MANUAL OF EMERGENCY FIRST AID AND ANTIDOTE TREATMENT OF CHEMICAL INJURIES IN THE INDUSTRIAL SETTING.

Dr Murlidhar V

Price: Rs 20

Participatory research in Asia (PRIA) 42, Tughlakabad Institutional Area, New Delhi - 110 062.
EMAIL: pria@sdalt.ernet.in
http://www.pria.org
Emergency First Aid and Resuscitation contributed by:

Dr Nobojit Roy
Consultant Surgeon
BARC Hospital, Mumbai

Comments given by:

Vijay Kanhere
Consultant
PRIA, New Delhi

Harsh Jaitli
Coordinator
COEH, PRIA, New Delhi

Dr N K Mehrotra
Ex-Dy Director. Industrial Toxicology Research Centre (ITRC), Lucknow.

Computer assistance:

Friends at Sabu Francis and Associates,
Vardhaman Market, Vashi - New Bombay.
Preface

Nagotane, Maharashtra blast in 1990...Bhopal, Madhya Pradesh MIC leak in 1984—Doctors did not know how to treat the affected victims. Doctors practicing around industries in big cities and around sprawling industrial estates, find it difficult to give first-aid to the victims injured due to chemicals.

In the orientation trainings on occupational health (over the past 3 years), doctors asked us regarding immediate treatment of chemically injured patients—they are not covered in the MBBS syllabus. Doctors around Lote industrial area (Maharashtra) and Aurangabad too came with a similar request.

Dr Murleddhar has painstakingly put together the present monograph on emergency first-aid and antidotal treatment for chemical injuries and Dr NK Mehrotra, Lucknow, with his vast experience in toxicology has scrutinized the same and we are thankful to them for their efforts.

As with any monograph or write ups, this will need many additions in future and it is expected that the doctors would give such inputs in the future.

The present monograph does not deal with standard first aid treatments e.g., for shock, pulmonary edema etc...because we have assumed that standard treatment in such cases are well known.

We will appreciate reader’s comments about the monograph. At a later date it will come out in a book form with improvements.

We hope that this monograph will prove to be useful in treating patients of industrial accidents.

Vijay Kanhere,
Consultant PRIA,
New Delhi.
July 1999.
TABLE OF CONTENTS:

PREFACE ........................................................................................................................................ 1

INTRODUCTION: .......................................................................................................................... 1

FIRST AID AT THE SITE OF ACCIDENT: .................................................................................. 1

PREPARATION ................................................................................................................................ 1
TRIAGE ......................................................................................................................................... 2
PRIMARY SURVEY ....................................................................................................................... 2
AIRWAY MAINTENANCE ............................................................................................................. 2
BREATHING AND VENTILATION ................................................................................................. 2
CIRCULATION WITH HEMORRHAGE CONTROL ........................................................................... 2
BLOOD VOLUME AND CARDIAC OUTPUT ....................................................................................... 2
Level of consciousness .................................................................................................................. 2
Pulse ............................................................................................................................................. 3
Bleeding ....................................................................................................................................... 3

DISABILITY (NEUROLOGIC EVALUATION) ............................................................................... 3

EXPOSURE .................................................................................................................................... 3

TRIAGE FOR CHEMICAL INJURIES ......................................................................................... 3

CRITERIA TO TRIAGE PATIENTS EXPOSED TO IRRITANT GASES ............................................ 3
CRITERIA TO TRIAGE PATIENTS BURNT WITH CORROSIVES .................................................. 3
INITIAL SITE: .............................................................................................................................. 4
DECONTAMINATION SITE: ............................................................................................................ 4
MAIN TRIAGE SITE: ..................................................................................................................... 4

TREATMENT PRINCIPLES : ........................................................................................................... 4

RESUSCITATION ........................................................................................................................... 4
AIRWAY ......................................................................................................................................... 4
BREATHING / VENTILATION / OXYGENATION ........................................................................... 4
CIRCULATION ............................................................................................................................... 4
URINARY AND GASTRIC CATHETERS ......................................................................................... 4
MONITORING ............................................................................................................................... 4
CONSIDER NEED FOR TRANSFER .............................................................................................. 5
GENERAL PRINCIPLES OF TREATMENT

SKIN : - CHEMICAL BURNS

INHALATION:

EYE CONTACT :

INGESTION :

CHEMICALS WHERE ACTIVATED CHARCOAL IS INEFFECTIVE:

INITIAL TREATMENT:

ANTIDOTES:

SPECIFIC EXPOSURES AND SPECIFIC TREATMENT :

GENERAL PRINCIPLES OF TREATMENT: [ FOR GASES LIKE AMMONIA, CHLORINE,
HYDROFLUORIC ACID, HYDROGEN SULPHIDE, NITRIC ACID, OXIDES OF NITROGEN SULPHUR
DIOXIDE AND PHOSGENE.]

CARBON MONOXIDE ( CO ) :

Treatment

METHAEMOGLOBINAEMIA

Causes:

Methylene Blue(A1)(Reducing Agent)

Dose:

CYANO- COMPOUNDS :

Treatment:

ORGANOPHOSPHOROUS PESTICIDES :

Additional first - aid measures :

Antidote :

Lab investigations:

CARBAMATE POISONING:

Synthetic Pyrethron and pesticides

ORGANOCHLORINE COMPOUNDS:

ALUMINIUM PHOSPHIDE:

METHANOL:

ARSENIC AND ITS COMPOUNDS:

Arsenic gas:

LEAD COMPOUNDS:

ORGANIC LEAD :

MERCURY AND ITS COMPOUNDS:

CADMIUM:

ZINC OXIDE:

MANGANESE:

CHROMIUM:

Treatment:

ALUMINIUM:

NICKEL CARBONYL:

Treatment:
INTRODUCTION:

In 1990, a blast occurred in the IPCL factory in Nagothane (100 miles from Mumbai) and 60 acutely burnt workers came nearly 10 hours late to Sion Hospital (Mumbai) without First Aid being given at the site of accident. Though such mishaps are highlighted in the press daily, many go unreported. Immediate relevant first aid is rarely given at the site and there are no round the clock first aid centers in the vicinity of such industrial belts. In such a scenario, the doctor who is available nearby is called to give his services. This manual is for such doctors who reside and practice near chemical industrial belts, and who may be called upon to attend an emergency case following a chemical accident. It is necessary that doctors should be informed before hand about the chemicals that are used in the industry surrounding them. They can visit the factories and request for Material Safety Data Sheets (MSDS) and also procure the disaster management plan that is usually brought out by the Government. It is to be noted that usually the victim comes with exposure to a mixture of chemicals, hence if prior knowledge is lacking regarding the chemicals he is exposed to it is impossible to diagnose a specific chemical toxicity in the emergency setting. In most cases supportive treatment is the basic line of action. The manual is also useful in areas like Mumbai, New Delhi etc. where patients come for treatment after being exposed to chemicals at the industries surrounding the cities and even from far away places, since at present there are very few first aid centers (equipped) surrounding the industrial belts in India. This manual would also dispel a common feeling among doctors that there is a magic antidote to many chemicals (the magic bullet like vaccines)—in reality there are very few antidotes, which are recognized worldwide.

