MOST FREQUENT INTOXICATIONS

Igor Batora

The study of the adverse effects of a toxicant on living organisms

- **Toxicant (Poison)**
  - any agent capable of producing a deleterious response in a biological system
  - a sac of water with target sites, Living organism
  - storage depots and enzymes

- **Adverse effects**
  - any change from an organism’s normal state
  - dependent upon the concentration of active compound at the target site for a sufficient time.
What is a Poison?

All substances are poisons; there is none that is not a poison.
The right dose differentiates a poison and a remedy.

Paracelsus (1493-1541)
Philippus Theophrastus Bombastus Von HOHENHEIM
PARACELSUS (1493-1541) :

„Sola dosis facit venenum“
Dose

The amount of chemical entering the body
This is usually given as
mg of chemical/kg of body weight = mg/kg
The dose is dependent upon
* The frequency of exposure
* The environmental concentration
* The properties of the toxicant
* The length of exposure
* The exposure pathway
Dose-Response Relationship: As the dose of a toxicant increases, so does the response.

DOSE DETERMINES THE BIOLOGICAL RESPONSE

0-1 NOAEL
2-3 Linear Range
4 Maximum Response
Exposure: Pathways

- Routes and Sites of Exposure
  - Ingestion (Gastrointestinal Tract)
  - Inhalation (Lungs)
  - Dermal/Topical (Skin)
  - Injection
    - intravenous, intramuscular, intraperitoneal

- Typical Effectiveness of Route of Exposure
  iv > inhale > ip > im > ingest > topical
ADME: Absorption, Distribution, Metabolism, and Excretion

• The body has defenses:

• Once a living organism has been exposed to a toxicant, the compound must get into the body and to its target site in an active form in order to cause an adverse effect.
  – Membrane barriers
    • passive and facilitated diffusion, active transport
  – Biotransformation enzymes, antioxidants
  – Elimination mechanisms
Absorption: ability of a chemical to enter the blood (blood is in equilibrium with tissues)

- **Inhalation**—readily absorb gases into the blood stream via the alveoli. (Large alveolar surface, high blood flow, and proximity of blood to alveolar air)

- **Ingestion**—absorption through GI tract stomach (acids), small intestine (long contact time, large surface area—villi; bases and transporters for others)
  - 1st Pass Effect (liver can modify)

- **Dermal**—absorption through epidermis (stratum corneum), then dermis; site and condition of skin
Distribution:
the process in which a chemical agent translocates throughout the body

- Blood carries the agent to and from its site of action, storage depots, organs of transformation, and organs of elimination
- Rate of distribution (rapid) dependent upon
  - blood flow
  - characteristics of toxicant (affinity for the tissue, and the partition coefficient)
- Distribution may change over time
Distribution: Storage and Binding

- Storage in Adipose tissue---Very lipophylic compounds (DDT) will store in fat. Rapid mobilization of the fat (starvation) can rapidly increase blood concentration
- Storage in Bone---Chemicals analogous to Calcium---Fluoride, Lead, Strontium
- Binding to Plasma proteins---can displace endogenous compounds. Only free is available for adverse effects or excretion
Target Organs: adverse effect is dependent upon the concentration of active compound at the target site for enough time

- Not all organs are affected equally
  - greater susceptibility of the target organ
  - higher concentration of active compound
- Liver—high blood flow, oxidative reactions
- Kidney—high blood flow, concentrates chemicals
- Lung—high blood flow, site of exposure
- Neurons—oxygen dependent, irreversible damage
- Myocardium—oxygen dependent
- Bone marrow, intestinal mucosa—rapid divide
Excretion:
Toxicants are eliminated from the body by several routes

- **Urinary excretion**
  - water soluble products are filtered out of the blood by the kidney and excreted into the urine

- **Exhalation**
  - Volatile compounds are exhaled by breathing

- **Biliary Excretion via Fecal Excretion**
  - Compounds can be extracted by the liver and excreted into the bile. The bile drains into the small intestine and is eliminated in the feces.

