MANAGEMENT OF SHOCK

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SHOCK

• Clinical syndrome that results from circulatory failure, reduction in oxygen delivery, inadequate peripheral tissue and oxygen perfusion leading to cellular hypoxia.

• Shock clinically is characterized by hypotension, SBP<90 mmhg or < 30 mmhg, mean arterial pressure< 655 mmhg, Oliguria, Altered mental status, organ failure.

Stages of Shock
1. Compensated (non-Progressive) shock
2. Uncompensated (Progressive) shock
3. Irreversible (refractory) shock
• Compensated is reversible stage during which compensatory mechanisms are effective and homeostasis is maintained. Metabolism changes at the cellular level from aerobic to anaerobic causing the lactic acid builds up which is removed by liver, but needs oxygen.

• Uncompensated stage begins when the body’s compensatory mechanisms fail. Aggressive interventions are needed to prevent MODS Syndrome.

• Irreversible is the final stage of shock, decreased perfusion, decreased cardiac output, exacerbate anaerobic metabolism, Lactic acid accumulates. Increased capillary permeability allows fluid to move into interstitial spaces.
Hypovolemic Shock

• Resulting from a decreased circulating blood volume.

• **Caused by** diarrhoea,
• vomiting, dengue, DKA,
• acute perforated appendicitis,
• Abdominal open trauma,
• closed fracture, APH, PPH,
• Hyperemesis gravid arum,
• Excessive diuresis,
• Major open surgeries
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood loss mL</td>
<td>&lt;750</td>
<td>750-1500</td>
<td>&gt;1500-2000</td>
<td>&gt;2000</td>
</tr>
<tr>
<td>Blood loss %</td>
<td>&lt;15</td>
<td>15-30</td>
<td>&gt;30-40</td>
<td>&gt;40</td>
</tr>
<tr>
<td>Heart rate (beat/min)</td>
<td>&lt;100</td>
<td>&gt;100</td>
<td>&gt;120</td>
<td>&gt;140</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>Normal</td>
<td>Normal</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>Normal</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Capillary refill normal</td>
<td>Delayed</td>
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<td>Delayed</td>
<td>Delayed</td>
</tr>
<tr>
<td>Respiratory rate (min)</td>
<td>14-20</td>
<td>20-30</td>
<td>30-40</td>
<td>&gt;35</td>
</tr>
<tr>
<td>Urine output (mL/h)</td>
<td>&gt;30</td>
<td>20-30</td>
<td>5-15</td>
<td>Minimal</td>
</tr>
<tr>
<td>Mental</td>
<td>Slightly</td>
<td>Anxious</td>
<td>Confused</td>
<td>Confused</td>
</tr>
<tr>
<td></td>
<td>COMPENSATED</td>
<td>DECOMPENSATED</td>
<td></td>
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<tr>
<td>--------------------------</td>
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<td>-----------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mental status</td>
<td>Anxiety</td>
<td>Lethargy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skin</td>
<td>Pale</td>
<td>Pale, cold, sweaty</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breathing</td>
<td>&lt;30/min</td>
<td>&gt;30/min</td>
<td></td>
<td></td>
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<tr>
<td>Pulse</td>
<td>&lt;120/min</td>
<td>&gt;120/min</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood pressure</td>
<td>&gt;100 mm Hg</td>
<td>&lt;100 mm Hg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>First aid</td>
<td>1. Lying position</td>
<td>1. Lying, elevated legs</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>2. Stop bleeding</td>
<td>2. Stop bleeding</td>
<td></td>
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<tr>
<td></td>
<td>3. I.V. fluid</td>
<td>3. I.V. fluid (NOT oral), blood transfusion</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>4. Oxygen mask</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>5. Prevent hypothermia</td>
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</tbody>
</table>
Fluid administration

- **Crystalloids**: increase intravascular volume through actual volume administered
- **Colloids**: pull fluid into the vascular space through osmosis

Fluid administration

- **Isotonic**: similar in composition to body fluid. Provides greater intravascular volume due to more fluid staying in the vascular space

- **Hypotonic fluid**: shift fluid into intracellular spaces. Useful in preventing cellular dehydration. They deplete circulatory volume

- **Hypertonic**: move fluid from cells to extravascular space, may be used to replace electrolytes and promote diuresis

Blood products (natural colloids)

- Fresh frozen plasma: contains all clotting factors. Used as a blood volume expander

- Albumin: preferred as volume expander when risk from producing interstitial edema is great (pulmonary and heart disease)

Blood products (natural colloids)

- Packed Red blood cell’s: Administer with normal saline
  - Increases oxygen affinity for hgb, and decrease oxygen delivery to the tissues
  - May cause: hypothermia, hyperkalemia, or hypocalcemia