The manual starts with common first aid treatments, followed by a brief information on specific chemicals, their relevant bio-chemical mechanisms (if known) and how they are countered by antidotes. Since ingestion of a chemical is rare in an industrial setting, it is given a cursory write-up. Ingestion as a mode of entry in an industrial setting can occur on swallowing spum laden with chemicals (daring the mechanism of removal of the substance from the lungs by the mucous-ciliary escalator system). Ingestion can be an additional factor in the poisoning when workers eat using contaminated hands. Most of the acute poisonings are due to inhalation and dermal absorption. The manual covers acute injuries and some chronic injuries due to exposure to chemicals and their specific treatments (if any). There are controversies in the use of many antidotes. Hence, most of the antidotal treatments referred to in this manual are non-controversial ones, having been standardized in many centers around the world. All specific treatments are referenced. The antidotes are classified as follows (based on WHO guidelines)

A. Required to be immediately available (within 30 Mts)
B. Required to be available within 2 hours.
C. Required to be available within 6 hours.

1. Effectiveness well documented.
2. Widely used but not yet universally accepted as effective, due to lack of adequate research data, and requiring further investigations concerning effectiveness or indications for use.
3. Questionable usefulness, as many data as possible regarding effectiveness should be collected.

E.g. administration of atropine in organophosphorous poisoning is classified as A1 (i.e. effectiveness is well documented and is required to be available immediately)

The manual does not give details of standard supportive treatment (e.g. for pulmonary Edema, Shock etc.) unless it is specifically different for the chemical toxicants. Similarly signs and symptoms of the poisoning are not given in this manual unless it is useful in understanding the biochemical action of the antidote. Biochemical mechanisms are mentioned as and where relevant to understanding of the actions of the antidote.

First aid at the site of accident.

It is unusual for the accident in the industrial setting to result in multiple casualties. Therefore, a MAJAX (Major Accident) protocol needs to be a part of the routine safety drill in all industries. It includes:

1. Preparation
2. Triage
3. Primary Survey (ABCs)
4. Resuscitation

PREPARATION

An action plan chalking out the team of people who will constitute an "emergency squad" to be summoned in case of an accident, is a part of the preparation. Linking with an appropriate facility and discussing a transfer and
transportation agreement, to minimize the transit time, (from accident to definitive care in a hospital) needs to be set up. Antidotes, first-aid equipment and resuscitation tools need to be checked on a regular basis and kept in optimal working condition.

TRIAGE

Triage is the sorting of patients based on the treatment and available resources to provide that treatment. Triage is rendered on the basis of ABC priorities as outlined below. Triage pertains to the sorting of patients in the field and the medical facility to which they are transported.

PRIMARY SURVEY

In the severely injured patient, logical sequential treatment priorities must be established based on overall patient assessment. The patient's vital functions must be assessed quickly and efficiently. Patient management must consist of a rapid primary evaluation, resuscitation of vital functions, a more detailed secondary assessment, and finally, the initiation of definitive care.

A  Airway maintenance
B  Breathing and Ventilation
C  Circulation with hemorrhage control
D  Disability: Neurologic status
E  Exposure

Airway maintenance

The airway should be assessed FIRST to ascertain patency. This rapid assessment for signs of airway obstruction should include inspection for foreign bodies and facial, mandibular, or tracheal fractures that may result in airway obstruction. Measures to establish a patent airway should protect the cervical spine. The chin lift or the jaw thrust maneuvers are recommended to achieve this task. The Pitfalls to watch out for are:

1. Foreign body in the airway.
2. Mandibular or maxillofacial fracture.
3. Tracheal or laryngeal disruption.

Breathing and Ventilation

Airway patency alone does not assure adequate ventilation. Adequate exchange of gases is mandatory to maximize oxygen transfer and carbon dioxide elimination. Ventilation involves adequate function of the lungs, chest wall and diaphragm. Each component should be examined rapidly.

The patient's chest should be exposed to assess ventilatory exchange in the lungs. Percussion may reveal the presence of air or blood in the chest. Visual inspection and palpation may reveal injuries to the chest wall that might compromise ventilation.

The most important conditions are:

1. Tension Pneumothorax
2. Flail chest
3. Open pneumothorax
4. Massive hemоторax

Circulation with hemorrhage control

Blood volume and cardiac output

Hemorrhage can be detected and rapidly treated. The two elements of observation that yield key information within seconds are:

Level of consciousness

When circulating blood volume is reduced, cerebral perfusion may be critically impaired, resulting in altered levels of consciousness. However, a conscious patient also may have lost a significant amount of blood.
Pulse
Pulses, usually the femoral or carotid, should be assessed bilaterally for quality and regularity. Rapid, thready pulses are early signs of hypovolemia, signifying the need of immediate resuscitation.

Bleeding
External, severe hemorrhage is identified and controlled IMMEDIATELY, by direct manual pressure on the wound. Tourniquets and hemostats should not be used because they crush tissues.

Pitfalls:
1. Intra-abdominal or intrathoracic injury
2. Fractures of the femur or pelvic
3. Penetrating injuries with arterial or venous involvement
4. External hemorrhage from any source

Disability (Neurologic Evaluation)
A rapid neurologic evaluation is performed at the end of the primary survey. It is done by the AVPU method:

A = Alert
V = Responds to Vocal stimuli
P = Responds only to Painful stimuli
U = Unresponsive

Pitfalls:
1. Head injury
2. Decreased oxygenation
3. Shock
4. Altered level of consciousness secondary to alcohol and/or other drugs.

Exposure
The patient should be undressed completely and checked for injuries.

Triage for chemical injuries
The fundamental principles of triage used for patients of trauma are also applicable to victims of chemical accidents.

Criteria to triage patients exposed to irritant gases

Group 1 : Life threatening injury; injured persons with intense exposure to strong irritants through the respiratory route, leading to cough, respiratory difficulty and general influence.

Group 2 : Serious injury; strong irritant causing severe cough, no general influence.

Group 3 : Moderate or slight cough and no general influence.

Criteria to triage patients burnt with corrosives

Group 1 : Life threatening injury: Dermal and full thickness skin burns > 50% of the body surface area—serious injury.

Group 2a : Full thickness injuries on 10 – 50% or dermal injuries on 20 – 50% of body.

Group 2b : Moderate injury: Full thickness injuries on 2 – 10% or dermal injuries on 10 – 20% of body.

Group 3 : Slight injury: Full thickness injuries on 2% of body or dermal injuries on less than 10% of the body.
INITIAL SITE:
The basic principle of paying immediate attention to airway, breathing and circulation (ABC) of emergency care is followed and decisions are made regarding the need for admission accordingly.

DECONTAMINATION SITE:
Here the victim of exposure is transferred away from the initial site of exposure. Apart from isolation, the site requires the health personnel engaged in decontamination to wear protective appliances such as full masks, gloves, protective coats with hoods, and rubber boots.

Victims have to be undressed completely since clothes may harbour residual toxins.

MAIN TRIAGE SITE:
Depending upon the seriousness of the patients they are sent to any of the following definitive-care sites:

1. Severe casualties site: non-breathing adults and children.
2. Moderate casualties site: breathing adults with systemic problems.
3. Children casualties site: Children < 10 years requiring admission.
5. Light Casualties site: Ambulatory patients who walk up here from the decontamination site.

TREATMENT PRINCIPLES:

RESUSCITATION

AIRWAY
The airway should be protected in all patients and secured in those patients whose ventilation is not adequate. The jaw thrust or chin lift may suffice in some cases. The use of a nasopharyngeal airway may initially establish and maintain airway patency in the conscious patient.

If the patient is unconscious and has no gag reflex, an oropharyngeal airway may be helpful.