- **Milk**    **Sweat**    **Saliva**
Metabolism:
adverse effect depends on the concentration of active compound at the target site over time

- The process by which the administered chemical (parent compounds) are modified by the organism by enzymatic reactions.

- 1° objective -- make chemical agents more water soluble and easier to excrete
  - decrease lipid solubility
    --> decrease amount at target
  - increase ionization
    --> increase excretion rate --> decrease toxicity

- Bioactivation -- Biotransformation can result in the formation of reactive metabolites
Biotransformation

• Key organs in biotransformation
  – LIVER (high)
  – Lung, Kidney, Intestine (medium)
  – Others (low)

• Biotransformation Pathways
  * Phase I--make the toxicant more water soluble
  * Phase II--Links with a soluble endogenous agent (conjugation)
Individual Susceptibility

- Age-old
  - changes in excretion and metabolism rates, body fat
- Nutritional status
- Health conditions
- Previous or Concurrent Exposures
  - additive - antagonistic
  - synergistic
Most frequent poisonings

Medicaments: analgetics (acetaminophen = paracetamol)  
psychopharmac. (benzodiazepines)
Alcohol, illicit drugs (heroin)
Household products (cleaners, solvents...)
Insecticides, plants...mushrooms
Lethality: < 1%
Statistics: overall not complete.
Most calls to PICs: side effects /adverse effects
## Grading of poisonings

<table>
<thead>
<tr>
<th>LD&lt;sub&gt;50&lt;/sub&gt;</th>
<th>Category</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥15 g/kg b.w.</td>
<td>non toxic</td>
</tr>
<tr>
<td>5-10 g/kg</td>
<td>light toxic</td>
</tr>
<tr>
<td>0,5 – 5 g/kg</td>
<td>moderate toxic</td>
</tr>
<tr>
<td>0,05 – 0,5 g/kg</td>
<td>high toxic</td>
</tr>
<tr>
<td>5 – 50 mg/kg</td>
<td>extremely toxic</td>
</tr>
<tr>
<td>&lt; 5 mg/kg</td>
<td>super-toxic</td>
</tr>
</tbody>
</table>
Most frequent symptoms

GI: nausea, vomiting, diarrhea
CNS: somnolence, sopor, coma, excitation, seizures
CVS: arrhythmia, cardiac failure, BP disturbances
Before calling PIC (Poison Control Centre)

Elemental questions:

What
How much
When
Who
Why

Abbreviations: HD hemodialysis, FD forced dialysis; HP hemoperfusion; GL gastric lavage
Paracetamol (*acetaminophene*)

Paralen, Panadol, Tylenol, Ataralgin...

**Effect:**
nausea, vomiting, liver and/or renal failure in 24 hr

toxic metabolite NAPQI \((N\text{-acetyl}-P\text{-benzoquinimine})\) \(\Rightarrow\)
Glutathion depletion

**Toxicity:**
hepatotoxic dose:  \(> 140 \text{ mg/kg b.w.};\) i.e. 5 g (10 tab)
children:  \(> 200 \text{ mg/kg b.w.}\)
Therapy:

Antidote NAC (N-acetylcysteine = glutathione precursor; Broncholysin inj, ACC inj, Fluimucil gra/tab. eff...)

Effectiveness even 16 hr after intake

Criteria for antidote treatment: suspect intake > 7.5 g

Secondary elimination:

*Not alternative to antidote!*

If antidote NAC available overall not necessary

FD, HP not effective

HD only in liver/renal failure
Benzodiazepines

diazepam (DIAZEPAM) flunitrazepam (ROHYPNOL)
midazolam (DORMICUM) oxazepam (OXAZEPAM)
alprazolam (XANAX, NEUROL...)

Mechanism of action:
↑ inhib. effect of GABA in CNS

Effect:
Central nervous + respiratory depression, ↓ BP, ↑T₀C
HEROINE

Mechanism of action:
4 types of receptors in CNS+ medulla
  effects: analgetic, respiration, mood, BP, GIT...