- Whole blood: can be administered without normal saline, reduces donor exposure
  - May require greater amt than packed RBC’s to increase oxygen-carrying capacity of blood
  - Not cost effective. Rarely used
Fluid Resuscitation

**Fluids loss**
- Fluids replacement: (NS) to restore the circulatory volume
  - Adult: at least 1000ml over 30 minutes bolus
  - Pediatrics: 20ml/kg
- Calculating the % loss
  - According to the sign and symptom
    - Dehydration: mild, moderate, severe
    - Blood loss: class I, II, III, IV
  - According to weight loss
    - (Previous healthy weight - current body weight) x 100%

**Fluids maintenance**
- Fluids maintenance: daily fluid loss (about 2L) + additional fluid deficit + ongoing loss
  - (fever - increase in 1 degree Celsius = 10ml/hr loss)
- Paediatrics age group - Must use Holliday-Segard Formula
- Adult - can use wt + 40 formula
- Maximum fluid maintenance for normal daily loss: 120ml/hr
## Hypovolemic Shock

### Nursing Management
- Ensure a patent airway *(always #1)*
- Make sure client has patent IV access
  - If they need something in an emergency you want them to have a patent line.
- Administer oxygen
- Place client in Modified Trendelenburg
- If overt bleeding, apply pressure to the site
- Monitor vital signs every 5 minutes
  - Those vitals can change very quickly.
- Administer meds as ordered
- Increase the rate of fluid delivered

### Rule of 4-2-1 (Holliday-Segard Formula)
- 4 ml per kg for the first 10 kg of body weight;
- 2 ml per kg for the next 10 kg (11-20 kg);
- 1 ml per kg for any weight >20 kg

### Weight + 40

<table>
<thead>
<tr>
<th>Rule of 4-2-1 (Holliday-Segard Formula)</th>
<th>Weight + 40</th>
</tr>
</thead>
<tbody>
<tr>
<td>- 4 ml per kg for the first 10 kg of body weight;</td>
<td>- 2 ml per kg for the next 10 kg (11-20 kg);</td>
</tr>
<tr>
<td>- 2 ml per kg for the next 10 kg (11-20 kg);</td>
<td>- 1 ml per kg for any weight &gt;20 kg</td>
</tr>
<tr>
<td>Example: Calculating maintenance fluid requirements for 70 kg male. 0-10 kg: 10 * 4 ml = 40 mL 11-20 kg: 10 * 2 mL = 20 mL 21-70 kg: 50 * 1 mL = 50 mL Total = 110 mL/hr</td>
<td>Example: Calculating maintenance fluid requirements for 70 kg male. 70+40 = 110 mL/hr</td>
</tr>
</tbody>
</table>
**DEFINITION**

- Cardiogenic shock is a state of systemic hypotension persisting >30 minutes, with reduced end-organ perfusion due to low cardiac output despite adequate filling pressures.

**Causes of cardiogenic shock**

- Acute MI
- LV Systolic failure
- RV infraction
- Papillary muscle rupture (1%)
- Acute VSD (1-3%)
- Free wall rupture (1-6%)
Management

- Rapid correction of haemodynamic compromise is essential, to avoid organ damage from hypoperfusion: ATN, MI, extension, shock liver

GENERAL MEASURES

- Management of reversible cause: 5H’s, 5T’s
  - Hypoxia, hypovolemia, hypokalemia, hypothermia, hydrogen ion, tamponade, t.pneumothorax, toxins, thrombosis
- Maintain SBP >90mm Hg and PCWP <20mm Hg
- Correct hypoxia, acidosis - ventilatory support if required.
- Control arrhythmias - brady or tachyarrhythmias
- Control hyperglycemia by insulin.
**Improvement of CO**

- If condition persists despite optimal LV filling, *inotropic support* is usually needed.
- High PCWP, in the presence of shock, necessitates *inotropic* or mechanical support.

