DEDER GENERAL HOSPITAL

POISON MANAGEMENT PROTOCOL

PREPARED BY*: *HSQU

JULY 2016 E.C

DEDER, EASTERN ETHIOPIA

PROTOCOL APPROVAL SHEET

NAME OF PROTOCOL: POISON MANAGEMENT PROTOCOL

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THIS PROTOCOL IS EFFECTIVE FROM JULY 2016 E.C TO JUNE 2018 E.C

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List of abbreviations and acronyms

2,4-D	2,4-dichlorophenoxyacetic acid
ABC	Airway, Breathing and Circulation
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
AV	Atrioventricular
BUN	Blood Urea Nitrogen
CBC	Complete Blood Count
CNS	Central Nervous System
CPK	Creatinine Phosphokinase
CT	Computed Tomography
D5W	5% dextrose in water
ECG	Electrocardiographic
ED	Emergency Department
FDA	Food and Drug Administration
GABA	ϒ-aminobutyric acid
GI	Gastrointestinal
H1	Histamine 1
H2	Histamine 2
Hcg	Human chorionic gonadotrophin
IM	Intramuscular
INH	Isoniazide
INR	International Normalized Ratio
IV	Intravenous
LP	Lumbar Puncture
MAO	Monoamine Oxidase Inhibitors
MRI	Magnetic Resonance Imaging
NAC	N-acetylcysteine
NSAIDs	Nonsteroidal anti-inflammatory drugs
PT	Prothrombin time
RNA	Ribonucleic acid
VD	Volume of Distribution
WHO	World Health Organization

1. INTRODUCTION

- Poisoning is an exposure to an amount of substance that is likely to produce untoward effects in an individual. Or *Poisoning* occurs when exposure to a substance adversely affects the function of any system within an organism.
- Advances in technology and social development have resulted in the availability of most drugs and chemical substances in the community.
- These chemical substances pose a significant threat due to their poisonous effect and extensive use in medicine, agriculture, industry, and residential environments.

1.1.Route of exposure

- Route of exposure for poisoning can be through ingestion, Injection, inhalation insufflations and contact. Toxicity level of a poisoned agent depends on the type of agent, doses, route of exposure, timing and the host health conditions. Oral ingestion is identified as the primary route of poisoning.

1.2.Types and common causes of poisoning

- Acute pesticide poisoning is one of the most common causes of intentional deaths worldwide. High doses of analgesics, tranquillizers, and antidepressants are the commonly used agents for intentional poisoning in industrialized countries and majority of pesticide exposure is seen more in middle and low-income countries due to increased use of agrochemicals in agricultural sector.
- Most poisoned patients seen in the emergency department are adults with acute oral drug overdoses.
- Childhood poisoning is usually accidental and tends to be associated with a low morbidity and mortality. In Western Europe and North America, it is most often due to household products and pharmaceuticals; in developing countries, paraffin, traditional medicines, snake's bites and insect stings are more commonly involved.
- In adults, self-poisoning is usually deliberate (suicide or parasuicide) and has a higher morbidity and mortality rate. Analgesics and psychotropics predominate in Western Europe and North America as causes of admission to hospital, though carbon monoxide is
- responsible for most deaths (the majority of which occur outside hospital). In developing

- countries, accidental and deliberate pesticide poisoning is probably the commonest cause of adult deaths.
- In Ethiopia, Organophosphates and household cleansing agents are the predominant agents of acute poisoning. This is not surprising in that organophosphate compounds (OPCs) are widely used in third world countries like Ethiopia to increase the yield of agriculture products to meet the highly increasing demand of the society.
- If the toxic effects occur immediately, usually within hours from the time of exposure, is called acute poisoning. Acute poisonings or drug overdoses constitute a significant source of aggregate morbidity, mortality, and health care expenditure. The true incidence is unknown due to under diagnosis and underreporting.
- Chronic poisonings or poisonings with delayed health effects are often more problematic in the long run. Chronic poisoning occurs from drug abuse or from environmental, industrial, and agricultural chemical exposure; medication reactions or interactions; and envenomation.
- Poisoning can be intentional or unintentional. Intentional poisoning often occurs in patients with depression or coping difficulties and may need extra psychological, familial or social attention. While the medical burden of intentional poisonings seems to be equal for similar severe cases. Its burden includes physical as well as mental disabilities.