Toxicokinetics: ENTRY
  p.o.
  snorting
  sniffing
  s.c.
  i.m.
  i.v. (lemon juice)
  rectal
Biotransformation:

heroíne → 6-MAM → morphine → M-6-glucuronid

Heroine : prodrug
6-MAM, morphine: active substances
Urine: 6-MAM; morphine; glucur. – morphines; 3 d
No heroine!

„Desirable effect: wellbeing
**Trias**

**Tox. effect:** resp. depression + unconscious + miosis

**Toxicity:** DL non-tolerant: 10 mg  
addictive > 200 mg!  
(standard conc. <10%; Afghanistan: cca 16% !!)

**Dg:** TRIAS  
opioids in urine  
effect of antidote (NALOXONE)

**Therapy:** A-B-C  
Pressure support ventilation (PSV)  
NALOXONE  
Diazepam iv if seizures
Amphetamines:

Methamphetamines (Czech Pervitin...) MDMA (Adam, Extasy) MDEA (Eve) „rave party drug“

Effect: $\uparrow$ conc. NORA, serotonine, dopamine $\rightarrow$ stimul. CNS nervousness, restlessness, insomnia, hallucinations, hyperthermia, seizures, diarrhea, vomiting...

Complications: high BP, tachycardia, DIC, rhabdomyolysis, renal+hepatic failure, pulmonary edema...

Toxicity: 1-5 tab lethal (50 mg)

„New complication“: hyperhydration (hypo-Na,K brain edema)

Therapy: cooling!(ice,DANTROLENE); diazepam,rehydratation
Solvents

DEFINITION

• Liquid compounds or their mixtures that dissolve other substances, generally solids, without any change in chemical composition.
Solvents

General Classification

- petroleum distillates
- ethers and acid esters
- alcohols and their derivatives
Organic solvents:

Most frequent professional exposure: TOLUENE!... benzin, acetone...xylene

„Sniffing of toluene“

Effect: CNS depression, arrhythmia
Toxicity: 1 ml/kg b.w. non-toxic
Therapy: inhal.: oxygen, stop exposure
          p.o. : aspiration of gastric content (if high dose)
          no GL, charcoal no effect!
          FD and HD no effect
Gasoline

• Routes of absorption
  - respiratory system
  - dermal exposure
  - GI tract

**Lethal oral dose** has not been precisely determined and ranges from several dozen to several hundred milliliters.
ETHYL ALCOHOL:

**Toxicokinetics:** max. plasma conc.: 30-60 min after intake
metabolism: 7-10 g/h

**Toxicity:** lethal over 4,5 ‰; survival even 15‰!!

**Therapy:**

NO! 1. Gastric lavage, forced vomiting (fast resorption!)
  2. Carbo adsorbens – charcoal (only in mixtures)
  3. Analeptics, Naloxon
  4. FD (via kidney only 5%)

YES! Sweet drinks/40% gluk i.v./; diazepam if seizures,
warming; thiamine...

**HD:** over 4 ‰, coma, CV failure, mixtures
Glycols \((\text{alcohols with two } -\text{OH})\)

Ethylene Glycol (car radiator antifreeze: FRIDEX, ALYCOL, NEMRAZOL, brake liquid, solvent...)

**Toxicity:**

EG ...glycoaldehyde (CNS)...glycolic acid...glyoxylic acid (acidosis)...oxalic acid (renal failure)

**DL:** 30-90ml (1,5ml/kg b.w.)

**Overdose:** up to 12 h: ethyl alcohol - like

12-24 h: CV failure, ARDS

24-72 h: Renal failure (oxalic acid...crystals)
Diagnosis:

Combinations of symptoms and/or lab findings:

- Ebrieta (tipsy) without alcoholic „foetor ex ore“
- MAC (art.pH<7,3) + anion gap
- unconscious + osmolal gap + crystals + hypo-Ca

Labs EG in blood : not available
Therapy: lavage up to 1hr
Antidot: to prevent biotransformation of toxic substr.

