. Colourless prismatic crystals that look like magnesium sulphate (Epsom salt) and zinc sulphate.

Mechanism of action

- **Local corrosive action.** Crystals and solutions over 10% corrode the gut mucosa; weaker solutions act as strong irritants.
- **Hypocalcaemia.** Absorbed oxalate binds serum calcium as insoluble calcium oxalate, so free calcium falls and nerves and muscles become hyperexcitable.
- **Nephrotoxicity.** Calcium oxalate crystals block and damage renal tubules, causing acute tubular necrosis and uraemia.
- **Shock.** Large doses cause rapid cardiovascular collapse; ventricular fibrillation may follow.

Signs and symptoms

- **Fulminant poisoning (15 g or more).** Sour, bitter burning taste, constriction of the throat and burning pain from mouth to stomach.
- **Coffee-ground vomiting.** Vomit contains altered blood and mucus.
- **Skin seldom corroded.** Local skin damage is slight; cuticle may peel later.

- **Hypocalcaemic tetany.** Muscle irritability and tenderness, carpopedal spasm and convulsions.
- **Paraesthesia.** Numbness and tingling of the fingertips and legs.
- **Cardiovascular collapse.** Weak, irregular pulse, bradycardia, ventricular fibrillation.
- **Central depression.** Stupor and coma in some patients.
- **Delayed renal failure.** Scanty or suppressed urine with blood, albumin and calcium oxalate crystals (oxaluria).
- **Oxaluria.** Envelope-shaped calcium oxalate crystals seen in urine under the microscope.

Treatment

- **Cautious gastric lavage allowed.** Corrosion is milder than with mineral acids, so a soft tube is passed carefully, adding calcium lactate or gluconate (2 teaspoons per lavage).
- **Chemical antidote:** calcium. Lime water, chalk suspended in water or milk, calcium lactate, gluconate or chloride form insoluble calcium oxalate. About 1.5 g chalk neutralises 1 g oxalic acid.
- **Calcium gluconate 10%, 10 mL slow IV.** Repeat at frequent intervals to correct hypocalcaemia and bind circulating oxalate.
- **Demulcent drinks.** They protect the mucosa from further corrosion.
- **Bowel evacuation.** Enema or castor oil removes unabsorbed poison.
- **Renal support.** Watch urine output and fluids; dialysis or exchange transfusion for renal failure.
- **Symptomatic care.** Calcium replacement and renal support carry treatment; parathyroid extract is obsolete.

Medicolegal aspects

- **Accident.** Mistaken for Epsom salt (magnesium sulphate) taken as a purgative, or for sodium bicarbonate.
- **Distinguishing Epsom salt.** Oxalic acid tastes sour, is strongly acid, sublimes on heating, bleaches ink stains and effervesces with sodium carbonate (Vij).
- **Suicide.** Occasionally, because it is easily available at home.
- **Homicide rare.** Sour taste, early signs and easy chemical detection in vomit and organs.
- **Forgery.** Used to bleach out ink writing and signatures on documents. BNS 336 (formerly IPC 463/465): forgery.
- **Abortifacient.** Rarely injected into the vagina. BNS 88 (formerly IPC 312).
- **Industrial and household source.** Brass and copper cleaning, leather, calico printing, stain removal.

AUTOPSY FINDINGS AND SAMPLES

- **Mouth and oesophagus bleached.** Strong solutions whiten the mucosa as if scalded; oesophagus corrugated with longitudinal erosions.
- **Stomach.** Red or bleached mucosa with dark brown or black streaks running lengthwise in a network; contents gelatinous and brown from acid haematin. Perforation is rare.
- **Kidneys.** Swollen and congested; proximal tubules necrosed and packed with calcium oxalate crystals.
- **Interpretation.** Up to about 20 mg is passed in urine daily from food, so quantity and the gross picture matter. Test: barium nitrate gives white barium oxalate, soluble in HCl or HNO_3.