**MECHANICAL SUPPORT**

- INTRACORONARY BALLOON PUMP (IABP)
- PERCUTANEOUS VENTRICULAR ASSIST DEVICES (pVAD)
- EXTRACORPOREAL MEMBRANE OXYGENATION (ECMO)

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**Tandem Heart LVAD-Extracorporeal devices**

- **Left atria-to-femoral arterial LVAD**
  - Low speed centrifugal continuous flow pump
  - Maximum flow 4L/minute

![Diagram of Tandem Heart LVAD](image)
Definition

- Septic Shock:
  - sepsis-induced hypotension (systolic blood pressure <90 mm Hg [or a drop of >40 mm Hg]) with
  - signs of tissue hypoperfusion
  - despite adequate fluid resuscitation for at least 1 hour
- Refractory septic shock
  - Septic shock that lasts for >1 h and does not respond to fluid or pressor administration
- Principle of mechanism
  1. Peripheral vasodilation and pooling of blood
Signs and Symptoms

- Symptoms: FEVER/hypothermia, depends on site of infection.
- Signs:
  - Warm peripheral extremities (due to vasodilation)
  - Febrile
  - Hypotension
  - Tachypnea, tachycardia
  - Oliguria
  - Rash
- History taking: comorbidities
  - DM,
  - Chronic lung disease
  - Alcoholism,
  - Liver cirrhosis,
  - Recent invasive procedure (especially in CKF)
  - HIV
  - Immunosuppressive agent (Steroid)
  - Malignancy
Management of Septic Shock

Management

Hemodynamic Instability

Infection

Fluid Challenge

- Mainstay of hemodynamic supports
- Fast and rapid wide bore fluid resuscitation
- Urine output rate should be kept at >0.5 mL/kg per hour by continuing fluid administration
- Central venous pressure should be maintained at 8–12 cmH₂O

Rate of administration should be reduced if cardiac filling pressure increase without concurrent hemodynamic improvement
Management of hemodynamic instability

ABC

Continuous ECG, BP, HR, Pulse oximetry monitoring

Bladder catheterization

Pulmonary arterial catheterization

Watch I/O carefully and be aware of other losses

Management of Infection

• C&S before empirical antibiotic

• Intravenous broad-spectrum antimicrobials should be initiated immediately (preferably <30 minutes) following the clinical diagnosis

• At dosing at the high end of the therapeutic range

• Duration of therapy: 7-10 days

• Empiric antimicrobial therapy should be adjusted to a narrower regimen within 48 to 72 hours if a plausible pathogen is identified or patient stabilizes clinically. Where possible, early source control should be implemented.
Septic Shock

- Nursing Management
  - Asepsis and hygiene
  - Culture & Sensitivity
  - Parenteral therapy and medication

Anaphylactic Shock

- An allergic, IgE mediated, hypersensitivity response to a foreign substance to which a patient has been previously sensitized
- Type I hypersensitivity
- Causes:
  - Drugs: penicillin, aspirin, streptomycin
  - Vaccines: measles
  - Blood products
  - Insect bites: bees
  - Food: seafood
Clinical Features

Onset:
- Commonly: 5-60min of exposure
- Delayed onset: after few hours
- Biphasic response: recurrence of symptoms 1-8 hrs later due to late phase reaction
- Protracted anaphylaxis: persistence of symptoms up to 48hrs despite therapy

Skin:
- Urticaria (200 cases): Area of focal dermal edema
- Angioedema (20 cases): Localized non-pitting deeper edematous process
- Pruritus
- Tingling of face (usually at mouth)
Angioedema

Urticaria
ABC
Bladder catheterization
ECG, RR, BP, SaO2
recumbent position

Remove the inciting agent

- High flow Oxygen with facemask → fail → ETT → difficult intubation due to severe laryngeal edema →
  - Tracheostomy / cricothyroidotomy proximally
  - Insect: flick out insect stinger with a tongue blade
  - Ingestion of allergen: gastric lavage and activated charcoal
Administer histamine antagonists in patients who remain hypotensive despite epinephrine.

- Block vasodilation, capillary leak, and shock
- $H_1$ blockade, 25–50 mg of diphenhydramine IV 6hrly
- $H_2$ blockade, 50 mg of ranitidine IV 6hrly
- Crystalloid or colloid

Aggressive fluid resuscitation

Still hypotension
IM aqueous epinephrine 0.3-0.5 ml of 1:1000

Repeat every 20 minutes if require multiple doses

Severe airway compromise / hypotension

IV Epinephrine 3 - 5ml 1:1000

Repeat every 5-10 min

Epinephrine Infusion

- Epinephrine is the mainstay of initial management and controlling symptoms and maintaining blood pressure.
Still Hypotension

Pulmonary artery catheterization

Inotropes

- Maintain MAP > 60-65 mmHg
- dopamine, isoprenaline infusion
Neurogenic Shock

- Causes:
  - Post-spinal surgery
  - Spinal injury
- Clinical features:
  - Bradycardia, hypotension, warm peripheral extremities

Obstructive Shock

- Causes:
  - Tension pneumothorax
  - Cardiac tamponade
  - Pulmonary embolism
Anaphylactic Shock

- **Nursing Implications**
  - Assess for allergies
  - Communication
  - Knowledgeable about s/s (and how to deal with them should they arise)
  - Teach about future exposures (and inform the families also so they can help)