1.3. Prevention methods of poisoning

Here are some globally proven techniques to decrease the burden of poisoning

- Store medicine, cleaning and laundry products, (including detergent packets) paints/varnishes and pesticides in their original packaging in locked cabinets or containers, out of sight and reach of children.
- The safest place to store poisonous products is somewhere a child can't see or reach. Purchase and keep all medicines in containers with safety caps. Discard unused medications.
- Never refer to medicine as –candy‖ or another appealing name, when giving to children.
- Never place poisonous products in food or drink containers. For E.g. keeping kerosene in water bottles, children can mistake it for water.

- Keep natural gas-powered appliances, furnaces, and coal, wood or kerosene stoves in safe working order.

- Maintain working smoke and carbon monoxide detectors, when available. But always use cooking charcoal out of the house or in a well-ventilated area. Carbon monoxide poisoning is a serious health hazard.
- Secure remote controls, key fobs, greeting cards, and musical children's books. These and other devices may contain small button-cell batteries that can cause injury if ingested.
- Always be on the lookout for a depressed family member, advice on psychiatric care when needed.
- Advice family members to monitor and administer anti depressant and other drugs to family members. They may escape taking the drugs or may overdose of given a large amount till their next follow up.

2. General approaches to acute poisoning

- In the management of acutely poisoned patients, we must consider the dose of the substance ingested, time since ingestion, clinical features, patient factors, geographical location, and available medical facilities.
- A highly organized approach is essential to ensure effective delivery of time-critical interventions while at the same time devising a management plan tailored to the individual patient's needs in that particular medical setting.
- The first priority in treating poisoned patients is assessment and stabilization of cardiopulmonary function (e.g., the ABCs, or *a*irway, *b*reathing, and *c*irculation).
- The general approach to the diagnosis and management of the poisoned patient can be described using a two-pronged model as depicted in Fig.1.1 as well as by the approach outlined in Box 1.1.
- The left-sided prong begins with basic emergency medical care-the ABCs (airway, breathing, circulation).
    - ✓ In most potentially poisoned patients, a rapid blood glucose measurement should be obtained, and any derangements corrected.
    - ✓ Supplemental oxygen, naloxone and thiamine should be considered in the appropriate cases and situations.
    - ✓ The various methods of decontamination should be considered in any poisoned patient based on each individual clinical situation.

- ✓ Once a poisoning has been identified, methods of enhanced elimination should be considered. Focused therapy involves antidote administration when appropriate or aggressive supportive care tailored to the poison in question.
- ✓ Finally, when treating any poisoned patient, it is prudent to consider early consultation with a toxicology service or local poison control center for further

- The right-sided prong on the diagram focuses on obtaining the poisoning and other patient history, performing a focused physical examination with attention to toxidrome recognition, and deciding on the appropriate diagnostic tests to be performed.

NB: The two prongs often occur simultaneously and are integral to the diagnosis and management of a poisoned patient.

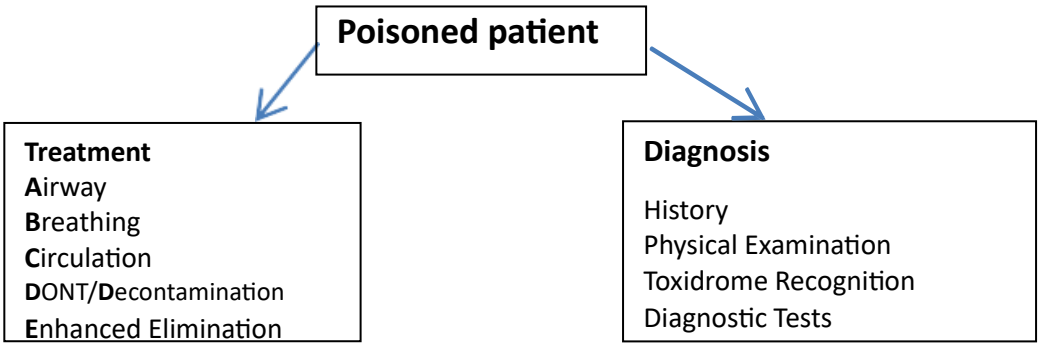

Figure 1: The two-pronged approach to the poisoned patient:

DONT stands for dextrose, oxygen, naloxone, and thiamine. It applies in case of unknown poisoning with unconsciousness and coma. Treat hypoglycemia with IV dextrose (glucose). Patients at risk of Wernicke 's encephalopathy also require thiamine, but do not require that it be administered before the dextrose. Altered mental status, when hypoglycemia cannot be excluded, is an indication for IV dextrose. Supplemental oxygen, thiamine, glucose, and naloxone are often administered empirically as a cocktail in cases of altered mental status.