> REMEMBER
>
> *Acid of sugar: looks like Epsom salt, steals calcium. Antidote is calcium: lime water, chalk, gluconate.*

Carbolic acid

Common names: Phenol, Phenic acid, Phenyl (household disinfectant, loosely), Hospital smell



Phenol (carbolic acid) crystals: long colourless needles that turn pink on exposure to air and smell of hospital disinfectant
Photo: Wikimedia Commons, Phenol (carbolic acid)04.jpg

SCIENTIFIC NAME	Hydroxybenzene; C_6H_5OH
CLASS OF POISON	Corrosive: organic (aromatic) acid, with local and remote action (CNS depressant)
FATAL DOSE	10–15 g (Reddy, Vij); 5–15 g (Rao)
FATAL PERIOD	3–4 hours (Reddy); a few hours, or some days if renal failure (Vij)
ACTIVE PRINCIPLE	Phenol. Colourless needle crystals that turn pink and liquefy in air; crude acid is a dark-brown liquid with cresol. Not truly acidic: it does not turn blue litmus red.

Mechanism of action

- **Protoplasmic poison.** Phenol precipitates protein and penetrates deeply, causing coagulation necrosis of skin and mucosa.
- **Local anaesthesia.** Nerve endings are first stimulated, then paralysed, so burning gives way to tingling and numbness.
- **CNS depression.** Absorbed phenol depresses the brain, especially the respiratory centre, causing coma and respiratory failure.
- **Renal and blood toxicity.** Haemolysis, methaemoglobinaemia and tubular damage; metabolites hydroquinone and pyrocatechol colour the urine.

Signs and symptoms

- **Corrosion with numbness.** Hot burning pain from mouth to stomach, then tingling and anaesthesia.
- **White hardened mucosa.** Lips, mouth and tongue turn white and hard; skin shows a white opaque painless eschar that leaves a brown stain.
- **Phenolic odour.** Strong carbolic smell in breath and vomit.
- **CNS depression.** Headache, giddiness, unconsciousness and coma.

- **Miosis.** Pupils contracted (Reddy, Vij).
- **Hypothermia and shock.** Subnormal temperature, cold clammy sweat, rapid feeble irregular pulse.
- **Respiratory failure.** Slow, laboured, stertorous breathing with dusky cyanosis; laryngeal and pulmonary oedema.
- **Carboluria.** Scanty urine, often normal or faintly green when passed, turning dark smoky green or black on standing.
- **Methaemoglobinaemia and haemolysis.** Characteristic in severe cases.
- **Chronic poisoning (phenol marasmus).** Anorexia, weight loss, dark urine and ochronosis: dark pigment in cornea and cartilage.

Treatment

- **Emetics fail.** The local anaesthetic effect blunts the vomiting reflex.
- **Gastric lavage is allowed, unlike in mineral acids.** Phenol hardens rather than softens the stomach wall, so the tube is safe (Reddy, Vij, Rao).
- **Lavage fluid.** Lukewarm water with activated charcoal, olive oil, castor oil, magnesium or sodium sulphate, saccharated lime or 10% glycerine; continue until washings are clear and odourless.
- **Leave in the stomach.** 30 g magnesium sulphate or liquid paraffin after lavage; olive or vegetable oil lifts phenol off the mucosa.
- **Demulcents.** Milk, egg white, barley water.
- **IV saline with sodium bicarbonate.** Supports circulation, dilutes blood phenol and promotes diuresis.
- **Methylene blue IV for severe methaemoglobinaemia.** Haemodialysis for renal failure; artificial respiration if breathing fails.
- **Skin contamination.** Remove clothes, wash with polyethylene glycol or soap and water for at least 15 minutes; olive oil or 10% ethyl alcohol act as solvents.