BREATHING / VENTILATION / OXYGENATION
Definitive control of the airway in patients who have compromised airways due to mechanical factors, who have ventilatory problems or who are unconscious is achieved by endotracheal intubation, either nasally or orally.

A tension pneumothorax compromises ventilation, and if suspected, chest decompression should be accomplished immediately. Also, every injured patient should receive supplemental oxygen therapy.

CIRCULATION
Two large-caliber intravenous catheters (IVs) should be established. First, at least 10 ml blood should be withdrawn for basic hematological investigations, including blood grouping and crossmatching. Vigorous intravenous fluid therapy should be initiated with Ringer Lactate solution.

URINARY AND GASTRIC CATHETERS
Urinary output is a sensitive indicator of the volume status of the patient. It is contraindicated in the following situations:

1. Blood at the penile meatus
2. Blood in the scrotum
3. Highly inflamed prostate

All of the above indicate urethral injury. A suprapubic catheter is preferable in these situations.

Gastric tubes reduce stomach distention and decrease the risk of aspiration.

MONITORING
Quantitative improvement of physiological parameters is best assessed by the following:

1. Pulse Oximetry: It measures the Oxygen saturation of hemoglobin.
2. The blood pressure (though it may be a poor measure of actual tissue perfusion)
3. ECG monitoring

CONSIDER NEED FOR TRANSFER

After life saving measures are initiated the patient must be evaluated for transfer to the nearest, appropriate facility for definitive care.

General principles of treatment

General principles of treatment have been evolved by analysing the route through which the body is subjected to the toxic effects of chemicals. There are four main routes of exposure (1) Skin, (2) inhalation, (3) Eye, (4) Ingestion.

SKIN > Chemical Burns

1. Remove all contaminated clothing, including shoes, watch, jewelry contact lenses etc.
2. Drench (irrigate) the affected area with running water for at least 15 - 20 minutes. This is important since chemicals continue to act on the skin and delayed burns will result e.g. exposure to hydrofluoric Acid (HF< 20% Conc.) causes delayed burns.
3. Check for burns. Standard treatment is followed for burns (volumes of Ringer lactate solutions infused as per Parkland Formula).

Parkland formula: 4ml of Ringer lactate % burns x body weight (max 50 kg) to be given in the initial 24 hours, half of it to be given in the first 8 hours.

Locally 1% silver sulfadiazine is used for all the burnt areas and covered by antiseptic dressings. In the following cases specific treatment is to be followed:

1. Hydrofluoric Acid Burns:
   - Solutions of 20% may not produce erythema up to 24 hours and concentrations of 20-50% produce effects within 1-8 hours.
   - Inhalation burns should be assumed in burns:
     a) Involving HF concentration of 50% or over.
     b) Of head and neck
     c) Of greater than 55 of body surface.
     d) Where clothing is soaked and there is delay in removing clothing.
     e) Occurring in confined spaces.
     - Strong intravenous analgesia
     - Tanning of nails / splitting of nails
     - Locally 1% to 2% benzylalkonium Chloride (Zephiran) or iced 25% MgSO4 or 10% Calcium gluconate gel is applied repeatedly. If pain persists local injection of 10% Ca gluconate. Dose = 0.5ml of 5% Ca gluconate per cm².

10% Ca gluconate is also infused intravenously.

- (Note: Ca gluconate gel can be prepared by mixing K - Y jelly with 10% Ca gluconate solution). 75 ml K - Y jelly plus 25 ml calcium gluconate (10%)
- Surgical gloves filled with gel is useful for hand burns
- Areas covered with gel redressed every 4 hours.
- For conc more than 20% to fingers/local infiltration Dose 0.5ml of 55 calcium gluconate per cm² using 27 or 30 gauge needle subcutaneously.
- Use of 1% calcium gluconate eye drops every 2-3 hours for eye injuries.
- IV calcium treatment should not wait all blood tests are available. In case IV infusions are not available then tablets can be give orally.
- In case of extremity burns, if pain persists intra arterial infusion can be tried if this also fails, then surgical excision.

Treatment of Systemic toxicity (in case of HF burns):

1. Pre-Hospital give calcium Gluconate tabs every 2 hours.
2. 20 ml of 10% calcium Gluconate I.V before levels of Ca ++ are known.
3. Monitor Ca ++ levels and ECG
4. I.V infusion of 40 ml of 10% calcium gluconate over 1 hour.
5. Serum Ca ++ level; if still not maintained, consider immediate burn excision and or haemodialysis.
1. In case of phenol burns, polyethylene glycol (PEG) 300 or 400 in 2:1 mixture of PEG and alcohol is used on the burn area.
2. Chronic Acid is to be reduced to its trivalent form by frequent washing with fresh aqueous solution of 10% Ascorbic Acid.
3. White Phosphorus: The skin should be continued to be kept wet and mechanical debridement is done. (Note: White P is quoted on coming in contact with air).

**INHALATION:**
1. Remove victim from the site of exposure.
2. Check breathing and pulse. Start cardiopulmonary resuscitation (CPR); if needed set up IV infusion.
4. Shift to hospital.

**EYE CONTACT:**
1. Wash eyes thoroughly with water for at least 10 min.
2. In case of HF exposure use 1% Ca gluconate drops every 2 - 3 hours. Eye is to be padded.
3. If corneal injury is suspected homatropine drops are to be used to relax the pupillary sphincter.

**INGESTION:**
1. Avoid oral feeding in cases of altered sensorium or coma.
2. Forced vomiting or gastric lavage should not be attempted for petroleum distillates (volatile hydrocarbons), corrosives and convulsants. Milk helps in the absorption of fat soluble substances and is only indicated in corrosives.

Syrup of ipecac, a natural product, is a very effective emetic and acts within 30 Mins. The adult dose is 30 ml. The other emetics are saline water and apomorphine (parenteral). Apomorphine can cause CNS depression as a side effect. Value of gastric lavage 72 hrs. after poisoning is extremely poor. If saline is used as an emetic, not more than 100 ml is given since large quantities pushes the gastric contents into the duodenum (Note: Syr. of ipecac is not manufactured in India).

Activated Charcoal: A preparation of activated charcoal is available with LOCOST (Vadodara). It is mixed with water and the adult dose is 50 - 100 GMS. Emesis and gastric lavage decrease the effect of activated charcoal. A saline cathartic or sorbitol should accompany the administration of activated charcoal to enhance GI transit time and possibly drug elimination.

**Chemicals where activated charcoal is ineffective:**

<table>
<thead>
<tr>
<th>Chemicals</th>
<th>Pesticides</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alkali</td>
<td>DDT</td>
</tr>
<tr>
<td>Boric Acid</td>
<td>Malathion</td>
</tr>
<tr>
<td>Boroa</td>
<td>N - methyl Carbamate</td>
</tr>
<tr>
<td>Cyanide</td>
<td></td>
</tr>
<tr>
<td>Ethanol</td>
<td></td>
</tr>
<tr>
<td>Lead</td>
<td></td>
</tr>
<tr>
<td>Mercury</td>
<td></td>
</tr>
<tr>
<td>Mineral Acids</td>
<td></td>
</tr>
<tr>
<td>Petroleum Distillates</td>
<td></td>
</tr>
<tr>
<td>Water Insoluble Compounds</td>
<td></td>
</tr>
</tbody>
</table>

Note: Sodium Formaldehyde sulfonate (20 g amp) is an effective neutralising agent for mercury salts.