Table 1: General approach to acute poisoning

i) **Resuscitation** – Airway – Breathing – Circulation – Seizure control – Correct hypoglycaemia – Correct hyperthermia – Resuscitation antidotes	ii) Risk assessment iii) Supportive care and monitoring iv) Investigations – Serum level (paracetamol, alcohol, aspirin – ECG v) Decontamination vi) Enhanced elimination vii) Antidotes viii) Disposition

i. Initial approach (Resuscitation)

1. Airway

- ✓ Loss of airway patency and reflexes may lead to obstruction, aspiration, or respiratory arrest.
- ✓ Maintain proper airway position; suction; use oropharyngeal or nasopharyngeal adjuncts as needed.
- ✓ Absent or depressed gag reflex in an unconscious or obtunded patient indicates an inability to protect the airway; so endotracheal intubation strongly considered if there is doubt about the patient's ability to protect the airway and avoid aspiration
- ✓ In-line cervical immobilization is required in patients with suspected occult trauma.

2. Breathing

- ✓ Respiratory failure is the most frequent cause of death in poisoned patients, and usually it is a result of (CNS) central nervous system depression.
- ✓ Assessing Breathing pattern, RR, Spo_2 measurement is very important
- ✓ Assist ventilation and administration of oxygen is important if the ventilation desaturation is compromised.
- ✓ Obtain and follow arterial blood gases.

3. Circulation

- ✓ Monitor blood pressure, pulse, and cardiac rhythm.
- ✓ Initiate intravenous line.
- ✓ If hypotension is present, administer fluid challenge with normal saline 10–20 mL/kg.
- ✓ If hypotension persists, administer vasopressor such as norepinephrine can also be used.

4. Disability

- ✓ Assessment of mental status (assessing GCS), gross motor movement, Pupillary size and reactivity to light.
- ✓ Determination of random blood glucose level. Rapid RBS test strips may be used to guide dextrose administration.
- ✓ Empiric administration of dextrose is recommended for the patient with altered consciousness when test strip glucose measurements are low or borderline low, not immediately available, or the accuracy of their results is questioned.

5. Exposure

- Complete exposure and examination of the patient
- Measurements of core temperature are essential.

❖ Indication for intubation in a poisoned patient or Suspected poisoning
- ✓ Respiratory failure
- ✓ Comatose patient who fails to protect air way
- ✓ Hypoxia refractory for no invasive oxygen administration
- ✓ Anticipation of Deterioration etc.

❖ Bradyarrhythmia associated with hypotension should be treated in the standard fashion with atropine or temporary pacing.

❖ In patients with calcium channel blocker or beta blocker intoxication, the administration of calcium and glucagon may obviate the need for further management.

- ✓ Drug-induced agitation is generally best treated with benzodiazepine administration, supplemented with high potency neuroleptics (haloperidol).
- ✓ Benzodiazepines to treat tachycardia secondary to sympathomimetic agents.
- ✓ Seizures, hypoglycemia, and hyperthermia must be detected and treated promptly to ensure good neurological outcome.
- ✓ Toxin-induced seizures tend to be global central nervous system (CNS) processes and are Grand mal or GTC in nature.
- ✓ Toxic seizures are usually controlled with iv benzodiazepines.
- ✓ Barbiturates are second line, and Pyridoxine is an additional option for seizures associated with poisoning from isoniazid.
- ✓ Phenytoin is not useful in the treatment of toxic seizures, and may worsen toxicity.
- ✓ Administration of an antidote may constitute an essential component of initial resuscitation.

i. Risk assessment

Following resuscitation, risk assessment is the next essential step in management of the poisoned patient (see box below). Risk assessment is a distinct cognitive process through which the clinician attempts to predict the likely clinical course and potential complications for the individual patient at that particular presentation. Risk assessment should be quantitative and take into account agent, dose, time of ingestion, current clinical status and individual patient factors (for example, weight and comorbidities). Risk assessment is vital as it allows the clinician to make specific decisions about all subsequent management steps (appropriate supportive care and monitoring; screening and specialized testing;

decontamination; enhanced elimination; antidotes and disposition) that are appropriate to the individual patient at that particular time.

- Distinct cognitive step
- Quantitative
- Takes into account:
  - Agent(s)
  - Dose(s)
  - Time since ingestion
  - Current clinical status
  - Patient factors

Table 2: Risk assessment

2.1. Diagnosis of poisoning

2.1.1. History taking

History taking should include the type of toxin or toxins, time of exposure (acute versus chronic), amount taken, and route of administration (i.e., ingestion, intravenous, iv, or inhalation). It is also important to understand why the exposure occurred (accidental, suicide attempt, euphoria, or therapeutic misadventure) and whether there is history of psychiatric illness or previous suicide attempts. Furthermore, it is important to inquire about all drugs taken, including prescription, over-the-counter medications, vitamins, and herbal preparations as well as the nature and progression of signs and symptoms. If unavailable from the patient, information solicited from family and friends may also prove helpful, acting in the patient‘s best interest. Further history can be obtained by consulting the patient‘s other physicians or by obtaining old medical records. In the case of an occupational exposure, one should obtain a description of the work environment and contact people at the site for relevant information. Information regarding specific toxins may also prove useful.