Medicolegal aspects

- **Suicide.** Once popular because it was cheap and available; less so today.
- **Accident.** Crude acid taken by mistake, children drinking household disinfectant, or medicinal over-use.
- **Homicide rare.** Strong smell and taste usually defeat attempts, though cases in drinking water and bread are recorded.
- **Abortifacient.** Injected into the vagina or uterus by quacks. BNS 88 (formerly IPC 312).
- **Vitriolage.** Occasionally thrown to disfigure. BNS 124 (formerly IPC 326A/326B).
- **Normal urine contains traces.** About 15 mg phenol is excreted daily from protein digestion (Engel), so quantity matters.
- **Historical.** Lister used carbolic acid for antiseptic surgery; carboluria warned of toxicity from dressings.

AUTOPSY FINDINGS AND SAMPLES

- **External.** Greyish-white or brown corrosion tracks from the angles of the mouth to the chin; skin burns darken and parchmentise after death. Phenolic smell.
- **Leathery stomach.** Mucosa thickened, opaque, grey or brown and leathery with swollen rugae; furrows dark red. Perforation is unusual because the wall hardens.
- **Contact necrosis.** Liver and spleen show whitish hardened patches where phenol passed through the stomach wall.
- **Kidneys.** Haemorrhagic nephritis if death is delayed; dark semifluid blood.
- Preserve viscera in saturated common salt solution, not alcohol, which masks phenol (Vij). Test: 10% ferric chloride turns urine violet or blue.

REMEMBER

Gastric lavage is allowed: phenol tans the stomach leathery. Green urine is carboluria.

Calomel

Common names: Calomel, Mercurous chloride, Subchloride of mercury, Raskapoor



Mercurous chloride (Hg_2Cl_2, calomel) in its natural mineral form: pale translucent crystals
Photo: Rob Lavinsky, iRocks.com, CC BY-SA 3.0, via Wikimedia Commons

SCIENTIFIC NAME	Mercury(I) chloride, Hg_2Cl_2
CLASS OF POISON	Irritant: inorganic, metallic
FATAL DOSE	1.5-2 g/70 kg (Vij). Reddy gives no figure for calomel; mercuric chloride 1-2 g (Reddy), 0.5-1 g (Vij).
FATAL PERIOD	Mercury salts: usually 3-5 days, can be a few hours (Vij, Reddy). Acute calomel deaths are rare.
ACTIVE PRINCIPLE	Mercurous ion (Hg2 2+). A heavy, white, tasteless powder, insoluble in water; its insolubility is the main bar to its toxicity (Vij).

Mechanism of action

- **Low solubility.** Mercurous salts dissolve poorly, so far less is absorbed than from mercuric chloride (Reddy, Vij).
- **Conversion to mercuric salt.** On exposure to light, calomel changes to the toxic mercuric chloride (Rao).
- **Sulphydryl (-SH) binding.** Mercury binds -SH groups of enzymes and cell proteins and stops their work.
- **Nephrotoxicity and hypersensitivity.** The kidney is the main target organ (Reddy); acrodynia is thought to be a hypersensitivity reaction to mercurous chloride (Vij).

Signs and symptoms

- **Acute:** metallic taste, abdominal pain, vomiting and purging. Calomel is a purgative, so diarrhoea is expected.
- **Acrodynia (pink disease, Swift disease, erythroedema).** Infants given calomel teething powder develop fever, a pink rash, irritability and photophobia (Vij).

- **Acrodynia:** extremities. Hands and feet turn puffy, pink, painful and paraesthetic, the skin peels and teeth may be shed (Reddy). Sweating is profuse.
- **Chronic mercurialism (hydrargyrism):** gingivitis and salivation (ptyalism). Gums are sore and swollen, teeth loosen, and a blue line may appear.
- **Mercurial tremor (Danbury tremor).** A coarse intention tremor starts in the fingers and shows first in handwriting; the advanced form is 'hatter's shakes'.
- **Erethism (erethismus mercurialis).** Shyness, timidity, irritability, insomnia and memory loss; the source of 'mad as a hatter'.
- **Mercuria lentis.** A brown reflex from the anterior lens capsule on slit lamp; both eyes, with no loss of vision (Reddy).
- **Nephrotoxicity.** Albuminuria, casts and membranous glomerulonephritis; chronic renal failure with uraemia.

