Poisoning

Mohammed Almazyad, MBBS, FRCPC, ABP
Assistant Professor of Pediatrics –KSU
PICU Consultant –KKUH

Objectives:

- Epidemiology
- Approach to the poisoned child
  - Hx
  - Physical exam
  - Lab/Diagnostic tests
- Toxidromes
- Principles of management

Color coding

★ **Pink** = Doctor's Notes

★ **Green** = Illustrated textbooks

★ This topic is not mentioned in Kaplan

Best of luck.. Sarah AlMutawa

Epidemiology

very common

- The number 1 cause of injury death in the United States.
  - Mostly unintentional.
  - CO Carbon monoxide and analgesics were the leading causes of poison-related fatalities in young children

We have 2 spikes in 2 age groups:

- 50% occur in children younger than 6 yr old.
- Poisoning exposures in children 6-12 yr old are much less common(2%)
- A second peak in pediatric exposures occurs in adolescence (intentional)
- Children younger than 6 yr account for <2% of all poisoning fatalities

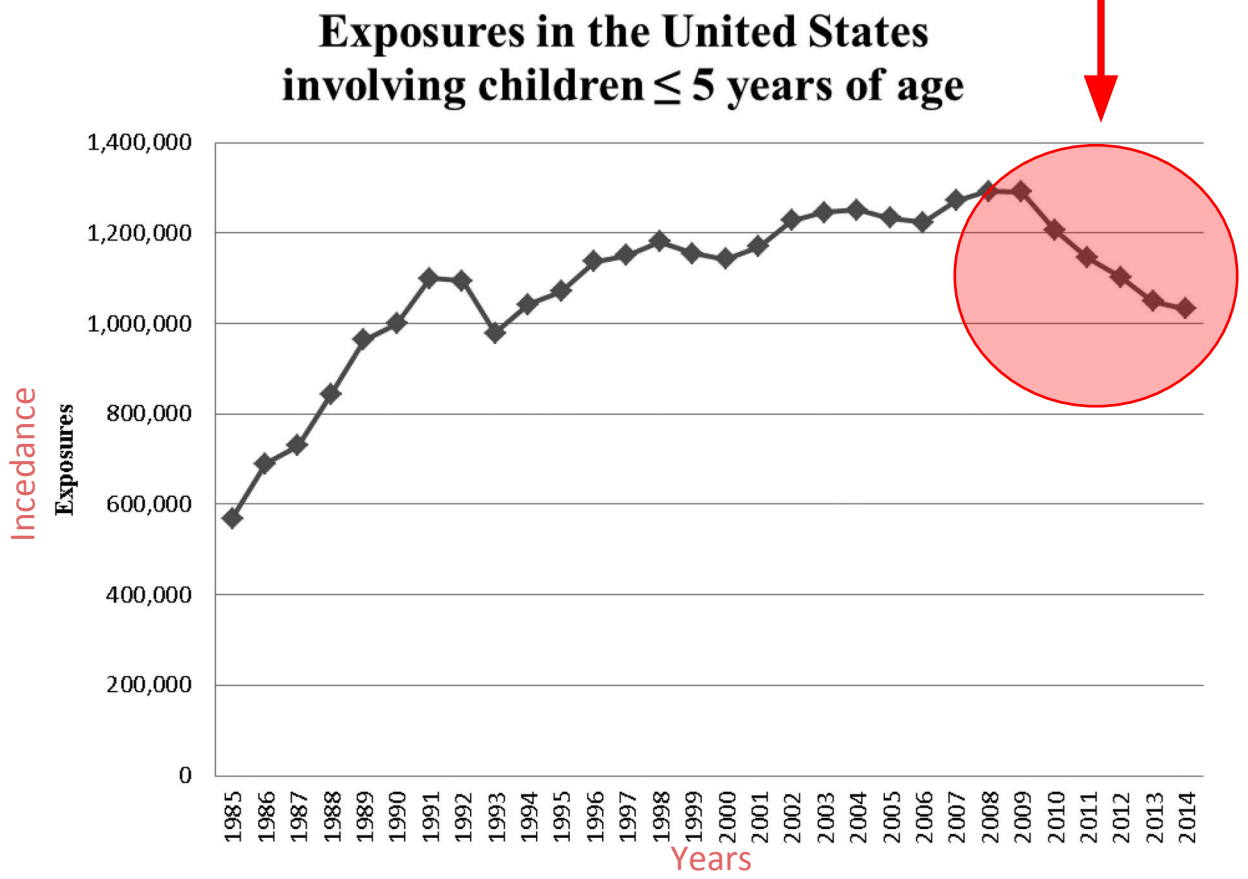
Why is it common below 6 yrs then becomes less between 6-12 yrs then goes back high above 12 yrs?