2.1.2. Physical examination

In the emergency setting, performing an overly detailed physical examination is a low priority compared with patient stabilization. A directed examination can, however, yield important diagnostic clues. Once the patient is stable, a more comprehensive physical examination can reveal additional signs suggesting a specific poison. One should take note also that a dynamic change in clinical appearance over time may be a more

important clue than findings on a single examination.

The following physical examinations might help to deduce the class of drug or toxin implicated in the poisoning.

- **Vital sign**: detecting signs such as tachycardia, hyperthermia, and hypotension through addressing the patient‘s vital sign help in making the differential diagnosis (Box 1.2).
- **Neurologic examination**: a systematic neurologic evaluation is important, particularly with patients exhibiting altered mental status. In contrast to the patient who has structural brain injury, the patient who has a toxic-metabolic cause of coma may exhibit ‗patchy‘‘ neurologic impairment. Toxicologic causes of coma rarely cause focal neurologic deficits. Seizures are common presentation of an unknown overdose, and the list of toxins that can induce a convulsion is lengthy. Classic pupillary findings include miosis (opioids, cholinergic, carbamates, clonidine, organophosphates, Phenothiazines, Sedatives-hypnotics) and mydriasis (sympathomimetics, anticholinergics, Withdrawal syndrome). Nystagmus suggests phenytoin, along with carbamazepine, lithium, ethanol, barbiturates, and sedative hypnotics. Vertical nystagmus largely represents brain stem lesion. Optic neuritis and vision loss, although seen in multiple sclerosis, may indicate advanced methanol poisoning. Other general neurologic signs include fasciculations (organophosphate poisoning), rigidity (tetanus and strychnine), tremors (lithium and methylxanthines), speech-mumbling (anticholinergics), and dystonic posturing (neuroleptic agents).
- **Skin:** a careful examination of the skin should be performed. The absence of diaphoresis is an important clinical distinction between sympathmimetics and anticholinergics. Bullous lesions may be associated with sedative-hypnotic drug-induced coma, but are classically described with barbiturate poisoning. Such lesions could also be indications of rhabdomyolysis or the development of compartment syndrome. A common skin finding is the presence of track marks, suggesting iv or subcutaneous (sc) opiate or cocaine abuse. Blue skin indicates methemoglobinemia or hypoxia; red skin may suggest niacin or boric acid exposure.

Table 3: Diagnosis of toxicity based on vital sign

- Hypothermia (**COOLS**)
  - **C**arbon monoxide
  - **O**pioids
  - **O**ral hypoglycemic, Insulin
  - **L**iquors (alcohol)
  - **S**edative-hypnotics
- Hyperthermia (**NASA**)
  - **N**euroleptic malignant syndrome, nicotine
  - **A**ntihistamines
  - **S**alycilates, Serotonin syndrome, sympathomimietcs
  - **A**nticholinergics, antidepressants, antipsychotics
- Hypotension (**CRASH**)
  - **C**lonidine, calcium channel blockers
  - **R**odenticides (arsenic, cyanide containing)
  - **A**ntidepressants, aminophilline, antihypertensives
  - **S**edative-hypnotics
  - **H**eroine or other opioids
- Bradycardia (**PACED**)
  - **P**ropranolol (β-blockers), Opiates
  - **A**nticholinergic drugs, antiarrythmics
  - **C**lonidine, calcium channel blockers
  - **E**thanol and other alchols
  - **D**igoxin, digitalis
- Tachycardia (**FAST**)
  - **F**ree base or other forms of cocaine
  - **A**ntihistamines, anticholinergics, alcohol withdrawl
  - **S**ympathomimetics (Cocaine, caffine, amphetamine)
  - **T**heophylline, TCAs

- **Odor:** some poisons produce odors characteristic enough to suggest the diagnosis, such as oil of wintergreen (methylsalicylates) or garlic (organophosphate insecticides, arsenic). Some odors may be more subtle and cannot be detected by a sizable number of the population, such as the freshly mowed hay smell of phosgene or the bitter-almond scent associated with cyanide. Certain odors may be overpowering and easily noted by anyone managing the patient. For example, sulfur dioxide and hydrogen sulfide produce a noxious rotten-egg smell.