Treatment

- **Remove from exposure.** Stop the teething powder or laxative; nothing else works while intake continues.
- **Acute ingestion.** Gastric lavage with 5% sodium formaldehyde sulphoxylate, leaving 100 mL in the stomach, and egg albumin to bind mercury (Vij, Rao).
- **BAL (dimercaprol).** 100 mg deep IM 4-hourly for 48 h (Vij); in chronic cases 100 mg every 8 h for 8-10 days after the first 2 days (Reddy).
- **Succimer (DMSA) orally.** 30 mg/kg/day for 5 days, then 20 mg/kg/day for 14 days (Reddy); DMPS is an alternative.
- **D-penicillamine.** 250 mg four times daily in adults; 25-40 mg/kg/day (maximum 1 g) in two doses for children, until urine mercury is low (Reddy). N-acetyl penicillamine is preferred for chronic inorganic mercury (Vij).
- **Contraindicated:** $CaNa_2EDTA$. It is nephrotoxic with mercury (Reddy).
- **Contraindicated:** BAL with penicillamine together. The pair may form a toxic compound (Reddy).
- **Supportive care.** Keep urine alkaline, oral hygiene, belladonna for salivation (Vij), and haemodialysis for renal failure.

Medicolegal aspects

- **Accidental poisoning of infants.** Calomel teething powders caused acrodynia (Vij, Reddy).
- **Laxative abuse.** Long use of calomel purgatives causes chronic mercury poisoning (Rao).
- **Low acute toxicity.** Calomel is used as a purgative because it is nontoxic in therapeutic dose (Vij); suicide and homicide with it are rare.
- **Abortifacient purgative.** Calomel is listed among drastic purgatives used to procure abortion (Reddy, Vij). BNS 88 (formerly IPC 312): causing miscarriage.
- **Industrial mercurialism.** Hat, thermometer and barometer workers suffer tremor and erethism; such disease may give a claim to compensation.
- **BNS 123 (formerly IPC 328):** causing hurt by poison, where it is given with intent.

AUTOPSY FINDINGS AND SAMPLES

- **Gut changes.** Inflammation of the stomach and selective ulceration of the caecum and colon, where mercury is re-excreted.
- **Kidneys.** Toxic nephritis with tubular necrosis or membranous glomerulonephritis.
- **Liver and heart.** Fatty change; subendocardial haemorrhages in the heart.

- **Preservation:** kidney and liver (highest mercury), blood and urine, plus hair and nails in chronic cases. Diet supplies background mercury (5–20 µg/day, Vij), so only levels above the reference range (blood about 3.6 µg/dL, urine about 15 µg/L) support poisoning.

> REMEMBER
>
> *Calomel teething powder caused pink disease. Mercurous salts are insoluble, so far less toxic than mercuric.*