- **Below 6 yrs**: the child doesn't understand the difference between dangerous and non dangerous, so they like to try which leads to unintentional poisoning
- **Above 12 yrs:** suicidal attempts becomes more prevalence which leads to intentional poisoning **(This age group are higher fatality)** All older children who have attempted to harm themselves must be assessed for risk of a repeated attempt: The risk of recurrence is increased by a number of factors including: thoughts of self-harm or suicide, a lack of regret, evidence of planning, e.g. leaving a note, and a lack of protective social factors and those who harm themselves as a result of experimentation with illicit drugs or alcohol.

Epidemiology - Where/how/what

? Related to newly designed safety packages

- 90% of toxic exposures in children occur in the home.
- Most involve only a single substance
  Single injury not polytrauma
- Ingestion most common route
- 50% of cases involve are nondrug
- Household cleaning products and cosmetics represent the majority of calls to poison centers

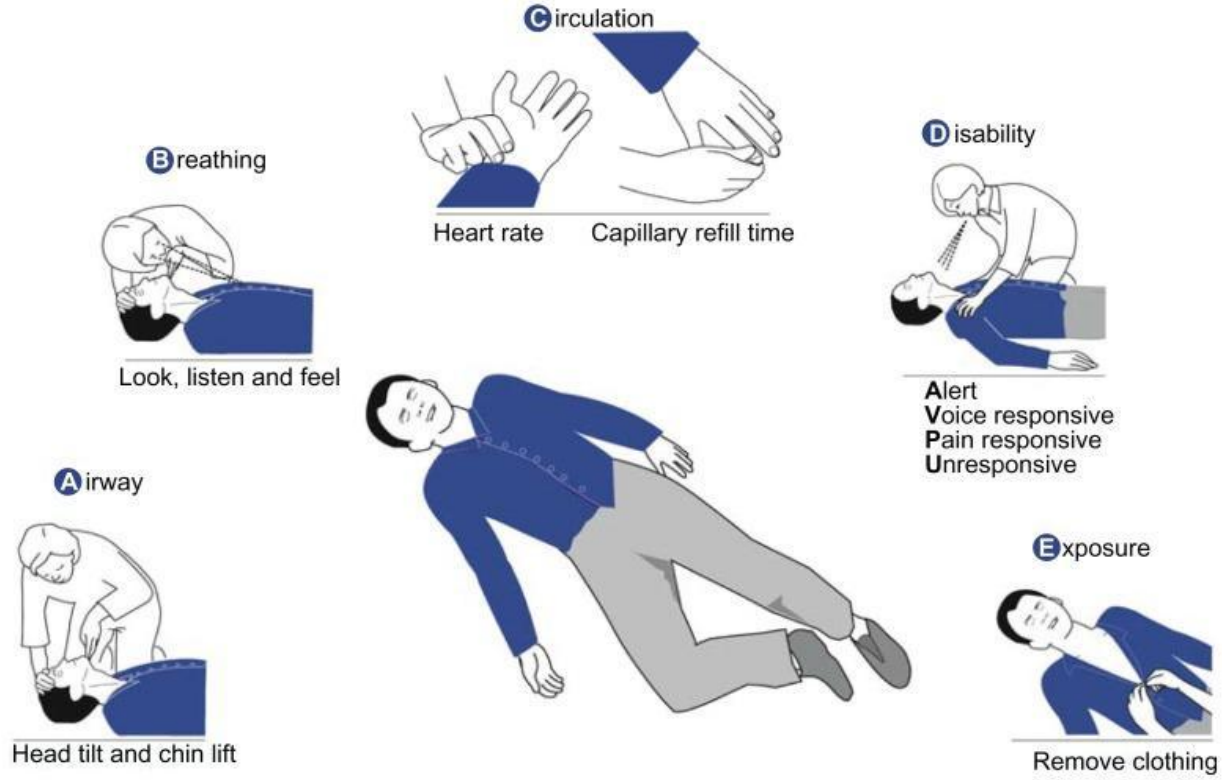


The incidence was increasing until the mid 2000 it starts to decrese and we think this is related to the new regulations in the state in regard of packaging and design for example:

- the introduction of child-resistant containers and use of blister packs for medicines
- reduction in the number of tablets per pack in analgesics bought over the counter.
- Tide soap balls they're trying to make it not attractive to children bc they look like candy

Approach

- Should be no different than that in any other sick child.
- Stabilization and ABCDEs
- Targeted history and physical examination



1. The 1st thing to do outside the hospital is to make sure that the **environment is safe!**
2. clean the baby to prevent continuous exposure
3. then ABCDE
4. Targeted History and examination

Approach – History

- Very important
- Some features may suggest poisoning
  - Acute onset
  - No prodrome
  - Sudden change in LOC
  - Multiorgan failure
- Description of the exposure:
  - When, how much , where , why
  - What (household product vs medical)
  - List of meds in house
  - Did the family brought it the product with them?
  - immediate- versus extended-release preparation

Some important Qs:

- ➔ Was the child witnessed or not?
- ➔ The last time the child was seen active?
- ➔ Does he have any medical conditions?
- ➔ Are there any accociated symptoms? like diarrhea fever tearing
- ➔ History of previous ingestion?