Neurogenic Shock

- **Nursing management**
  - Elevate and maintain HOB 30 degrees
    - Most everyone on a neuro floor has the HOB up 30
  - Support cardiovascular and neurologic function
  - Prevent blood pooling in lower extremities
    - Apply TED hose
    - Prevent DVTs

- **Clinical features:**
  - Bradycardia, hypotension, warm peripheral extremities
- **Mx:**
  - ABC + Supine position with leg elevated
  - Fluid resuscitation
  - NE
  - Anal wink or bulbocarvenosus reflex
Nursing Assessment (Cont’d)

- **Brief history**
  - Events leading to shock
  - Onset and duration of symptoms
- **Details of care received before hospitalization**
- **Allergies**

Nursing Assessment (Cont’d)

- **CABs**: circulation, Airway, breathing, and Focused assessment of tissue perfusion
- **Vital signs**
- **Peripheral pulses**
- **Level of consciousness**
- **Capillary refill**
- **Skin** (e.g., temperature, color, moisture)
- **Urine output**

Nursing Implementation

- **Health Promotion**
  - Identify patients at risk (e.g., elderly patients, those with debilitating illnesses or who are immunocompromised, surgical or accidental trauma patients)
  - Planning to prevent shock (e.g., monitoring fluid balance to prevent hypovolemic shock, maintenance of handwashing to prevent spread of infection)

Nursing Implementation (Cont’d)

- **Acute Interventions**
  - Monitor the patient’s ongoing physical and emotional status to detect subtle changes in the patient’s condition
  - Plan and implement nursing interventions and therapy
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  - Monitor the patient’s ongoing physical and emotional status to detect subtle changes in the patient’s condition
  - Plan and implement nursing interventions and therapy

- Respiratory status
  - Respiratory rate and rhythm
  - Breath sounds
  - Continuous pulse oximetry
  - Arterial blood gases
  - Many patients will be intubated and mechanically ventilated

- Neurologic status: Orientation and level of consciousness
- Cardiac status
  - Continuous ECG
  - VS, capillary refill
  - Hemodynamic parameters: central venous pressure, PA pressures, CO, PAWP
  - Ongoing assessment of CO

- Urine output
- Tympanic or pulmonary arterial temperature
- Skin: Temperature, pallor, flushing, cyanosis, diaphoresis, piloerection
- Bowel sounds
Nursing Implementation (Cont’d)

- Nasogastric drainage/stools for occult blood
- I&O, fluid and electrolyte balance
- Oral care/hygiene based on O2 requirements
- Passive/active range of motion

- Assess level of anxiety and fear
  - Medication PRN
  - Talk to patient
  - Visit from clergy
  - Family involvement
  - Comfort measures
  - Privacy

Others

- Prevention of stress ulcer
  - Ranitidine or PPI
- Prevention of deep vein thrombosis
  - UF heparin or LMW heparin if no C/I
- Prevention of ARF
  - Induce diuresis by furosemide (make sure adequate fluid therapy) → look for hyperkalemia
  - IV 2-5micro g/kg/minute of dopamine (low dose)
- Glucose control
  - Insulin to prevent DKA in DM patient
- Metabolic Acidosis
  - treat in severe cases only.

Complications of Shock

- CNS
  - Encephalopathy
- CVS
  - Reduced myocardial contractility
- Renal
  - Acute Renal Failure
- Pulmonary
  - ARDS
  - Atelectasis
- GIT
  - Stress Ulcer
  - Mesenteric Ischemia
  - “Shock” liver
- Hematology
  - DIC
- Metabolic
  - Hyperglycemia
  - Lactic Acidosis
- Skeletal
  - gangrene
MANAGEMENT OF POISONING
Poison

- A poison is any substance that is harmful to the body.
- Many poisonous substances are products people have around the house. Even medicines that aren't taken as directed can be harmful.

Poisoning

- Poisoning is a common medical emergency in any country.
- Poisoning occurs when any substance interferes with normal body functions after it is swallowed, inhaled, injected, or absorbed.
POISON

- A Poison is any chemical that harms the body
- It can be
  - Accidental
  - Occupational
  - Recreational
  - Intentional (killing)
  - Natural or Manufactured toxins
Routes of Poisoning

- Inhalation
- Absorption
- Injections
- Ingestion
1. According to the site and mode of action (A) LOCAL ACTION

**Corrosive:**

Strong Acid: mineral acid: eg H₂SO₄, HCl: organic acid: Carbolic, oxalic, acetic, salicylic