Quick-reference table

NAME	CLASS	FATAL DOSE	FATAL PERIOD	ACTIVE PRINCIPLE
Abrus precatorius	Irritant: organic, vegetable	1–2 seeds (90–120 mg) by injection	3–5 days	Abrin, a heat-labile toxalbumin
Dhatura	Neurotic: cerebral, deliriant	0.6 to 1 g seeds; 50 to 125 seeds	24 hours (Reddy, Vij); 3 to 24 hours (Rao)	Hyoscine, hyoscyamine, traces of atropine
Croton	Irritant: organic, vegetable	4–5 seeds or 1–2 mL oil	6 hours to 3 days (Reddy); few hours to few days (Vij)	Crotin (toxalbumin); crotonoleic acid resin in oil
Castor	Irritant: organic, vegetable	5–10 chewed seeds; ricin about 6 mg	2–5 days (Reddy); 2 to several days (Vij)	Ricin, a toxalbumin; oil holds none
Marking nut	Irritant: organic, vegetable	5–10 g	12–24 hours	Semecarpol and bhilawanol in the pericarp juice
Nux vomica	Neurotic: spinal poison (convulsant)	Strychnine 15 to 100 mg; one crushed seed	1 to 2 hours (Reddy, Rao); a few hours (Vij)	Strychnine and brucine in the seeds
Aconite	Cardiac poison (Reddy, Vij); neurotoxic in Rao	Root 1 g; aconitine 2 to 5 mg	2 to 6 hours (Reddy); average 6 hours, range 45 minutes to 24 hours (Vij)	Aconitine and allied alkaloids, most in the root
Poppy husk	Neurotic: cerebral, somniferous (narcotic)	Not fixed; morphine 200 mg fatal in non-addict	6 to 12 hours (Reddy); a few hours to 2 days (Vij), as for opium	Trace morphine, codeine, noscapine, papaverine
Poppy capsule	Neurotic: cerebral, somniferous (narcotic)	As opium: about 2 g; morphine 200 mg	6 to 12 hours (Reddy); a few hours to 2 days (Vij)	Latex: morphine, codeine, thebaine, noscapine, papaverine
Poppy seeds	Non-poisonous food item from the opium poppy (Neurotic: somniferous plant)	Not poisonous in ordinary use	Not applicable	No opium; yields bland edible oil
Crude opium	Neurotic: cerebral, somniferous (narcotic)	Opium 2 g; morphine 200 mg	6 to 12 hours (Reddy); a few hours to 2 days (Vij)	Morphine 10%, codeine, thebaine; noscapine, papaverine
Tobacco	Cardiac poison (Reddy, Vij)	Nicotine 40 to 100 mg; tobacco 15 to 30 g	5 to 15 minutes (Reddy); a few minutes to a few hours (Rao, Vij)	Nicotine and anabasine in dried leaves
Cannabis	Neurotic: cerebral, deliriant (Reddy, Rao); hallucinogen (Vij)	Charas 2 g, ganja 8 g, bhang 10 g/kg	Several days (Reddy); 12 to 24 hours (Vij)	Delta-9-tetrahydrocannabinol (THC) in the resin
Cerbera thevetia	Cardiac poison (vegetable cardiac glycoside)	8 to 10 seeds; 15 to 20 g root	2 to 3 hours (Reddy, Vij); death often comes later, over 1 to 3 days, from arrhythmia.	Thevetin, thevetoxin, nerifolin, peruvoside, cerberin: cardiac glycosides