40% EtOH (antidotum) 2ml/kg in juice /15-30min
Later i.v. (blood conc. 1‰); bicarbonates
optimal antidotum: fomepizol (4-MP; ANTIZOL)
If no effect:: HD, CAVHF

Indications for HD:
MAC (pH < 7,25) unresponsive to therapy
Renal failure
Blood EG  >500mg/L
Deteriorating vital signs
Electrolyte imbalances unresponsive to therapy

The same therapy in METHANOL intoxication!!!
Pesticides

Chemicals that are used to kill pests

Insecticides  – compounds that are used to kill insects and related species (e.g. organophosphates, carbamates)

Rodenticides – are used to kill rats (anticoagulants)

Herbicides  – are used to kill weeds (e.g. diquat)

Fungicides  - are used to kill fungi

Fumigants  – gases that are used to sterilize products (e.g. ethylene dibromide)
Pesticides:

Insecticides, rodenticides, fungicides, herbicides, moluscocides...

Most frequent serious human intoxications:

organophosphorous insecticides:

Most frequent not serious human intoxications:

pyrethroids

SOLVENT (VEHICULUM) IS SOMETIMES MORE TOXIC THAN PESTICIDE!

Most toxic pesticides (OP, CARB) penetrate all barriers (skin, mucuous).
Organophosphates:

Forms of intoxications:

1. Acute cholinergic crisis: endogenous Ach intoxications
2. Intermediate Syndrome: 1.-4. day; upper extrem. paralysis
   neck and respiratory mm.
3. OP Induced Delayed Neuropathy: 8. – 14. day; lower extr.
   paralysis, peripheral axon degeneration
4. Chronic OP Induced Neuropsychiatric Disorder: subtle
   impairment of cognitive functions *(different opinions)*
Signs and Symptoms

AChE inhibition

Cholinergic hyperstimulation

Cholinergic toxidrome
Signs and Symptoms

Vapour Exposure

- **Eyes**
  - Miosis, eye pain, headache, injection, lacrimation

- **Nose**
  - Rhinorrhea

- **Oral**
  - Salivation

- **Airways**
  - Bronchoconstriction, bronchorrhea

Seconds to minutes after exposure
Signs and Symptoms

Liquid Exposure
- 10 minutes to 18 hours after exposure

Skin
- Localized sweating, fasciculations

GI
- Diarrhea, nausea, vomiting

Cardiac
- Brady, heart block
Signs and Symptoms

Severe exposure

Previously described vapour and liquid effects plus …

CNS
- LOC, seizures, fasciculations
- Weakness, paralysis

Resp
- Apnea

GI/GU
- Bowel/bladder incontinence

Seconds to minutes (vapour)
Minutes to hours (liquid)
Diagnosis:

*Presence of muscarinic (M) and nicotinic (N) symptoms*

Activity of **BuChE** in plasma (no AChE!) *only informative*
Activity of **Ery-AChE** *more reliable!*
OP metabolites in urine/blood

EMG: when IMS and/or OPIDN is suspect.
Antidotes

Atropine

- 2 mg IV/IM 5-15 min to effect
- **Muscarinic action**
  - smooth muscle
  - glandular epithelium
  - cardiac muscle

Stop Atropin if: mydriasis + tachycardia + dry axilla
Antidotes

**Pralidoxime Chloride**

- 1 g IV over 15-30 min q 1 hr to effect
- **Nicotinic action**
  - skeletal muscle
- **Aging**: Irreversible binding of nerve agent to AChE
  - **Soman**: 2 minutes
  - **VX**: 48 hours

In EU: obidoxim (TOXOGONIN)
Antidotes

Diazepam

- Seizure prophylaxis and treatment
- 10 mg IV at onset of severe symptoms regardless of seizure activity
Carbon monoxide (CO)

Most common poisoning in industry. Odorless, colorless, tasteless, highly reactive gas. **Source:** by incomplete combustion of organic comp.