**INITIAL TREATMENT:**

After first-aid, the initial treatment consists of clinical evaluation, administration of specific antidote if any and supportive treatment before shifting to a major tertiary care hospital.
ANTIDOTES:

Antidotes are therapeutic substances that in a specific way can prevent, eliminate or at least reduce the harmful effects of certain poisons. Some have intrinsic activity and may cause adverse reactions. They should be given as early as possible during the course of poisoning and in an adequate dose.

In most poisoning cases, symptomatic and supportive care is the mainstay of treatment. Without a history of occupational exposure, it is nearly impossible to identify the chemical toxin by symptoms alone e.g. cyanosis can be due to respiratory failure or due to methaemoglobinemia. Also many clinical signs (like miosis in organophosphorous poisoning) occur later and immediate treatment would be delayed. Hence, occupational history and prior knowledge of workplace, working atmosphere and the list of hazardous chemicals is extremely important when giving antitodal treatment.** The occupational physician should visit the workplace!

Antidotes can be classified as:

(a) Immediately available, available within 2 hours or

(b) available within 4 - 6 hours.

Based on the mechanism of action, antidotes can be classified into 4 groups.

1. Formation of inert complexes by binding the poison.
   (a) In the GI tract: e.g. activated charcoal.
   (b) Formation of chelates e.g. demercaprol (BAL) for Arsenic.
   (c) Formations of other complexes e.g. hydroxycobalamine for cyanide.

2. Elimination or modification of toxicity by influencing the metabolism of the poison. e.g. ethanol occupies alcohol dehydrogenase and hence prevent the metabolism of methanol and ethylene glycol.

3. Antagonizing the action of the poison at receptor sites.
   e.g. atropine in handling cholinergic crises caused by organophosphorous pesticides.

4. Countering Chemical Injury:
   e.g. Methylen blue in Methaemoglobinemia

Specific exposures and specific treatment:

General Principles of treatment: [For Gases like Ammonia, Chlorine, hydrofluoric acid, hydrogen sulphide, nitric acid, oxides of nitrogen, sulphur dioxide and phosgene.]

1. The agent inhaled cannot be washed away or otherwise removed, unless, when only the skin is involved.
2. Arterial blood gases are of primary importance in determining the extent of the damage.
3. Supportive measures include suctioning (intubation may be required), volume cycle ventilator (positive end expiratory pressure (PEEP), steroids and antibiotics.

For gases with delayed action on the lungs:

1. Phosgene may cause pulmonary edema often seen several hours after exposure. Treatment is similar to that for irritant gases except PEEP is extremely useful.
2. A unique property of hydrogen sulphide (H₂S) is that it can kill by directly paralyzing the respiratory center within seconds of heavy exposure. It also causes olfactory fatigue, making its presence undetectable by persons exposed to smaller amounts. Here too delayed pulmonary edema can occur. Hyperbaric oxygen is valuable in cases of H₂S poisoning.
3. Oxides of nitrogen (nitrogen dioxide and nitrogen tetroxide) also have a delayed action on the lungs. A worker may be exposed for 8 hours during the day with no ill effects and later while at home may lapse into pulmonary edema.

CARBON MONOXIDE (CO):

8
CO is readily absorbed by the lungs. It combines with haemoglobin, myoglobin and cytochrome oxidase system and affects cellular respiration. The affinity of CO for haemoglobin is 250 times that of oxygen.

**Treatment**

Apart from routine measures high concentration of oxygen (A1) should be administered. Also hyperbare oxygen is useful.

**Methaemoglobinemia**

The haem ion is oxidised into ferric state, which is called methaemoglobin. Normal 1%

**Causes:**

1. Inherited defect
2. Nitrates and Nitrates (nitrates are converted to nitrates in the GI tract)
3. Analine
4. Acetanilide and phenacetin
5. Aromatic amino compounds
6. Cyanogen
7. Chlorates
8. Toluidine

Individuals chronically exposed to such chemicals may have blueness of lips with methaemoglobin levels in the range of 10%. They may not have any other overt effects. Significant symptoms are present if the levels in blood rise to more than 40%.

In addition to causing methaemoglobinemia, most of the above chemicals cause a Heinz-body haemolytic anaemia. This process is characterized by oxidative denaturation of haemoglobin, leading to the formation of pancellate membrane-bound red cell inclusions known as Heinz bodies. Oxidative damage to the red cell membrane also occurs. Haemolysis will be most prominent part of the clinical picture in individuals with Glucose-6-Phosphate dehydrogenase (G6PD) deficiency.

**TREATMENT**

**Methylene Blue (A1) (Reducing Agent)**

**Dose:**

1-2 mg/kg/dose, 6-10.2 ml/kg of 1% solution. I.V. over 5 minutes followed immediately by 15-30 ml. Fluid flush minimises local pain. May be repeated in 30-60 minutes.

**NOTE:**

1. Do not use in case of any nitrile treatment after CN poisoning.
2. Haemolysis will occur in patients having G6PD deficiency.
3. Higher concentration of methylene blue can itself cause methaemoglobinemia.
4. Methaemoglobinemia caused due to chlorates does not respond to methylene blue.

**Cyano- compounds:**

Inorganic cyanides are readily hydrolysed by water and decomposed by mineral acids and carbon dioxide to form hydrocyanic acid.

Similarly nitriles when heated decompose and release hydrogen cyanide.

**Acute exposure causes death by asphyxia when inhaled or when absorbed through skin.** Its toxic properties result from its ability to form complexes with heavy metal ions which inhibit enzymes required for cellular respiration, primary cytochrome oxidase. This prevents the uptake of oxygen resulting in cellular hypoxia. Part of the cyanide is converted into less harmful thiocyanate and is excreted.

**Treatment:**

1. Hyperbaric oxygen (C2) if available.
2. Nitriles (A2) inhaled for 30 seconds every minute alternately with 100% oxygen. New ampoule should be used every 3 minutes. It produces about 3% methaemoglobinemia which attaches itself forming harmless cyanometh-haemoglobin.
Later sodium thiosulphate (A1) is given that is convert cyan-meth-haemoglobin to thiocyanate by binding with CN that is excreted.

**Dose:** Adults: 12.5 g (50 ml 25% solution I.V.)

Children: 1.65 ml / kg (25% solution I.V.)

**Note:** Excess thiocyanate in the body cause muscle weakness and paralysis.

Hydroxy cobalamine (A1) or dicobalt edatate (A1) are also good antidotes.

**Dose of Hydroxy cobalamine:** 4 gm I.V. in 5% dextrose. Repeat in case of massive poisoning. 4 - 8 g of thiosulphate is co-administered.

**Dose of Dicobalt edatate:** Adults: 1 - 2 ampoules (300 - 600 mg) I.V. over 1 - 5 min. A third ampoule can be injected I.V. over 1 - 5 min. A third ampoule can be injected I.V. if there is no sufficient clinical improvement.

**Note:** Dicobalt edatate is not to be given in mild cases.

**Organophosphorous pesticides:**

70 - 80% of the poisoning is accidental and is common in agricultural and industrial workers. The mode of entry is inhalational and dermal and occasionally accidental in workers. It crosses the blood - brain barrier. They are potent inhibitors of acetylcholinesterase and pseudocholinesterase. The inhibition of these enzymes is due to irreversible binding of phosphate radicals of organophosphates to active sites of enzymes. In case of carbamates this binding is reversible. The toxicological effects are due to accumulation of acetylcholine at synapses resulting in initial stimulation followed by paralysis of neurotransmission at cholinergic synapses. The cholinergic synapses are present in CNS, somatic nerves, autonomic ganglion, parasympathetic nerve endings and some sympathetic nerve endings like in sweat glands.