Nerium oleander	Cardiac poison (vegetable cardiac glycoside)	15 to 20 g root; 5 to 15 leaves	20 to 36 hours (Reddy); 24 to 36 hours (Vij, Rao).	Oleandrin, nerin, folinerin; neriodorin, neriodorein, karabin (Vij)
Hydrochloric acid (HCl)	Corrosive: mineral (inorganic) acid	15–20 mL concentrated acid	18–36 hours (Reddy, Vij); 15–24 hours (Rao)	Hydrochloric acid; normally present dilute in gastric juice
Nitric acid (HNO_3)	Corrosive: mineral (inorganic) acid	10–15 mL concentrated acid	12–24 hours (Reddy, Vij); 24–30 hours (Rao)	Nitric acid (aqua fortis); fuming, choking odour
Sulphuric acid (H_2SO_4)	Corrosive: mineral (inorganic) acid	5–10 mL concentrated acid	12–24 hours (Reddy, Vij, Rao)	Sulphuric acid (oil of vitriol); oily, non-fuming, chars tissue
Malathion	Agrochemical: organophosphorus insecticide (alkyl phosphate)	About 1 g orally	Death usually within 24 hours if untreated (Reddy; Vij). Vij's table gives 0–6 hours. Up to 10 days in treated cases that fail (Reddy).	Malathion; activated to malaoxon in the liver
Parathion	Agrochemical: organophosphorus insecticide (aryl phosphate)	175 mg oral (Reddy); 100 mg (Vij)	Death usually within 24 hours if untreated (Reddy; Vij). Vij's table gives 0–6 hours.	Parathion; liver converts it to paraoxon
DDT	Agrochemical: organochlorine (chlorinated hydrocarbon) insecticide	15–30 g	Up to 6 hours (Rao); Reddy and Vij give none for DDT	DDT, a chlorinated hydrocarbon
Lead tetroxide	Irritant: inorganic, metallic	Lead salts 10-40 g; red lead: no separate figure	1-2 days (Reddy, Rao); within 24 h in severe cases, usually 2-3 days (Vij).	Lead; scarlet insoluble Pb_3O_4 powder (sindoor)
Lead sulphide	Irritant: inorganic, metallic	Least toxic lead salt; chronic intake 1-2 mg/day	Chronic course over weeks to months; acute lead poisoning 1-2 days (Reddy, Rao), 2-3 days (Vij).	Lead; galena (PbS) sold as surma
Oxalic acid	Corrosive: organic acid, with remote (systemic) action	15–20 g	1–2 hours in fulminant cases; within 12 hours from hypocalcaemia; 2–14 days from uraemia	Oxalic acid; crystals resemble Epsom salt
Carbolic acid	Corrosive: organic (aromatic) acid, with local and remote action (CNS depressant)	10–15 g	3–4 hours (Reddy); a few hours, or some days if renal failure (Vij)	Phenol (hydroxybenzene); carbolic hospital smell
Calomel	Irritant: inorganic, metallic	1.5-2 g (Vij)	Mercury salts: usually 3-5 days, can be a few hours (Vij, Reddy). Acute calomel deaths are rare.	Mercurous chloride; white, tasteless, insoluble powder

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11. **Poppy capsule**: Unripe Papaver somniferum capsule, lanced. Photo: Wikimedia Commons, Opium pod cut to demonstrate fluid extraction1.jpg https://commons.wikimedia.org/wiki/File:Opium_pod_cut_to_demonstrate_fluid_extraction1.jpg
12. **Poppy seeds**: Poppy seeds (khus khus). Photo: Wikimedia Commons, Poppy seeds.jpg https://commons.wikimedia.org/wiki/File:Poppy_seeds.jpg
13. **Crude opium**: Crude opium (afeem). Photo: Wikimedia Commons, Black tar (raw) opium seized in Afghanistan.jpg https://commons.wikimedia.org/wiki/File:Black_tar_(raw)_opium_seized_in_Afghanistan.jpg
14. **Tobacco**: Nicotiana tabacum (tobacco). Photo: Wikimedia Commons, P1000485 Nicotiana tabacum (tobacco) (Solanaceae) Plant.JPG https://commons.wikimedia.org/wiki/File:P1000485_Nicotiana_tabacum_(tobacco)_(Solanaceae)_Plant.JPG
15. **Cannabis**: Cannabis sativa leaf. Photo: Wikimedia Commons, Marijuana.jpg https://commons.wikimedia.org/wiki/File:Marijuana.jpg
16. **Cannabis**: Charas (hashish). Photo: Wikimedia Commons, Pure Manali hashish (1 tolla = 10 g) (4142310413).jpg https://commons.wikimedia.org/wiki/File:Pure_Manali_hashish_(1_tolla_=_10_g)_(4142310413).jpg
17. **Cerbera thevetia**: Yellow oleander (Thevetia peruviana, pila kaner) fruit and seed. Photo: Wikimedia Commons, Thevetia peruviana - Fruits.jpg https://commons.wikimedia.org/wiki/File:Thevetia_peruviana_-_Fruits.jpg
18. **Cerbera thevetia**: Yellow oleander nuts. Photo: Wikimedia Commons, Thevetia peruviana MHNT.BOT.2007.27.24.jpg https://commons.wikimedia.org/wiki/File:Thevetia_peruviana_MHNT.BOT.2007.27.24.jpg
19. **Nerium oleander**: Nerium oleander (white or pink kaner). Photo: Wikimedia Commons, Nerium oleander flowers leaves.jpg https://commons.wikimedia.org/wiki/File:Nerium_oleander_flowers_leaves.jpg
20. **Hydrochloric acid (HCl)**: Hydrochloric acid (muriatic acid, spirit of salt), concentrated 35%. Photo: Wikimedia Commons, Hydrochloric acid 02.jpg https://commons.wikimedia.org/wiki/File:Hydrochloric_acid_02.jpg
21. **Nitric acid (HNO_3)**: Fuming nitric acid (aqua fortis). Photo: Wikimedia Commons, Nitric acid fuming.jpg https://commons.wikimedia.org/wiki/File:Nitric_acid_fuming.jpg
22. **Nitric acid (HNO_3)**: Fuming nitric acid. Photo: Wikimedia Commons, Fuming nitric acid 40ml.jpg https://commons.wikimedia.org/wiki/File:Fuming_nitric_acid_40ml.jpg