**Mechanism of action:** binding with high affinity to Hb (250x that O2) → carboxy-Hb COHb → ANOXIA!

**Symptoms:** headache, nausea, pale skin color, dizziness, confusion, muscle weakness (unable to escape), stupor, unconscious

**Therapy:** PSV (O2 or 95%O2+5% CO2) compression chamber (hyperbaric O2, 2 atm)
The patient’s presentation is more reliable than the COHb level (%)

0 – 10  minimal manifestation
10 - 20  moderate dyspnea, headache
30 – 40  dizzy, weakness, nausea, vomiting
40 – 50  confusion, tachycardia, synkope
50 – 60  + Cheyne-Stokes, stupor, convulsions
60 – 70  coma, convulsions, resp-heart failure
70 – 80  DEATH

NO CORRELATION COHb and SYMPTOMS
Carbon dioxide (CO2)

a colorless, odorless, tasteless gas, about one and one-half times as dense as air under ordinary conditions of temperature and pressure. It does not burn, and under normal conditions it is stable, inert and nontoxic, but it can cause death by suffocation if inhaled in large amounts (replacing oxygen in air)

Soluble in water, the resultant weakly acidic aqueous solution - **carbonic acid** $\text{H}_2\text{CO}_3$ →

**metabolic acidosis**
Sources: volcanic outgassing, the combustion of organic matter and respiration processes of living aerobic organisms; produced by various microorganisms from fermentation and cellular respiration.

Symptoms: Headache, shortness of breath, dizziness, drowsiness, ringing in the ears...

Treatment: oxygen
Chlorine

• Description
  – At room temperature, yellow-green gas with a pungent irritating odour.
  – Only slightly soluble in water, but on contact with moisture it forms hypochlorous acid (HClO) and hydrochloric acid (HCl). HClO readily decomposes, forming oxygen free radicals.
  – Not explosive but reacts or forms explosive compounds with other substances (e.g. NH₃, acetylene)

• Routes of exposure
  – Inhalation
  – Skin/Eye contact
Clinical effects

Chlorine

Skin
- Irritation, frostbite

Eyes
- Irritation, ocular burns

Upper airway
- Nasal, pharynx, tracheal inflammation
- Laryngospasm

Lower airway
- Inflammation and loss of pulmonary capillary integrity
  
  Pulmonary edema, hypoxia

May occur minutes to 24 hours after exposure
ED Management

• Decontamination if not done previously

• Resp:
  – Fluid restriction in patients with pulmonary edema/ARDS
  – Beta agonists
  – If intubation needed perform under direct visualization (avoid blind techniques)

• Burns:
  – Treat as thermal
Caustic or corrosive exposure

Acids, amonia, alkali, drain cleaners...

**Skin and eyes:** irrigate 15 min – 3 hr

**Ingestion:** rinse mouth, small amounts milk/water

**NO EMETIC, LAVAGE, ACTIVATED CHARCOAL!!!**
Lead
Sources of lead

- Commonly used in the building industry for roofing and flashing and for soundproofing
- Used in pipes
- When combined with tin, it forms solder, used in electronics and in other applications to make connections between solid metals
- Lead is also used in ammunition

Note: Lead shots have been banned in United States, Canada, Netherlands, Norway and Denmark

- Lead is used in batteries and sinkers in fishing
Sources (contd.)

• **Used in paints**
  Lead is also used in corrosion-resistant paints and has a bright red color

• **Used in ceramics and dishware**
  The leaching of lead from glazed ceramics used to prepare food is a major source of dietary lead

• **In the past, lead salts were used as coloring agents in various foods**
Inhibition of heme synthesis

Heme Oxidase (microsomal)

Protoporphyrin IX* ALA-S

Coproporphyrinogen IX* Copro

Coproporphyrin Copro

ALA- aminolevulinic acid
↑ in plasma and urine
COPRO- coproporphyrinogen
↑ in urine
Protoporphyrin
↑ accumulates in the RBC