2.1.3 Toxidromes recognition

- The term *toxidrome* refers to a syndrome or constellation of physical findings attributed to a specific class of toxins that can provide important clues to narrow the differential diagnosis.
- The most common toxidromes are the anticholinergic syndrome, Cholinergic syndrome, sympathomimetic syndrome, opioids, and serotonin syndrome.

Table 4: Common Toxidromes

Toxidromes	**Mental status**	**Pupils**	**Vital signs**	**Other manifestation**	**Examples of toxic agent**
Sympathomimetic	agitation, hallucinations, paranoia	Mydriasis	Hyperthermia, Tachycardia; hypertension, tachypnea,	Diaphoresis, tremors ,hyperreflexia, seizures	✓ Cocaine ✓ amphetamines ✓ Ephedrine ✓ Theophylline
Anticholinergic	Agitation Hallucination delirium coma	Mydriasis	Hyperthermia, tachycardia, hypertension, Tachypnea	• Dry skin • decreased bowel sounds • urinary retention • Myoclonus	• Antihistamines • TCAs phenothiazines • atropine • scopolamine
Opioids	CNS depression Coma	Miosis	Hypotension Bradycardia Hypothermia Bradypnea, apnea	Hyporeflexia, pulmonary edema, needle marks	Opiates (eg, heroin, morphine, methadone, pethidine etc)
Sedative-hypnotic	CNS depression Stupor, confusion, coma	Miosis (usually)	Hypotension Bradycardia Hypothermia Bradypnea	Hyporefelxia	Benzodiazepines Barbiturate Ethanol
Cholinergic	Confusion, coma	Miosis	Bradycardia, HTN, Tachycardia Bradypnea	Salivation, Lacrimation, Urination, Emesis Defecation, fascicuation etc..	Organophosphates Carbamates etc
Serotonin syndrome	Confusion, agitation, coma	Mydrasis	Hyperthermia, tachycardia, hypertension, Tachypnea	Tremor,Myoclus, regidity,Clonus hyperreflexia,Trismus, diaphoresis,Diarrhea	MAOIs alone or with: SSRIs, meperidine, TCAs dextromethorphan,

2.1.4. Laboratory tests

❖ Symptomatic patients and those with an unreliable or unknown history should be investigated with at a minimum of:

- CBC
- Urinalysis
- Serum electrolytes
- BUN and creatinine
- Liver enzymes and Liver function test
- Serum glucose level
- Routine urine pregnancy testing is strongly recommended in all women of childbearing age

- The ordering of other laboratory studies should be individualized and is somewhat dependent upon the results of initial laboratory studies:
  - Arterial blood gas, co-oximetry, and serum lactate measurements may be necessary in patients with acid-base, cardiovascular, neurologic, or respiratory disturbances.
  - The presence of an anion gap metabolic acidosis may be the first clue to a toxic ingestion and should prompt measurement of serum salicylates, ethylene glycol, and methanol.\
  - Co-oximetry can aid in the rapid diagnosis of carbon monoxide poisoning and methemoglobinemia.
  - Toxic screening is rarely necessary when patients with a nonintentional ingestion are asymptomatic or have clinical findings that are consistent with the medical history.
  - Drugs of abuse" immunoassay screens can be used to detect opiates, benzodiazepines, cocaine metabolites, barbiturates, tricyclic antidepressants. in urine

2.1.5. Electrocardiogram (ECG)

It is a useful test to detect cardiac conduction abnormalities and identify patients at increased risk of toxin-induced adverse cardiovascular events. an ECG should be performed on all patients who are symptomatic or who have been exposed to potentially cardiotoxic agents.

*Table 5: **drug and toxin induced electrocardiographic abnormalities***

Brady arrhythmias/AV blocks	**Supraventricular Tachyarrhythmia**	**Ventricular Tachycardia**	**QT and QT prolongation**
Beta blockers	Amphetamine	Amphetamine	Anti depressants
Calcium channel blockers	Cocaine	Cocaine	Anti Physchotics
Digoxin	Theophyline	Theophyline	Quinidine
Organophosphate	TCAs	TCAs	Amiodarone
Opoids	Atropine	Phenothiazines	Diphenyhydramine
Magnesium	Phenothiazines		
		Cardiac glycoside	Organophosphate
Sedative hypnotics	Epinephrine/Dopamine		

2.1.6. Radiographic studies

- Imaging studies are not required in every patient but may be useful in several situations
  - Certain radiopaque toxins (summarized by the mnemonic "CHIPES") may be visualized by plain (Table-3)
  - Ingested drug packets of "body packers" may be seen on plain film(figure-1)
  - Noncardiogenic pulmonary edema and/or the acute respiratory distress syndrome due to exposure to certain toxic agents may be suggested by the appearance of the chest radiograph(patients with Organophosphate poisoning, CO poisoning, Beta Blockers etc..)