23. **Sulphuric acid (H_2SO_4)**: Sulphuric acid (oil of vitriol) charring cloth. Photo: Wikimedia Commons, Sulphuric acid on a piece of towel.JPG https://commons.wikimedia.org/wiki/File:Sulphuric_acid_on_a_piece_of_towel.JPG
24. **Sulphuric acid (H_2SO_4)**: Dehydration of sugar by concentrated sulphuric acid. Photo: Wikimedia Commons, Dehydration of sugar by sulfuric acid.png https://commons.wikimedia.org/wiki/File:Dehydration_of_sugar_by_sulfuric_acid.png
25. **Malathion**: Malathion. Structural formula drawn for these notes
26. **Parathion**: Parathion. Structural formula drawn for these notes
27. **DDT**: DDT (dichlorodiphenyltrichloroethane). Photo: CDC Public Health Image Library (US government), Public domain, via Wikimedia Commons https://commons.wikimedia.org/wiki/File:DDT_WWII_soldier.jpg
28. **DDT**: DDT sprayed over swamp water for mosquito (malaria) control, 1958. Photo: CDC Public Health Image Library (US government), Public domain, via Wikimedia Commons https://commons.wikimedia.org/wiki/File:DDT_spray_1958.jpg
29. **Lead tetroxide**: Lead tetroxide (Pb_3O_4, red lead, sindoor adulterant, minium pigment). Photo: Wikimedia Commons, Red lead.jpg https://commons.wikimedia.org/wiki/File:Red_lead.jpg
30. **Lead sulphide**: Lead sulphide (PbS, galena). Photo: Rob Lavinsky, iRocks.com, CC BY-SA 3.0, via Wikimedia Commons https://commons.wikimedia.org/wiki/File:Galena-40599.jpg
31. **Oxalic acid**: Oxalic acid (acid of sugar) crystals. Photo: Wikimedia Commons, Oxalic acid dihydrate.jpg https://commons.wikimedia.org/wiki/File:Oxalic_acid_dihydrate.jpg
32. **Carbolic acid**: Phenol (carbolic acid) crystals. Photo: Wikimedia Commons, Phenol (carbolic acid)04.jpg https://commons.wikimedia.org/wiki/File:Phenol_(carbolic_acid)04.jpg
33. **Calomel**: Mercurous chloride (Hg_2Cl_2, calomel) in its natural mineral form. Photo: Rob Lavinsky, iRocks.com, CC BY-SA 3.0, via Wikimedia Commons https://commons.wikimedia.org/wiki/File:Calomel-154907.jpg