Heme

Cytoch-C

Glycine Succinyl-Coa

Ferro-C 4Fe++

ALAS

Bilirubin + Fe

anaemia
General Signs and Symptoms of Lead Toxicity

- Fatigue
- Irritability
- Lethargy
- Paresthesia
- Myalgia
- Abdominal pain
- Tremor
- Headache
- Vomiting
- Weight loss
- Constipation
- Loss of libido

- Motor neuropathy
- Encephalopathy
- Cerebral edema
- Seizures
- Coma
- Severe abdominal cramping
- Epiphyseal lead lines in children (growth arrest)
- Renal failure
- Anaemia!
Early Symptoms of Lead Poisoning

- Fatigue
- Headaches
- Irritability
- Metallic Taste

- Uneasy Stomach
- Poor Appetite
- Weight Loss
- Reproductive Problems
Diagnosis

• Evaluation of clinical symptoms and signs
• CBC, Basophilic stippling RBC
• Serum iron levels, TIBC, ferritin
• ALA- aminolevulinic acid ↑ in plasma and urine
• COPRO- coproporphyrinogen↑ in urine
• Abdominal radiographs (for recent ingestion of lead-containing material)
• Whole blood lead level
• EMG-peripheral neuropathy
Treatment

• Environmental inspection/hazard reduction
• Nutritional supplementation
• Chelation therapy (EDTA, DMSA, DMPS)
Mercury
Mercury

- Occurs in three forms (elemental, inorganic salts, and organic compounds)
- Contamination results from mining, smelting, and industrial discharges. Mercury in water can be converted by bacteria to organic mercury (more toxic) in fish.
- Can also be found in thermometers, dental amalgams, fluorescent light bulbs, disc batteries, electrical switches, folk remedies, chemistry sets and vaccines.
Mercury - Exposure

- **Elemental**
  - liquid at room temperature that volatizes readily
  - rapid distribution in body by vapor, poor in GI tract
- **Inorganic**
  - poorly absorbed in GI tract, but can be caustic
  - dermal exposure has resulted in toxicity
- **Organic**
  - lipid soluble and well absorbed via GI, lungs and skin
  - can cross placenta and into breast milk
Elemental Mercury

- At high concentrations, vapor inhalation produces acute necrotizing bronchitis, pneumonitis, and death.
- Long term exposure affects CNS.
  - Early: insomnia, forgetfulness, anorexia, mild tremor
  - Late: progressive tremor and erethism (red palms, emotional lability, and memory impairment)
  - Salivation, excessive sweating, renal toxicity (proteinuria, or nephrotic syndrome)
- Dental amalgams do not pose a health risk.
- **Orally not toxic!**
Inorganic Mercury

- Gastrointestinal ulceration or perforation and hemorrhage are rapidly produced, followed by circulatory collapse.
- Breakdown of mucosal barriers leads to increased absorption and distribution to kidneys (proximal tubular necrosis and anuria).
- Acrodynia (Pink disease) usually from dermal exposure or vapour (Hg$^0$)
  - maculopapular rash, swollen and painful extremities, peripheral neuropathy, hypertension, and renal tubular dysfunction.
acrodynia
Diagnosis and Treatment

• Dg made by history and physical and lab analysis. Inorganic mercury can be measured in 24 hour urine collection;

• The most important and effective treatment is to identify the source and end the exposure

• Chelating agents (DMSA) may enhance inorganic mercury elimination. Dimercaprol may increase mercury concentration in the brain.
Amanita phalloides = Death cap
Pathophysiology:

The clinical manifestations of an *A phalloides* ingestion are the result of the cyclopeptide toxins, phalloidin and amatoxin. Phalloidin causes gastroenteritis-like effects 6-12 hours after initial ingestion. Phalloidin, a cyclic heptapeptide, interrupts the actin polymerization-depolymerization cycle and impairs cell membrane function. Phalloidin has limited gastrointestinal absorption, and symptoms improve within hrs of supportive care. Amanitins, primarily alpha-amanitin, are responsible for the hepatic, renal, and encephalopathic effects. Amatoxin, an octapeptide, inhibits ribonucleic acid (RNA) polymerase II, therefore interfering with DNA and RNA transcription.
Stage I: Sudden onset of nausea, vomiting, watery diarrhea, and cramping abdominal pain occurs 6-24 hours after ingestion. Pts may become dehydrated and hypotensive. Pts often present during this stage, and, if misdiagnosed, may be discharged erroneously without further care.