The signs and symptoms which are due to muscarinic and nicotinic effects appear within a few minutes to a few hours (av.: 6-8 hrs.). The critical period is the first 24 hrs.

**Additional first - aid measures:**

1. Keep the patient in a quiet room in case of convulsions.
2. Cardiac monitoring.

**Antidote:**

Atropine (A1) 2.5 mg I.V in adults and in children 0.05 mg / Kg I.V slowly, every 5-10 min till early signs of atropinisation occur (clearing of rales, drying of pulmonary secretions) Note: tachycardia and pupillary dilation are not very good indicators.

(b) Chymes (cholinesterase enzymes reactivators.)

Pralidoxime (A1) is given to all patients with severe toxicity (muscle weakness and respiratory depression). It rapidly ameliorates muscle weakness, fasciculations and coma.

Dose: 1-2 GMS in adults or 25-50 mg/kg in children over 5-10 minutes I.V. The dose may be repeated after 1 hour.

Drugs contraindicated: Opiates, phenothiazines, parasympathomimetic drugs.

**Lab investigations:**

*Do not wait for cholinesterase estimation before starting treatment.

(a) Latent poisoning (Serum Cholinesterase activity 50-90% of normal value):- No treatment.

(b) Mild (20-50% of normal value):- Needs treatment.

(c) Moderate (10-20% of normal value)

(d) Severe (Serum Cholinesterase less than 10% of normal)
Carbamate Poisoning:

Atropine - (A1) is a specific antidote. Do not give PAM in carbamate poisoning except in case of combined organophosphate and carbamate poisoning.

**Synthetic Pyrethrons and pesticides**

There is no specific antidote. Dermatitis (bullous) can be treated by application of vitamin E (tocopherol acetate) (A3).

**Organochlorine Compounds:**

There is no specific antidote. Convulsions can be treated by diazepam (5-10 mg I.V. over 2-3 minutes) - Max dose:
Adults: 30 mg, Children: 10 mg.

If seizures are uncontrolled Phenobarbitone (loading dose 10mg/ kg) can be given. Drugs Contraindicated: Adrenaline, Atropine as they can induce arrhythmias.

**Aluminium Phosphide:**

No specific antidote is available. Since phosphine is excreted through breath and urine, adequate hydration and renal perfusion by low dose dopamine 4-6 μg/kg/min is to be used. Shock and pulmonary edema are treated by standard measures.

Magnesium Sulphate can be used as an antioxidant and is also useful as an anti-arrhythmic and anaphylactic agent.

A dose of 1 gm of MgSO4 I.V. stat is given followed by 1g for next 2 hours and finally 1.0-1.5g after 4-6 hours for 3-5 days or final outcome of these patients.

**Methanol:**

It is well absorbed following inhalation. The metabolism is principally in the liver, by the metalloenzyme alcohol dehydrogenase to produce formaldehyde. Thereafter formaldehyde is converted to formate by enzyme systems like aldehyde dehydrogenase. Formic acid formed is associated with metabolic acidosis and retinal toxicity. It also inhibits cytochrome oxidases and causes optic neuritis.

Treatment measures:

1. Correction of acidosis by IV sodium bicarbonate (preferably with ABG monitoring)

2. Ethanol therapy (A1) should be started in all cases before blood levels are available. The usual dose given is aimed to maintain a blood level of 100 mg / dl of ethanol. Therapy should begin with a loading dose of 0.8 g/ kg followed by maintenance dose of 130 mg / kg / hr. When dialysis is performed, maintenance dose of 250 - 300 mg / kg / hr are required, as ethanol is also dialysed along with methanol.

1. Folinic acid (5-formyl tetrahydrofolate acid) (B1) enhances the elimination of formic acid. Intravenous folate (Folinic or folic acid) at 50-70 mg, every 4 hours for at least 24 hours can be given.

2. 4-methylpyrazole (A1) which inhibits alcohol dehydrogenases is a suitable adjunct to ethanol. Also depression, which is common with ethanol will not be a problem.

**Arsenic and its compounds:**

\[
\begin{align*}
\text{Arsenic} & : 3.4 A. \quad 1.9 L
\end{align*}
\]
Arsenic trioxide gets into the body through inhalation. Over 23% of the particles are larger than 4 µm. Particles of this size get deposited in the upper ciliary tract, cleared by the mucociliary system and swallowed, thus entering by GI route.

Arsenic acts through the formation of covalent bonds with the sulphur atom of mercapto-groups (also called sulphhydryl or thiol) in the body. This is the only biological pathway that arsenic can enter a physiological reaction. It blocks the pyruvate oxidase and α-glutathione oxidase systems.

**Arsenic gas**

Arseine is one of the most potent intravascular haemolysins. It may be difficult to detect the causal relation of workplace arsine exposure to an acute haemolytic episode. Death can occur directly due to complete loss of RBC's (A haematocrit of zero has been reported). Acute renal failure can occur due to the massive release of haemoglobin into the circulation. At higher concentration Arsine can cause pulmonary edema and renal failure due to direct action.

**Treatment**

Arsenic compounds can be chelated by BAL and penicillamine.

Dose: *British Anti-Lewisite* (BAL) (Dimercaprol) (B3)

Adults: 3 - 5 mg / kg. Every 4 - 6 hours by deep IM injection for the first 2 days, and then every 12 hours for a week.

Children: 3 - 5 mg / kg /dose by deep IM injection.

**Note:**

1. Do not give BAL to patients allergic to peanuts.
2. Persistent hypopyrexia can occur.
3. Maintain the urinary pH alkaline to prevent dissociation of metal - BAL complex.
4. BAL may cause haemolysis in patients with G6PD deficiency.
5. *In cases of Arsine poisoning BAL is not effective. Treat with exchange transfusion.*

**Lead Compounds:**

Lead dust and compounds are absorbed after inhalation. Deposition in the lungs is maximal (63%) at particle size 1 µm and minimal (39%) at 0.1 µm for resting persons. Larger particles are deposited in the upper respiratory tract and cleared or swallowed. A crude estimate of the percentage of inhaled lead reaching the blood stream would be about 30 - 40%.

All enzyme systems are potentially susceptible to heavy metals. The biochemical interactions of Lead with sulphhydryl groups are of great toxicological importance. Lead also has a high affinity for amines and simple amino acids.

During haem synthesis lead inhibits ALA dehydrogenase initially and coproporphyrigen dehydratase latter. Basophilic stippling of the erythrocytes is probably made up of fragments of deranged mitochondria, microsomal remnants and RNA.

**Treatment**

1. Convulsions can be controlled by diazepam.
2. In lead encephalopathy, combined chelation with BAL (C2) and CaNa₂EDTA (C2) for 5 - 7 days. This is followed by a course of oral penicillamine.

Dose: *British Anti-Lewisite* (BAL) (Dimercaprol) (B3)

Adults: 3 - 5 mg / kg. Every 4 - 6 hours by deep IM injection for the first 2 days, and then every 12 hours for a week.

Children: 3 - 5 mg / kg /dose by deep IM injection.