*Strong alkali:* Hydrates and carbonates of Na, K, & ammonia

Metallic salts: Mercuric Chloride, KCN

**Irritant**

1) **Agricultural**
2) **Inorganic**
   - Nonmetallic: P, Iodine, Cl, bromine
   - Metallic: Arsenic, Antimony, Pb, Cu, Zinc
   - Mechanical: Glass, Diamond dust, Hair
3) **Organic**
   - Animal: Snakes, insects, Cantharides
   - Vegetable: Abrus, Castor, Croton, Calotropis


Main types of poisons

- Acids
- Alkalies
- Medication
- Metal poisoning
- Organophosphorous poisoning
- Petroleum products
- Oils
Acids
- Nitric acids
- H2SO4
- HCL
- Carbolic acid
- Acetic acid

Alkalies
- Drain cleaners
- Dishwashing – detergents, ammonia
- Bleacher
Medication

- Aspirin & Aspirin containing medications
- NSAIDS
- Hallucinogen
- Barbiturates
- Alcohol
- (in homeo treatment: spirit)

Metal poisoning

- Iron
- Copper
- Cyanide
- Lead
- Ethylene glycol
OP poisoning

- Insecticides naphthalene
- Pesticides
- Opium
- Castor oil
- Mushroom
- Tobacco
- Cannabis
baby with birth defects attributable to pesticides (PRP)
Principle of Management

1. Initial resuscitation and stabilization
2. Removal of toxin from the body
3. Prevention of further poison absorption
4. Enhancement of poison elimination
5. Administration of antidote
6. Supportive treatment
7. Prevention of re-exposure
1. Resuscitation and initial stabilization

- Airway
- Breathing
- Circulation (consider stabilizing the C-spine)
- **D1** Drugs
- ACLS as necessary to resuscitate the patient
- universal antidotes
- **D2** Draw bloods
- **D3** Decontaminate
- Expose (look for specific toidromes)/Examine the Patient
- Full vitals, ECG monitor, Foley, x-rays, etc...
- Give specific antidotes
2. Diagnosis of type of poison

- History :
- Examination :
  - head-to-toe
- Laboratory Investigations :

**Examination**

- signs of trauma
- signs of seizures
- signs of infection (meningitis)
- signs of chronic alcohol abuse
- signs of drug abuse
- mental status
- Specific toxidromes

**Hx content of poisoned patient**

- Identification of the patient and toxic agent.
- **What?** Description of the toxin.
  Product names (brand, generic, chemical) and ingredients, along with their concentrations

- **Bring container to hospital with patient.**

- **How much?** Magnitude of the exposure.
  Determine as accurately as possible how much of the substance has been consumed by counting the remaining tablets or measuring the remaining volume of liquid or gas after we ask the previous amount.

- **It is better to overestimate than to underestimate**
3. Nonspecific therapy

- Gastric Decontamination
  - emesis, gastric lavage, and use of activated charcoal and cathartics
- Whole bowel irrigation
- Enhancing Excretion
  - Forced alkaline diuresis
  - Dialysis: Peritoneal and haemodialysis

❖ Progression of symptoms.
Knowing the nature and progression of symptoms is very helpful for assessing the need for immediate life support, the prognosis, and the type of intervention needed

❖ What interventions have been done?
  – Traditional home remedies may be harmful
Gastric Emptying

- Emesis: achieved by using syrup of ipecac
  - Dosing: 15 ml for 1-12 yo and 30 ml for adults; may repeat once if no emesis in 12 hr
- 90% vomit within 20 minutes of first dose and 97% vomit with second dose
- Usually 3-5 episodes of emesis and resolve in two hours; if protracted emesis occurs consider toxin as etiology

Activated Charcoal

- Most appropriate agent to decontaminate GI tract
- Adsorbs toxin in gut lumen
- Safety proven in adults and children
- Dose 1g/kg
- Indications: any drug known to absorb it or after unknown ingestions by patient’s with protected airways
Multi-Dose Charcoal

- One dose usually sufficient
- Indications for multi-dose activated charcoal: ingestion of large doses, substances that form bezoars, slow release toxins, toxins that slow gut function, toxins with enterohepatic or enteroenteric circulation
- Repeat dose is 0.25-0.5 g/kg

Cathartics

- Osmotic cathartic usually given with activated charcoal
- 70% sorbitol (1 g/kg) or 10% magnesium citrate
- Shown to decrease transit time of activated charcoal
- No definitive clinical human data suggest that a cathartic limits toxins bioavailability or changes patient’s outcome
Alkalization