Stage II: Clinical improvement occurs with supportive care. Despite the resolution of sy, hepatic and renal damage is ongoing, which is evident by rising lab test.

Stage III: If discharged, pts may return to the hospital 2-6 days later with severe coagulopathy, renal failure, and encephalopathy
Diagnosis:

1. Macroscop. identification of the mushroom (atlas)
2. Microscop. identif. of the spore: food, gastric content, feces
3. Wieland lignine test: HCL + A.ph. on newspaper = green spot (not specific!)
4. Amanitine in blood - RIA method (rarely available)

Reality: 1. latent period > 6 hr
2. potential confusion (champignon)
3. ↑ LFT

if 2 positive ⇒ Amanita intox.
Therapy:

1. Early gastric lavage + charcoal + WBI
2. Silibinine (LEGALON – Sil. amp.) 20mg/kg/day
3. PNC megadose if silibinine not available (!)
4. Vitamins K, B, C
5. Fresh frozen plasma
6. FD, HD, HP, PF not useful – amanitine in urine as early as 1 hr after consuming
7. TX liver: hepatic failure... F V. < 10-20%.

Method of choice
Carduus marianus.
Legalon.
Mariendistel.
Milk thistle.
Silybin.
Silybum marianum.
Silybum.
Silychristin.
Silydianin.
Silymarin
Primary elimination methods:

1. Induced vomiting (mechanically, NaCl, Sir. of ipecac.)
2. Gastric lavage
3. Multiple-Dose Activated Charcoal, MDAC
4. Whole Bovel Irrigation, WBI
5. cathartics
Contraindications of GL

Unless intubated, the loss of airway protective reflexes: in a patient with a depressed state of consciousness.

Ingestion of a corrosive subst.: strong acid or alkali.

Ingestion of a hydrocarbon with high aspiration potential.

Patients who are at risk of hemorrhage or GI perforation due to pathology, recent surgery, or other medical condition, that could be further compromised by the use of GL.
Complications

Aspiration pneumonia.

Laryngospasm.

Hypoxia and hypercapnia.

Mechanical injury to the throat, esophagus, and stomach.

Fluid and electrolyte imbalance.

Combative pts may be at greater risk of complications.
Gastric lavage should not be employed routinely in the management of poisoned patients. In experimental studies, the amount of marker removed by gastric lavage was highly variable and diminished with time. There is no certain evidence that its use improves clinical outcome and it may cause significant morbidity. Gastric lavage should not be considered unless a patient has ingested a potentially life-threatening amount of a poison and the procedure can be undertaken within 60 minutes of ingestion. Even then, clinical benefit has not been confirmed in controlled studies.
Substitute for GL

Activated Charcoal - powder (Carbosorb plv)

Dosage:

1. bolus 1g/kg b.w.
2. continue 20-50 g/2-6 hr (MDAC)

End of therapy:
good health status, ↓blood conc.
Effect of activated charcoal:

Surface 1 g AU 1 000 – 3 200 m²

Adsorption of toxins mol. w. 100-1 000 dalton

1. Adsorption of toxin
2. Interruption of E-H circulation
3. Enterocapillary exsorption (*like* HP)
ACTIVATED CHARCOAL

Compounds known not to be effectively bound

• elemental metals (Pb, Fe)
• boric acid
• fluorides
• cyanide
• petroleum distillates
• other organic solvents
• ethanol • methanol • ethylene glycol
• acids and alkali
SNAKE BITE

North viper, European common adder (*Vipera berus*) – only species of venomous snake in SR and EU