***Table 6:** Agents possibly radiopaque on plain x-ray*

C	Chlorinated hydrocarbons(e.g chloral hydrate,carbon tetrachloride)
	Calcium salt (e.g calcium carbonate)
	Crack vials
H	Heavy metal (e.g iron,arsenic,mercury,thallium,lead)
I	Iodinated compound (e.g thyroxine)
P	Psychotropics (e.g phenothiazines, lithium, cyclic antidepressants)
	Packets of drugs (e.g cocaine and heroin –body packers‖)
	Play-Doh
	Potassium salts
E	Enteric – coated tablets(e.g aspirin)
S	Salicylates
	Sodium salts
	Sustained – release preparations

2.2. Decontamination

- Following initial patient stabilization, patient decontamination may be performed if indicated. The sooner decontamination is performed, the more effective it is at preventing poison absorption.\
- Decontamination ideally occurs in a separate area adjacent to the ED, minimizing cross-contamination.
- Decontamination of severely poisoned patient must only be performed after careful consideration of the potential risks and benefits of the decontamination procedure.

2.2.1. Skin decontamination

- Corrosive agents rapidly injure the skin and must be removed immediately.
- Many toxins are readily absorbed through the skin, and systemic absorption can be prevented only by rapid action.
- Remove contaminated clothing and flush exposed areas with copious quantities of tepid (lukewarm) water or saline. Wash carefully behind ears, under nails, and in skin folds. Use soap and shampoo for oily substances.
- Health care providers should always wear personal protective equipments to prevent secondary contamination to themselves.

2.2.2. Ocular decontamination

- Eye exposures need prolonged irrigation with copious amount of water or saline
- Alkalis produce greater injury than acids due to deep tissue penetration via liquefaction so that prolonged irrigation (1 to 2 h) may be required.
- Ophthalmologic consultation is indicated for all ocular alkali injuries.

2.2.3. Gastrointestinal decontamination

- Gastric decontamination is not a routine part of poisoned-patient management
- Gastric decontamination may be considered in individual patients after a three- question risk-benefit analysis:

  - (i) is this exposure likely to cause significant toxicity?;
  - (ii) is gastrointestinal decontamination likely to change clinical outcome?; and
  - (iii) is it possible that gastrointestinal decontamination will cause more harm than good?

A. Gastric lavage

- Is the process of irrigating the gastric cavity to remove recently ingested material? (within two hours post ingestion)

Indication:

- Gastric lavage may be considered in cases of ingestion of a life-threatening amount of poison within the previous hour where institution of supportive care and antidotal therapy would not ensure full recovery once absorbed.
- In certain circumstances, such as delayed gastric emptying accompanying intoxication with anticholinergic drugs and phenobarbitone, benefit may be noted longer after ingestion.

Procedure:

- Ensure a protected airway if consciousness level is reduced.
- Use a 36 to 40F-gauge orogastric tube (22 to 24F in children).
- Position the patient on the left side with the head down 20 degrees.
- Pass lubricated tube down the esophagus, a distance equal to that between chin and xiphoid process.
- Confirm tube position by insufflation of air.
- Gently lavage with 200 mL (10 mL/kg in children) of warm tap water.
- Continue until returned fluid is clear.
- Consider administration of activated charcoal via orogastric tube before removal.
- Liquid agents can be lavaged with a smaller diameter nasogastric tube, but extraction of pill fragments requires use of a largebore tube (36–40 F).
- Large bore tubing may only be placed via the orogastric route to avoid trauma to the nasopharynx.

Complications:

- Placement of an orogastric tube is a distressing procedure to perform in an awake patient and may be complicated by gagging and aspiration.
- Other serious complications such as laryngospasm, dysrhythmia and perforation.
- Aspiration pneumonia/hypoxia
- Water intoxication
- Hypothermia
- Time consuming, resulting in delay instituting other definitive care

Contraindication:

- Contraindicated in cases of acid, alkali or hydrocarbon ingestion, and in comatose patients with absent gag reflex because of the risk of aspiration. (Airway should be protected by intubation first, in these patients).
- Supportive care/antidote likely to lead to recovery
- Unstable, requiring further resuscitation (hypotension, seizures)

B. Activated charcoal

Indications:

- Activated charcoal minimizes absorption of drugs by adsorbing them onto its surface.
- Charcoal administration has become the decontamination strategy of choice to prevent poisoning after toxicant ingestion and is most effective when used in the 1st hour after ingestion.
- Possesses large surface area that when administered orally, adsorbs ingested xenobiotics within the gastrointestinal tract thereby preventing systemic absorption.
- Some agents such as metals, ions and alcohols do not bind to charcoal.
- Significant increase in clearance for a number of drugs when repeated doses of 0.5 to 1 g per kg of activated charcoal are given every 4 to 6 h.