- Beneficial in certain ingestions: 2-4-D (herbicide), phenobarbital, chlorpropamide, salicylates, methanol
- Alkalization achieved by IV dose of bicarbonate at 1-2 mEq/kg, followed by intermittent boluses or continuous bicarbonate drip for urine pH 7.5-8.0
- Profound hypokalemia may result, must aggressively replace

Hemodialysis/Hemoperfusion

- Dialysis reserved for specific toxins: salicylates, methanol, ethylene glycol, lithium, theophylline, amanita (mushrooms)
- Benefits: removal of toxins already absorbed by gut, ability to remove parent compound and active metabolite,
- Less effective when toxin has large volume of distribution (>1 L/kg), has large molecular weight, or highly protein bound
## 4. Specific therapy

<table>
<thead>
<tr>
<th>Toxin</th>
<th>Antidote</th>
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<tbody>
<tr>
<td>Acetaminophen</td>
<td>N-acetylcysteine</td>
</tr>
<tr>
<td>Anticholinergics</td>
<td>Physostigmine</td>
</tr>
<tr>
<td>Arsenic/Lead</td>
<td>BAL chelation</td>
</tr>
<tr>
<td>B-Blockers</td>
<td>Glucagon</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td>Flumazenil</td>
</tr>
<tr>
<td>CO</td>
<td>O₂, HBO</td>
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<tr>
<td>Cyanide</td>
<td>Nitrites</td>
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<tr>
<td>Digoxin</td>
<td>Digibind</td>
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<tr>
<td>Ethylene glycol/Methanol</td>
<td>Fomepizole/Ethanol</td>
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<tr>
<td>Iron</td>
<td>Deferoxamine</td>
</tr>
<tr>
<td>INH</td>
<td>B₆/Pyridoxine</td>
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<tr>
<td>Lead/Mercury</td>
<td>Succinimide</td>
</tr>
<tr>
<td>Methemoglobinuria</td>
<td>Methylene blue</td>
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<tr>
<td>Opioids</td>
<td>Naloxone</td>
</tr>
<tr>
<td>Organophosphates</td>
<td>Atropine</td>
</tr>
<tr>
<td>TCA’s</td>
<td>Sodium bicarbonate</td>
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</tbody>
</table>
5. Supportive care

- most cases of poisoning is largely supportive.
- It is important not to waste time in locating an antidote.
- preserve the vital organ functions till poison is eliminated.
- proper care for coma, seizures, hypotension, arrhythmias, hypoxia, and acute renal failure.
ORGANOPHOSPHATE POISONING

- Organophosphates (pesticides and nerve agents) irreversibly bind and deactivate cholinesterases, including acetylcholinesterase.

- Acetylcholine accumulates at neural synapses, causing central and peripheral cholinergic overdrive.

- Mortality is secondary to respiratory failure: Weakness of respiratory muscles
  Bronchorrhea and bronchoconstriction
  Central depression of respiratory drive
Pediatric Considerations

- Symptoms are difficult to differentiate in toddlers
- Common symptoms: Miosis, salivation, and muscle weakness
- Seizure activity in 25% of pediatric cases:
- Only 3% in adults

Classic presentation: Cholinergic toxidrome:

DUMBELS:
- Diarrhea/diaphoresis
- Urination
- Miosis/muscle fasciculations
- Bradycardia, bronchorrhea, bronchospasm
- Emesis
- Lacrimation
- Salivation
May have garlic odor
• **CNS:** Muscle fasciculation then flaccid paralysis, Respiratory muscle weakness, Incoordination and ataxia, Agitation, Tremors, Confusion

• **Visual:** Pinpoint nonreactive pupils

• **Respiratory:** Respiratory muscle weakness, Bronchorrhea

• **Cardiovascular:** Bradycardia

• **GI:** Nausea/vomiting, Abdominal cramps

• RBC and plasma cholinesterase levels to confirm diagnosis:

• RBC (true) cholinesterase level is best for synaptic inhibition.

• Plasma (pseudo) cholinesterase level not as reliable but more timely:

• These are markers for poisoning

• Depending on the agent and the patient, these levels may vary
TREATMENT

PRE HOSPITAL
• Decontamination is initial priority:
• Remove all clothes and store as toxic waste
• Decontaminate skin with soap and water:
• INITIAL STABILIZATION/ THERAPY
• Maintain airway and oxygenate.
• For unstable airway, intubate, and ventilate.
• IV access with D5W 0.9% NS
• Altered mental status: Administer thiamine, glucose, and naloxone

ED TREATMENT
• Atropine: Administer test dose 1–2 mg IV/IM:
• No clinical response: Double dose q5min until muscarinic findings subside
• Dose: 1–4 mg IV q5min (peds: 0.05–0.2 mg/kg)
• Pralidoxime: 1–2 g (peds: 25–50 mg/kg) dissolved in 0.9% NS over 30 min IV; then q6h as needed:
Aluminum phosphide poisoning

- Aluminum phosphide poisoning is known worldwide, especially in developing countries like India and Iran.
- Lethal dose of ALP is 1–1.5 g. Deaths are reported even with a dose of 150–500 mg.