Edetate Calcium Disodium: 30 - 50 mg / kg / day or 500 - 1000 mg / m² / day by deep/IM injection or slow I.V. infusion in 2 - 3 divided doses per day for 5 days, may be repeated for a second course after a minimum of 2 days.
Adults: Max: 2g
Children: Max: 1gm. (75 mg / kg / kg for encephalopathy)
Dilute 1 ampule (1 g) in 250 - 500 ml of isotonic saline or 5% dextrose. Infuse I.V. slowly over 15 - 20 min or by continuous infusion over 4 - 6 hours.

Note: Toxicity of EDTA is due to renal tubular necrosis and chelation of other metals.
D - Penicillamine (C2)
Dose: Adults: 250 mg orally 4 times a day half an hour before meals for 5 days or 20 - 40 days depending on severity.
Children: 20 mg / kg / day in 2 divided doses before meals. Maximum: 1 gm / day.

DMSA (2,3 dimercaptoprohecinic acid) (B3) has advanced therapeutic options in lead poisoning. It is less toxic and does not cause depletion of body stores of essential cations such as zinc, copper and iron.

Dose: Adults: 30 mg / kg / day in 3 divided doses for 5 days followed by 20 mg / kg / day in 2 divided doses for 14 days.
Children: Initial dose is 10 mg / kg or 350 mg / m2 every 8 hours for 5 days, then every 12 hours for next 14 days.

Note:
1. Monitor liver function.
2. DMSA can be mixed with food.
3. DMSA can be given after a course of BAL / EDTA after an interval of 4 weeks.

Organic lead:
Alkyl lead (Tetra-ethyl lead) poisoning: Only symptomatic treatment, chelating agents have no proved role.

Mercury and its compounds:
Elemental mercury and mercurous compounds are readily absorbed from the alveoli of the lungs and are oxidised to mercuric salts. Inhalation results in 80% deposition and retention.

Treatment of inorganic mercury poisoning:
D-acetyl penicillamine (C3):

Dose: Adults: 250 - 500 mg orally, 4 times a day for 6 - 10 days, totaling 20 - 30 mg / kg. In case of prolonged therapy give for 1 - 2 weeks intermittently.

D-Penicillamine (C2):

Dose: Adults: 250mg, orally 4 times a day half an hour before meals for 5 days or 20 - 40 days depending on severity.
Children: 20 mg / kg / day orally in 2 divided doses.

Note:
1. In patients, not able to take oral chelators, give BAL (except in associated liver failure).
2. There is no specific antidote for poisoning due to organic mercury compounds.

Cadmium:
Depending upon the size of the particles, 10% to 50% of the inhaled cadmium is deposited in the alveoli.

The absorption of the retained Cadmium depends on its chemical form. For CdO it is 60%. Acute inhalation injury due to freshly generated cadmium fumes may be caused after several hours. Symptoms are similar to metal
fume fever and a significant decrease in PFT.

Cadmium accumulates in the renal cortex giving rise to a functional lesion involving the proximal tubule giving rise to tubular type of proteinuria (β2-microglobulin, retinal binding protein etc.).

Sometimes even high molecular weight proteins (suggesting glomerular dysfunction) like albumin, transferrin, IgG etc. are excreted.

This may be associated with amino-aciduria, glycosuria and calcuria.

Treatment:

1. Symptomatic.
2. Vitamin D in cases of osteomalacia
3. Unithiol (DMPS) (C3)
   Dose: 5 mg / kg 50% solution IM of SC, 3 - 4 times a day during the first 24 hours, 2 - 3 times a day on day 2 and once or twice daily on subsequent days.
   
   Note: Enhances urinary elimination of Pb, Hg, Cu, Zn.
4. Edetate Calcium Disodium: (C3) 30 - 50 mg /kg / day or 500 - 1000 mg / m2 / day by deep IM injection or slow I.V. infusion in 2 - 3 divided doses per day for 5 days, may be repeated for a second course after a minimum of 2 days gap.
   
   Adults: Max: 2g
   
   Children: Max: 1gm. (75 mg / kg / kg for encephalopathy)
   
   Dilute 1 ampoule (1 g) in 250 - 500 ml of isotonic saline or 5% dextrose. Infuse I.V. slowly over 15 - 20 min or by continuous infusion over 4 - 6 hours.

**Zinc Oxide:**

Inhalation causes metal fume fever with severe respiratory irritation. Malaise, chills and fever (101 - 102 F) may occur 4 - 6 hours after exposure. It can also lead to pulmonary edema.

Treatment:

1. Supportive treatment
   
   Edetate Calcium Disodium: (C3) 30 - 50 mg /kg / day or 500 - 1000 mg / m2 / day by deep IM injection or slow I.V. infusion in 2 - 3 divided doses per day for 5 days, may be repeated for a second course after a minimum of 2 days gap.
   
   Adults: Max: 2g
   
   Children: Max: 1gm. (75 mg / kg / kg for encephalopathy)
   
   Dilute 1 ampoule (1 g) in 250 - 500 ml of isotonic saline or 5% dextrose. Infuse I.V. slowly over 15 - 20 min or by continuous infusion over 4 - 6 hours.

**Manganese:**

Inhalation of dust or fumes is the major route of entry. The term manganism is usually referred to the chronic neurological disorder caused by inhalation of manganese laden dust.

Manganese neurotoxicity results from the enhanced auto-oxidation of dopamine by a higher valency Mn 3+ ion with increased generation of cytotoxic free radicals. Mn 3+ complexes are formed in the brain and catecholamines are destroyed.

Treatment:

1. Supportive.
2. Levodopa (controversial) (C3)
3. Vitamin E (Not proven yet) (C3)
4. For acute exposure, Ca- EDTA can be used. (C3)
   
   Edetate Calcium Disodium: 30 - 50 mg /kg / day or 500 - 1000 mg / m2 / day by deep IM injection or slow I.V.
infusion in 2 - 3 divided doses per day for 5 days, may be repeated for a second course after a minimum of 2 days gap.

Adults: Max: 2g.
Children: Max: 1gm. (75 mg / kg / kg for encephalopathy)
Dilute 1 ampoule (1 g) in 250 - 500 ml of isotonic saline or 5% dextrose. Infuse I.V. slowly over 15 - 20 min or by continuous infusion over 4 - 6 hours.

Chromium:

Cr³⁺ is more hazardous than Cr⁶⁺. Compounds of Cr³⁺ is poorly absorbed from the GI tract and also form stable complexes with proteins in the superficial layers of the skin. They do not cause sensitization. The chrome ulcers are also cause due to Cr³⁺ Similarly they cause GI bleed and lung cancer.

Treatment:

Ascorbic acid (10%) is used to reduce the hexavalent chromium to Cr⁶⁺.(see also pg. 7). Skin cuts and abrasions however slight should be cleaned and treated with 10% sodium EDTA ointment. Together with the use of frequent renewed impervious dressing rapid healing occurs of any ulcer that may develop. Although EDTA does not chelate Cr³⁺ at room temperature it reduces it to Cr⁶⁺ and the excess Cr⁶⁺ is chelated. An ointment of EDTA can also be used to prevent nasal septum perforation.

Aluminium:

1. Inhalation of aluminium fumes (bauxite) cause both obstructive and restrictive pulmonary disease, hence will need both lung physiotherapy as well as bronchodilators.
2. Systemic toxicity causing neurological syndrome will have to be treated with chelating agent desferoxamine (C 1).
3. Vitamin D and calcium to treat osteomalacia
4. Iron deficiency to be treated.
Nickel carbonyl:
After acute exposure initial symptoms can be mild nausea, chest pain and dyspnoea. These symptoms disappear after a few hours. After 12 to 36 hours and occasionally as long as 5 days after exposure, severe pulmonary symptoms develop. Human fatalities have occurred 4 to 13 days after exposure from diffuse interstitial pneumonitis and cerebral edema.