**Occurrence:** 800 M above sea level; sunny, warm, damp

**Length:** male 60 cm, female 80 cm

**Color:** ash–gray, brown-gray, zig-zag stripe

„Dry bite 40%“

*(Taipan in Central Australia 1 bite kill 25,000 mice)*
Vipera berus venom

complex mixture of proteases effects: cytotoxic and haemotoxic. The cytotoxic element disrupts vascular endothelial linings and this manifests itself clinically as progressive oedema spreading proximally from the site of bite. Progressive cutaneous signs of soft tissue haemorrhage are common. Haemotoxicity is rarer but coagulation disorders have been documented.
Vipera berus venom

complex mixture of proteases effects: cytotoxic and haemotoxlic. The cytotoxic element disrupts vascular endothelial linings and this manifests itself clinically as progressive oedema spreading proximally from the site of bite. Progressive cutaneous signs of soft tissue haemorrhage are common.
The common European viper venom does not provide substantial neurotoxic effect !!!
**Symptoms:**
2 symmetric bleeding wounds, pain, swelling...

**Therapy:**

NO ! Excision  
damage of vessels + nerves

   Sucking  
infection of the wound

   Stifling  
↑ absorption

   Burning/Icing  
↑ local damage

YES !  
Limb immobilization

Transport to the hospital

**ANTI - SERUM**

**INDICATION:**

shock

serious digestive/neurol. smpt.

coagulation disorder

extreme local sy,ptom
Snake bite general symptoms:

two puncture wounds
swelling and redness around the wounds
pain at the bite site
difficulty breathing
vomiting and nausea
blurred vision
sweating and salivating
numbness in the face and limbs
First aid myths:

Do not use a tourniquet.
Do not cut into the snake bite.
Do not use a cold compress on the bite.
Do not give the person any medications unless directed by a doctor.
Do not raise the area of the bite above the victim’s heart.
Do not attempt to suck the venom out by mouth.
Do not use a pump suction device.
**Indications for antivenom** (WHO, 2010)

Antivenom treatment is recommended if and when a patient with proven or suspected snake-bite develops one or more of the following signs:

**Haemostatic abnormalities:** Spontaneous systemic bleeding (clinical), coagulopathy (20WBCT or other laboratory tests such as prothrombin time) or thrombocytopenia (<100 x 109/litre or 100 000/cu mm) (laboratory).

**Neurotoxic signs:** ptosis, external ophthalmoplegia, paralysis etc (clinical).

**Cardiovascular abnormalities:** hypotension, shock, cardiac arrhythmia (clinical), abnormal ECG.

**Acute kidney injury (renal failure):** oliguria/anuria (clinical), rising blood creatinine/urea (laboratory).

**Haemoglobin-/myoglobin-uria:** dark brown urine (clinical), urine dipsticks, other evidence of intravascular haemolysis or generalised rhabdomyolysis (muscle aches and pains, hyperkalaemia) (clinical, laboratory).
The drug of choice/anti-serum, is antivenom of *Vipera berus* venom produced in BIOMED® Sera and Vaccines Manufacturing Company in Warsaw. The product is derived from the serum of horses immunized with the venom of the common European viper. The specific equine IgG binds and neutralizes toxic activities of venom by specific bond: antibody (antivenom) – antigen (viper venom).
Xenogen protein present in the product, the **anaphylactic reactions**, including shock may appear after antivenom administration.

The following symptoms may be present: fever, itching, rash, dry cough, nausea, vomiting, colic, diarrhoea, tachycardia. In some patients the life-threatening symptoms such as bronchial spasm, hypotension, angioedema may occur.
Pressure immobilisation first aid method for snakebite used in Australia and Papua New Guinea
Remember!

All substances are poisons... (PARACELSSUS)

Benzodiazepines: HD, FD not effective (↑blood protein binding)

Extasy: cooling!(ice, DANTROLENE)

Organic solvents: no GL no charcoal

Alcohol: no GL no charcoal

Mercury elemental p.o. not toxic

Death cap: silymarin antidotum

Gastric lavage: limited indications

Snakes: strong limited anti serum indications

European adder: extremely rare serious intoxications

Do not use tourniquet
Thank you