- Severe toxicity of ALP particularly affects the cardiac and vascular tissues, which manifests as profound and refractory hypotension, congestive heart failure, ECG abnormalities, myocarditis, pericarditis and subendocardial infarction.
- The frequency of hypotension varied from 76% to 100%, which is a cardinal feature in ALP toxicity.
Suspected accidental inhalation/overexposure at workplace

- Open area/Fresh air
  - Maintain airway, breathing, circulation

- Early arrival to ERS
  - History, examination
  - Monitoring of vitals

  Suspected AIP poisoning
  ± silver nitrate test
  - Consult regional poison centre
  - Gut decontamination
    (gastric lavage with KMnO₄, activated charcoal, coconut oil)

- Routine investigation to detect organ insult
  - Watch for worsening of sign/symptoms
  - Provide appropriate advanced support

- Arrhythmias
  - Anti-arrhythmic agents
    ± MgSO₄

- Shock
  - CVP/PAWP guided fluid therapy
    ± vasoactive agents
    ± pacemaker
    ± mechanical support of heart (IABP)

- ALI/ARDS
  ± ventilatory support

- Metabolic acidosis
  ± Sodium bicarbonate
  ± hemodialysis

- Others
  - Maintain electrolytes & blood sugar level
    ± MgSO₄
    ± steroids, vit C, NAC

Note: CVP = central venous pressure, PAWP = pulmonary artery wedge pressure, IABP = intra-aortic balloon pump, ECLS = extra corporeal life support, ALI/ARDS = acute lung injury/acute respiratory distress syndrome, NAC = N-acetylcysteine
Hydrocarbon Poisoning

- Most commonly ingested hydrocarbons - gasoline, lubricating oil, motor oil, mineral spirits, lighter fluid/naphtha, lamp oil, and kerosene.
- Other common sources of hydrocarbons - dry cleaning solutions, paint, spot remover, rubber cement, and solvents

- Vomiting & diarrhea, which may be bloody
- Dyspnea and cyanosis
- Mild tracheobronchitis, severe necrotizing bronchopneumonia & pul. hemorrhage
- Atelectasis, pneumatocele, bacterial infection (secondary), pneumomediastinum
- Mild to moderate fever within 48 hours
- CNS - tremors, irritability, confusion, drowsiness, sz, & coma
Paracetamol (Acetaminophen) Poisoning

- mostly used & available at home
- analgesic and antipyretic
- overdose cause fatal and nonfatal hepatic necrosis with certain risk factors, but is nearly always good if the antidote, N-acetylcysteine (NAC), is administered within 8 to 10 hours of ingestion

- Correction of hypoxia & acidosis
- NO emesis
- NO lavage
- NO prophylactic Antibiotic
- NO STEROIDS
Patterns of exposure could be:

1) Intentional: more common in older children and adolescents with a single event and high dose.
2) Unintentional: common among younger children, occur through "exploratory" behavior or inappropriate dosing

- Clinical Manifestations:
  - if untreated, poisoned patient passes through 4 stages:
    - Stage I (up to 24 hrs) – Asymptomatic, but less commonly: nausea, vomiting, and, in large doses, lethargy and malaise
    - Stage II (24 to 72 hours after overdose) – RUQ pain, elevation in liver enzymes, prothrombin time (PT) and, in severe cases, evidence of nephrotoxicity (elevated blood urea nitrogen, creatinine, oliguria) and/or pancreatitis (elevated serum amylase, lipase)
• Stage III (72 to 96 hrs) – Evidence of liver failure and, in severe cases, renal failure and multi-organ failure; death most commonly occurs in this stage

• Stage IV (4 to 14 days) – Recovery

Management:
- Supportive
- Emesis/lavage
- NAC/Mucomyst
PHENOBARBITONE POISONING

- **Pathophysiology**
  - 50% of phenobarbitone is non-protein-bound, available to equilibrate with tissues.
  - Ability to cross cell membrane (BBB) is inversely correlated with its degree of ionization.
  - Cause depression of the brainstem RAS with resultant generalised depression of the CNS.