Contraindication:

- In patients with an unprotected airway (e.g., deeply comatous, depressed gag reflex) or a disrupted GI tract (e.g., after severe caustic ingestion, hypoactive bowel sound) or in patients in whom charcoal therapy may increase the risk and severity of aspiration (e.g., hydrocarbons).
- In addition, nontoxic ingestion, in this case toxin not adsorbed by activated charcoal and recovery will occur without administration of activate charcoal.

Complications:

- Bowel perforation or obstruction following multidose charcoal administration, vomiting, aspiration of the activated charcoal and impaired absorption of orally administered antidotes.

Dose and Administration:

- Poisoning (reduction of absorption), Oral: as soon as possible after ingestion of poison, **Adult,** 50–100 g as a single dose; **Infant,** 1 g/kg as a, single dose; **Child** 1–12 years, 25 g as a single dose (50 g in severe Poisoning).
- Poisoning (active elimination or multiple dose), Oral: **Adult,** 50 g every 4 hours (in case of intolerance 25 g every 2 hours); **Infant,** 1 g/kg every 4–6 hours; **Child** Over 1 year,

25–50 g every 4–6 hours.

C. Whole bowel irrigation

Indications:

- It uses a laxative agent such as polyethylene glycol to fully flush the bowel of stool and unabsorbed xenobiotics. Not recommended for routine use in the poisoned patient.
- May be considered for substantial ingestions of iron, sustained release products, enteric coated products and symptomatic acute lead toxicity with known lead particles in the gastrointestinal tract. It has been used for other metal ingestions (e.g., lead), overdoses of sustained-release medications (e.g., lithium, theophylline), ingested pharmaceutical patches, and ingestions of vials or packages of illicit drugs.
- It might also be useful in particularly massive and/or late-presenting overdoses for which the efficacy of gastric emptying and/or charcoal is expected to be suboptimal. The technique may be used by mouth in cooperative patients or by NG tube; the usual recommended dosing is 500 mL per hour (25ml/kg/hr, maximum 2L/h) in children and 2 L per hour in adolescents and adults.

Contraindication:

- In ileus, bowel obstruction or perforation, and in patients with hemodynamic instability. Unprotected airway, hemorrhage, Intractable vomiting

Complications:

- Nausea, vomiting, Pulmonary aspiration, Time consuming; possible delay instituting other definitive care

2.3. Enhanced elimination

- In severely poisoned patient, enhancing the toxin elimination may improve outcomes for some poisonings.
- Procedures to enhance elimination of poisons include forced diuresis, urine ion trapping, hemodialysis, hemoperfusion, hemofiltration, and exchange transfusion. Various measures are useful in selected circumstances.

A. Urine alkalization:

Indication:

- It may be considered for agents that are excreted as weak acids in the urine (Moderate to severe salicylate toxicity not meeting criteria for hemodialysis).
- By alkalinizing the urine through use of iv sodium bicarbonate, these weak acids will remain in a more polar ionized form in the urine that limits reabsorption and enhances elimination.
- Urine alkalinization may be considered for, 2, 4-dichlorophenoxyacetic acid (2,4 D), methotrexate, phenobarbital and salicylates.

Contraindications:

- Preexisting fluid overload, renal impairment, and uncorrected hypokalemia.

Complications:

- Hypokalemia, volume overload, alkalemia and Hypocalcemia (usually mild)

B. Dialysis:

Dialysis is used to remove toxins or overdose of drugs and can be hemodialysis or hemoperfusion.

❖ **Hemodialysis:** Hemodialysis is movement of solute down a concentration gradient across a semipermeable membrane.

Indications: It may be considered for poisons that are amenable to filtration across dialysis membranes. These include agents that possess low volume of distribution (Vd), low protein binding, low endogenous clearance, low molecular weight. Examples of agents that are commonly encountered and may require hemodialysis include Lithium, Metformin lactic acidosis, Phenobarbital, Salicylates, Valproic acid, Methanol/ethylene glycol, Metformin-induced lactic

Contraindication: Hemodynamic instability, infants (generally), poor vascular access and significant coagulopathy.