**Treatment:**
- Oxygen (preferably humidified)
- Rest
- Corticosteroids and prophylactic antibiotics
- Sodium Diethylthiocarbamate (DTC) has been suggested as an antidote—further research needs to be done.
Classified lists of antidotes and other agents.

Group 1: List of antidotes
Group 2: Agents used to prevent the absorption of poisons, to enhance their elimination, or to treat symptomatically their effects on bodily functions.
Group 3: Other useful therapeutic agents for the treatment of poisonings.
Group 4: List of antidotes and therapeutic agents considered obsolete.

The antidotes listed in groups 1 and 2 are considered useful in the treatment of acute human poisoning and their availability in terms of urgency of use may be classified as follows:

- A: required to be available immediately (within 30 minutes)
- B: required to be available within 2 hours.
- C: required to be available within 6 hours.

Their effectiveness in practice can be classified as follows:

<table>
<thead>
<tr>
<th></th>
<th>Effectiveness</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Effectiveness well documented, e.g. reduction of lethality in animal experiments and reduction of lethality or of severe complications in human poisoning.</td>
</tr>
<tr>
<td>2</td>
<td>Widely used but not yet universally accepted as effective, owing to lack of research data and requiring further investigations concerning effectiveness or indications for use.</td>
</tr>
<tr>
<td>3</td>
<td>Questionable usefulness; as many data as possible regarding effectiveness should be collected.</td>
</tr>
</tbody>
</table>

The classification in terms of urgency of availability (A, B, C) or proven effectiveness (1, 2, 3) is given next to the main indication for the antidote. The classification is also given in the right-hand column of Group 1 list when an antidote has other possible applications. If there is doubt as to the classification of the antidote, the lower score is always given, e.g. B2 instead of A1.
<table>
<thead>
<tr>
<th>Antidote</th>
<th>main indication or pathological condition</th>
<th>Other possible applications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetylcystine</td>
<td>paracetamol (B1)</td>
<td>theophylline (B1)</td>
</tr>
<tr>
<td>N-acetyl penicillamine</td>
<td>mercury (inorganic and vapour) (C3)</td>
<td>calcium antagonists (B3)</td>
</tr>
<tr>
<td>atropine</td>
<td>cyanide (A2)</td>
<td>malignant-neuropathic syndrome</td>
</tr>
<tr>
<td>benzyl penicillin</td>
<td>organophosphorous compounds</td>
<td>aluminium (C2)</td>
</tr>
<tr>
<td>β-blockers (β₁ and β₂, preferentially short acting)</td>
<td>And carbamates (A1)</td>
<td>chloroquine (A2)</td>
</tr>
<tr>
<td>calcium gluconate or other soluble calcium salts</td>
<td>amanitin (B3)</td>
<td>gold (C3), mercury inorganic (C3)</td>
</tr>
<tr>
<td>dantralene</td>
<td>β-adrenergic agonists (A1)</td>
<td>methanol (B3)</td>
</tr>
<tr>
<td>deferoxamine</td>
<td>H₂, fluorides and oxalates</td>
<td>Methanol, disulfiram (B2)</td>
</tr>
<tr>
<td>diazepam</td>
<td>drug-induced hyperthermia</td>
<td></td>
</tr>
<tr>
<td>cobalt edetate</td>
<td>iron (B1)</td>
<td></td>
</tr>
<tr>
<td>digoxin-specific antibodies</td>
<td>organophosphates (A2)</td>
<td></td>
</tr>
<tr>
<td>dimercaprol</td>
<td>digoxin/digitoxin (A1)</td>
<td></td>
</tr>
<tr>
<td>4-dimethyl amino phenol</td>
<td>arsenic (B3)</td>
<td></td>
</tr>
<tr>
<td>edetate calcium disodium</td>
<td>cyanide (A1)</td>
<td></td>
</tr>
<tr>
<td>ethanol</td>
<td>lead (C2)</td>
<td></td>
</tr>
<tr>
<td>fumarazone</td>
<td>methanol, ethylene glycol (A1)</td>
<td></td>
</tr>
<tr>
<td>folinic acid</td>
<td>benzodiazepines (B1)</td>
<td></td>
</tr>
<tr>
<td>glucagon</td>
<td>folinic acid antagonists (B1)</td>
<td></td>
</tr>
<tr>
<td>glucose (hypertonic)</td>
<td>β-blockers (A1)</td>
<td></td>
</tr>
<tr>
<td>hydroxycobalamin</td>
<td>insulin (A1)</td>
<td></td>
</tr>
<tr>
<td>isoprenaline</td>
<td>cyanide (A1)</td>
<td></td>
</tr>
<tr>
<td>methionine</td>
<td>β-blockers (A1)</td>
<td></td>
</tr>
<tr>
<td>4-methylpyrazole</td>
<td>paracetamol (B1)</td>
<td></td>
</tr>
<tr>
<td>methylibromine chloride</td>
<td>ethylene glycol (A1)</td>
<td></td>
</tr>
<tr>
<td>(Methylene Blue)</td>
<td>methaoglobinemia (A1)</td>
<td></td>
</tr>
<tr>
<td>naloxone</td>
<td>opiates (A1)</td>
<td></td>
</tr>
<tr>
<td>neostigmine</td>
<td>curare type agents (B2)</td>
<td></td>
</tr>
<tr>
<td>obidoxime</td>
<td>organophosphorus insecticides (B2)</td>
<td></td>
</tr>
<tr>
<td>oxygen</td>
<td>cyanide, carbon-monoxyde</td>
<td></td>
</tr>
<tr>
<td>oxygen, hyperbaric</td>
<td>sulphide (A1)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>carbon monoxide</td>
<td></td>
</tr>
</tbody>
</table>

Cyanide, hydrogen sulphide, carbon tetrachloride.
<table>
<thead>
<tr>
<th>Antidote</th>
<th>Main indication or pathological condition</th>
<th>Other possible applications</th>
</tr>
</thead>
<tbody>
<tr>
<td>penicillamine</td>
<td>copper (C1)</td>
<td>Lead, mercury (inorganic) (C2)</td>
</tr>
<tr>
<td>penicatic acid</td>
<td>cobalt (C3)</td>
<td>radio-active metals</td>
</tr>
<tr>
<td>phenolamine</td>
<td>α-adrenergic poisoning (A1)</td>
<td></td>
</tr>
<tr>
<td>physostigmine</td>
<td>central anti-cholinergic syndrome (from atropine)</td>
<td></td>
</tr>
<tr>
<td>vitamin K</td>
<td>coumarin derivatives (C1)</td>
<td></td>
</tr>
<tr>
<td>prussian blue</td>
<td>thallium (B2)</td>
<td></td>
</tr>
<tr>
<td>pyridoxime</td>
<td>organophosphorous (A2)</td>
<td></td>
</tr>
<tr>
<td>penalderol</td>
<td>β-blockers (A1)</td>
<td></td>
</tr>
<tr>
<td>protamine sulfate</td>
<td>heparin (A1)</td>
<td></td>
</tr>
<tr>
<td>pyridoxine</td>
<td>noniazid/hydrazines (A2)</td>
<td></td>
</tr>
<tr>
<td>sildian</td>
<td>amantin (B2)</td>
<td></td>
</tr>
<tr>
<td>sodium nitrite</td>
<td>cyanide</td>
<td></td>
</tr>
<tr>
<td>sodium atropinsid</td>
<td>ergotism (A1)</td>
<td></td>
</tr>
<tr>
<td>sodium thiosulfate</td>
<td>cyanide (A1)</td>
<td></td>
</tr>
<tr>
<td>succimer (DMSA)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>trinitrines</td>
<td>antimony,arsenic, bismuth, cadmium, cobalt, copper, gold, lead, mercury (organic and inorganic) (B2)</td>
<td>mercury (elemental), platinum, silver (C3)</td>
</tr>
<tr>
<td>trithiol (DMPS)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>cobalt, copper, gold, lead, mercury (inorganic, nickel) (C2)</td>
<td>cadmium, mercury (organic) (C3)</td>
</tr>
</tbody>
</table>
Group 2. Agents used to prevent absorption of poisons, to enhance their elimination, or to treat symptomatically their effects on bodily functions