- **May result in:**
  - Mild sedation, sleep or
  - High doses—coma, & respiratory arrest
  - Absorbed from oral ingestion
  - Onset of effects in 20-60 min.
  - Slowly metabolized by liver microsomal enzymes & are eliminated—half-lives of 2-6 days.
Snake Bite

- Majority of the bites being on the lower extremities.
- Males:Female: 2:1
- 50% of bites by venomous snakes are dry bites that result in negligible envenomation.
- In the world 3000 species, 500 poisonous
Early Signs and Symptoms of Venomation

- Increasing local pain (burning, bursting, throbbing) at the site of the bite
- Local swelling that gradually extends proximally up the bitten limb and tender
- Painful enlargement of the Regional Lymph nodes.

However, bites by kraits and sea snakes may be virtually painless.

Local Symptoms and Signs

- Local pain
- Local bleeding
- Bruising
- Lymphangitis
- LN Enlargement
- Blistering
- Local infection & Abscess formation
- Necrosis
Local Symptoms and Signs

- Local pain
- Local bleeding
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- Necrosis

Systemic Symptoms

General
- Nausea
- Vomiting
- Malaise
- Abdominal pain
- Weakness
- Drowsiness
- Prostration
Management of Snake Bite

- First aid treatment
- Transport to hospital
- Rapid clinical assessment and resuscitation
- Detailed clinical assessment and species diagnosis
- Investigations/laboratory tests

- Antivenom treatment
- Observation of the response to antivenom:
  - decision about the need for further dose(s) of antivenom
- Supportive/ancillary treatment
- Treatment of the bitten part
- Rehabilitation
- Treatment of chronic complications
• Zinc Phosphide is a highly effective rodenticide, dark in colour usually mixed with food for catching rats.

• If ingested it reacts with water and acid in the stomach and produces phosphine gas – it disrupts mitochondrial function through blocking cytochrome C oxidase.

• Phosphide produces toxicity rapidly with in 30 minutes of ingestion, and death may follow with in 6 hours.
• Phosphide ingestions over 500 mg are often fatal. It is manifested by Vomiting, abdominal pain, tachypnoea, hyperapnoea, dyspnoea, cough, chest tightness, hepatomegaly, raised transaminase, metabolic acidosis.

Management:
Checking PT – INR every 24 hours to ensure no effect on coagulation pathway, if coagulopathy is documented, Vitamin – K, Fresh Frozen Plasma may be needed.

PYRETHROID TOXICITY

• Pyrethroids are insecticides that are synthetic modifications of natural pyrethrins. Highly effective in insects, comparatively low toxic to mammals.

• These are ion channel toxins acts on neuronal voltage- sensitive voltage channels thereby by prolonging neurological excitation.
Clinical features

<table>
<thead>
<tr>
<th>Mild pyrethroid toxicity</th>
<th>Moderate pyrethroid toxicity</th>
<th>Severe pyrethroid toxicity</th>
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<tr>
<td>Paresthesia</td>
<td>CNS depression</td>
<td>Seizures</td>
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<td>Nausea</td>
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<td>Headache</td>
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<td>Dizziness</td>
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<td>Fatigue</td>
<td>Blurred vision</td>
<td></td>
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<tr>
<td>Anorexia</td>
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</tbody>
</table>

Treatment

- There is no antidote for pyrethroid poisoning.
- Treatment consists in preventing further exposure together with supportive and symptomatic measures.
- In case of dermal exposure rinse the skin with abundant water and soft detergents.
- After accidental ingestion administer activated charcoal (2g/kg).
- Spasms can be treated with anticonvulsants (e.g. diazepam). If ineffective, fenobarbital or pentobarbital can be tried.
- Hypersalivation can be treated with atropine.
CORROSIVE POISONING

• Corrosives are the group of chemicals that have the capacity to cause tissue injury on contact by a chemical reaction. Commonly affects GIT, Resp. System an Eyes.

• Acids
  • Car battery fluid (sulfuric acid)
  • Descalers (hydrochloric acid)
  • Metal cleaners (nitric acid)
  • Rust removers (hydrogen fluoride)
• Alkalis
  • Bleach (hypochlorite)
  • Sodium hydroxide (liquid lye)
MANAGEMENT

- Early Admission: With in 48 – 72 hours of corrosive ingestion upper GI endoscopy to be performed on day 1 – 2. Endoscopy mild lesions – medicines and discharge, if found severe lesions Gastrostomy to be done.

- Delayed Admission: With in 72 hrs to 3 weeks of corrosive ingestion no endoscopy is indicated. Gastrostomy is indicated in severe dysphagia. If stricture present endoscopic dilatation can be done.

- Late Admission: More than 3 weeks of ingestion Endoscopic dilatation of the stricture is advised if the procedure is unsuccessful Surgical Gastrostomy is indicated.