❖ **Hemoperfusion:** Hemoperfusion is movement of toxin from blood, plasma, or plasma proteins onto a bed of activated charcoal (or other adsorbents).

Indications: It is useful for toxins with low VD, low endogenous clearance, and bound by activated charcoal. Examples of agents are Theophylline (high-flux hemodialysis is an alternative), Carbamazepine (multidose activated charcoal or high-efficiency hemodialysis also effective) and Paraquat (theoretical benefit only if instituted early after exposure).

Contraindication: Hemodynamic instability, infants (generally), poor vascular access, significant coagulopathy and toxin not bound to activated charcoal

2.4. Antidotes

Although most poisonings are managed primarily with appropriate supportive care, several specific antidote agents may be employed.

The **Universal antidotes** are four classic compounds, which includes:

- **Oxygen:** for any cause of hypoxia
- **Dextrose:** insulin, hypoglycemics, coma, or protracted vomiting causing hypoglycemia
- **Naloxone:** Narcotics
- **Thiamine**: Wernicke`s or chronic alcohol abuse and in Malnourished patients.

The use of these antidotes should be individualized to the patient‘s condition.

***Table 7:** List of commonly used antidotes*

S. No.	Antidote	Poisoning indication
1.	N-Acetyl cysteine injection, 140 mg/kg PO, then 70 mg/kg q4h for up to 17 doses.	Acetaminophen
2.	Polyvalent Immune Fab, ovine (Snake Venom Antiserum polyvalent Injection, 10ml)	Snake bite of unknown snake type
3.	Atropine Sulfate Injection, 1mg/ml in 1 ml ampoule	Organophosphorus and N-methyl Carbamates
4.	Calcium chloride *Injection, 10% (100mg/ml)*	Fluoride, Calcium Channel blockers
5.	Calcium gluconate *Injection, 10% in 10ml ampoule*	Fluoride, Calcium Channel blockers, Magnesium sulfate
6.	Calcium disodium EDTA†	Lead
7.	Calcium trisodiumpentetate (CaDTPA)	Plutonium, Americium or Curium
8.	Cyanide Antidote Kit* or Hydroxicobalamine HCl	Cyanide
9.	Deferoxamine mesylate	Iron
10.	Digoxin Immune Fab (Ovine) Digoxin specific, antibody fragments Powder for injection, 40mg	Cardiac glycosides/ Steroids
11.	Ethanol	Methanol or Ethylene glycol
12.	Flumazenil*	Benzodiazepine

	Injection, 0.1 mg/ml in 5 ml ampoule	
13.	Glucagon HCl	B-blocker, Calcium channel blockers
14.	Methylene blue	Methemoglobinemia
15.	Naloxone HCl Injection, 0.02mg/ml in 2ml ampoule, 0.4mg/ml in 1ml and 10ml ampoule, 1mg/ml	Opioid and Clonidine
16.	Octeriotide acetate	Sulphonylurea
17.	Physostigmine salicylate Injection, 1mg/ml in 1ml and 2ml ampoule	Anticholinergic syndrome
18.	Pralidoxime chloride Powder for injection, 1g/vial	Organophosphates and N-methyl Carbamate insecticides
19.	Pyridoxine hydrochloride *Injection, 50mg/ml in 2ml ampoule, 150mg/ml*	INH, Hydrazine
20.	Sodium bicarbonate Injection :	Sodium channel blockers;TCA
21.	Phytomenadione (Vitamin K inj.)	Warfarin, Rodent poisons
22.	Protamine Sulphate Inj.	Heparin
23.	Sodium Polystyrene Sulphonate Powder	Hyperkalemia
24.	Dextrose 40% injection	Insulin, oral hypoglycemic agents
25.	Thiamine	Alcohol intoxication
26.	Trimethoprim, methotrexate,	Leucovorin(Folinic acid)
27.	Penicillamine	Lead, copper, mercury,

2.5. Disposition

- Following initial evaluation, treatment, and a short period of observation, disposition of the patient is based upon the observed and predicted severity of toxicity.
- Patients who develop only mild toxicity and who have only a low predicted severity can be observed in the emergency department until they are asymptomatic.
- An observation period of four to six hours is usually adequate for this purpose.
- Patients with moderate observed toxicity or those who are at risk for such based on history or initial laboratory data should be admitted to the wards for continued
- Monitoring and treatment.
- Patients with significant toxicity should be admitted to an ICU.
- All patients with intentional overdose require psychiatric assessment prior to discharge.

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