Emetics

- apomorphine
- ipecacuanha

Cathartics and solutions for whole gut lavage

- magnesium citrate/sulfate/hydroxide (B3)
- mannitol/sorbitol/lactulose (B3)
- polyethylene glycol electrolyte lavage solution (B2)

Agents to alkalise urine or blood

- sodium bicarbonate (A1)

Agents to prevent absorption of toxic substances in the G.I. tract

- Activated charcoal (A1)

**Agents to prevent skin damage**

- calcium gluconate gel (A1) - for Hydrofluoric Acid
- polyethylene glycol (Macrogol 400) for phenol
### Group 3. Other therapeutic agents useful for the treatment of poisoning

Listed below are certain therapeutic agents that are not antidotes according to the accepted definition; however, because of their established value and sometimes specific role in the management of poisoning, they border on the concept of antidotes. In practice, these agents are used frequently in cases of poisoning and in other medical circumstances. Most of them are considered essential drugs and therefore are required to be available for immediate use.

<table>
<thead>
<tr>
<th>Agent</th>
<th>Indications/Symptoms arising from poisoning</th>
</tr>
</thead>
<tbody>
<tr>
<td>benzatropine</td>
<td>Dystonia</td>
</tr>
<tr>
<td>chlorpromazine</td>
<td>psychotic states with severe agitation</td>
</tr>
<tr>
<td>corticosteroids</td>
<td>acute allergic reactions, laryngeal oedema (systemic/topical), bronchoconstriction, mucosal oedema (inhaled)</td>
</tr>
<tr>
<td>diazepam</td>
<td>convulsions, excitation, anxiety, muscular hypertonia</td>
</tr>
<tr>
<td>diphenhydramine</td>
<td>dystonia</td>
</tr>
<tr>
<td>dobutamine</td>
<td>myocardial depression</td>
</tr>
<tr>
<td>dopamine</td>
<td>myocardial depression, vascular relaxation</td>
</tr>
<tr>
<td>adrenaline</td>
<td>anaphylactic shock, cardiac arrest</td>
</tr>
<tr>
<td>furosamide</td>
<td>fluid retention, left ventricular failure</td>
</tr>
<tr>
<td>glucose</td>
<td>hypoglycemia</td>
</tr>
<tr>
<td>haloperidol</td>
<td>hallucinatory and psychotic states</td>
</tr>
<tr>
<td>heparin</td>
<td>hypercoagulability states</td>
</tr>
<tr>
<td>magnesium sulfate</td>
<td>cardiac arrhythmias</td>
</tr>
<tr>
<td>mannitol</td>
<td>cerebral oedema, fluid retention</td>
</tr>
<tr>
<td>oxygen</td>
<td>hypoxia</td>
</tr>
<tr>
<td>pancuronium</td>
<td>muscular rigidity, convulsions</td>
</tr>
<tr>
<td>promethazine</td>
<td>allergic reactions</td>
</tr>
<tr>
<td>salbutamol</td>
<td>bronchoconstriction (systemic/inhaled)</td>
</tr>
<tr>
<td>sodium bicarbonate</td>
<td>acidosis</td>
</tr>
</tbody>
</table>
## Group 4. List of antidotes and related agents now considered obsolete

<table>
<thead>
<tr>
<th>Antidote</th>
<th>Indicated for</th>
</tr>
</thead>
<tbody>
<tr>
<td>acetazolamide</td>
<td>modification of urinary pH</td>
</tr>
<tr>
<td>ascorbic acid</td>
<td>methemoglobinemia</td>
</tr>
<tr>
<td>aurintricarboxylic acid</td>
<td>beryllium</td>
</tr>
<tr>
<td>β-aminopropiononitrile</td>
<td>caustics</td>
</tr>
<tr>
<td>castor oil</td>
<td>as cathartic</td>
</tr>
<tr>
<td>copper sulfate</td>
<td>as emetic</td>
</tr>
<tr>
<td>cyclophosphamide</td>
<td>gold-paraquat</td>
</tr>
<tr>
<td>cysteamine</td>
<td>paracetamol</td>
</tr>
<tr>
<td>diethylthiocarbamate</td>
<td>thallium</td>
</tr>
<tr>
<td>fructose</td>
<td>ethanol</td>
</tr>
<tr>
<td>guanidine precursors</td>
<td>botulism</td>
</tr>
<tr>
<td>levorphan</td>
<td>opiates</td>
</tr>
<tr>
<td>nalorphine</td>
<td>opiates</td>
</tr>
<tr>
<td>potassium permanganate</td>
<td>fluonides</td>
</tr>
<tr>
<td>sodium chloride</td>
<td>as emetic</td>
</tr>
<tr>
<td>sodium salicylate</td>
<td>beryllium</td>
</tr>
<tr>
<td>strychnine</td>
<td>CNS depressants</td>
</tr>
<tr>
<td>sulfadimidine</td>
<td>amantadine</td>
</tr>
<tr>
<td>tannins</td>
<td>alkxloids</td>
</tr>
<tr>
<td>thiocac acid</td>
<td>antamidine</td>
</tr>
<tr>
<td>vitamin E</td>
<td>paraquat</td>
</tr>
<tr>
<td>trilonium chloride</td>
<td>methemoglobinemia</td>
</tr>
<tr>
<td>universal antidote</td>
<td>ingested poisons</td>
</tr>
</tbody>
</table>


References:

3. NIOH-Poison information cards (Poison information centre) -National Institute of Occupational Health,Meghani Nagar,Ahmedabad.
   a) chemicals and methemoglobinemia
   b) organophosphorus pesticides
   c) aluminium phosphate
   d) synthetic pyrethroid pesticides
   c) organochlorine pesticides
   f) inorganic acids
5. WHO,Geneva,United nations environmental programme,International labour organisation,Environmental health criteria series:
   a) Ammonia (54,1986)
   b) Cadmium (134,1992)
   c) Carbon Monoxide (13,1979)
   d) Chlorine and Hydrogen Chloride (21,1982)
   e) Hydrogen sulphide (19,1981)
   f) Lead (3,1977)
   g) Mercury (1,1976)
   h) Mercury inorganic (118,1991)
   i) Nitrogen oxides (4,1991)
   j) Organophosphorus pesticides (63,1986)
   k) Trichloro Phosphate (110,1990)
7. Harrison's Principles of internal medicine;14th ed;Mcgraw-Hill Book company New York