Developmental hx is very imp bc child with developmental delay are at higher risk of accidental injury or even intentional abuse

Please be alert about the fact that the injury might be caused by the caregiver themselves or maybe the older siblings bc they're jealous in a condition we call: Munchausen By Proxy (MSBP)

How to identify it = what are the red flags in history?

- Signs of inconsistency in the story rise a suspicion of abuse
- pattern of injury (like the buttock in burn, bc if someone immerge 6 m old baby the legs will be up so buttock will be affected)

It's imp to ask *why* in teenagers, to identify if it's accidental or suicidal

What are the available medications at home that the child has access to? or sometimes u can find open meds at scene

In extended release, the peak of symptoms will be late and the time of observation will be longer

In the case of an unknown exposure, clarifying **where** the child was found (e.g., garage, kitchen, laundry room, bathroom, backyard, workplace) can help to generate a list of potential toxins.

Approach – History

- Details of the symptoms
  - When did the symptom start in relation to time of ingestion
  - Progression
- Past Medical History:
  - Underlying diseases can make a child susceptible to the effects of a toxin.
  - Current medication list (Drug-Drug interaction)
  - Psychiatric illness (more prone to substance abuse, misuse, intentional ingestions, and polypharmacy complications)
  - A developmental history (a report of a 6 mo old picking up a large container of laundry detergent and drinking it should raise a red flag)
- Social History

Approach –Physical Exam

- Targeted aiming to identify potential toxin and assess the severity.
- Findings might suggest a toxidrome and help to build a DDx
- Initial effort should be directed to ABCs
- Key features of the physical exam:
  - V/S Vital signs
  - LOC/GCS Level of consciousness / Glascow Coma Scale
  - Pupils
  - Nystagmus
  - Skin Inspection if there is any toxins in the skin
  - Bowl sounds
  - Odor
  - Respiration: Apnea, Hypopnea, Tachypnea

Start with your assessment triangle:

1. How the patient look?
2. Is the patient breathing?
3. Is the patient conscious?

This defines if we should proceed with CPR or we can take our time

Overview of some physical findings

The following slides are busy of lots of tables
You don't need to know them all!
The most imp and significant ones are highlighted

(I think the dr means underlined, So I highlighted them in blue)

ODOR	
Bitter almonds	**Cyanide**
Acetone	Isopropyl alcohol, methanol, paraldehyde, salicylates
Alcohol	Ethanol
Wintergreen	Methyl salicylate
Garlic	**Arsenic**, thallium, **organophosphates**, selenium

Organophosphate is a common poison so if you smell garlic you may think of it

OCULAR SIGNS	
Miosis Constricted pupils	**Opioids** (except propoxyphene, meperidine, and pentazocine), **organophosphates** and other **cholinergics**, clonidine, phenothiazines, sedative–hypnotics, olanzapine
Mydriasis Dilated pupils	**Anticholinergics** (e.g., antihistamines, TCAs, atropine), **sympathomimetics** (cocaine, amphetamines, PCP) postanoxic encephalopathy, opiate withdrawal
Nystagmus	**Anticonvulsants**, sedative–hypnotics, alcohols, PCP, **ketamine**, dextromethorphan
Lacrimation	**Organophosphates**, irritant gas or vapors
Retinal hyperemia	Methanol

CUTANEOUS SIGNS	
Diaphoresis	**Cholinergics** (organophosphates), sympathomimetics, withdrawal syndromes
Alopecia	Thallium, arsenic
Erythema	Boric acid, elemental mercury, cyanide, carbon monoxide, disulfiram, scombroid, anticholinergics, vancomycin
Cyanosis (unresponsive to oxygen)	**Methemoglobinemia** (e.g., benzocaine, dapsone, nitrites, phenazopyridine), **amiodarone**, silver Antiarrhythmic medication

ORAL SIGNS	
Salivation	Organophosphates, salicylates, corrosives, ketamine, PCP, strychnine
Oral burns	**Corrosives**, oxalate-containing plants
Gum lines	Lead, mercury, arsenic, bismuth
GASTROINTESTINAL SIGNS	
Diarrhea	Antimicrobials, arsenic, iron, boric acid, cholinergics, colchicine, **opioid withdrawal**
Hematemesis	Arsenic, iron, caustics, NSAIDs, salicylates

CARDIAC SIGNS Imp table to know	
Tachycardia	**Sympathomimetics**, **anticholinergics**, antidepressants, antipsychotics, methylxanthines (theophylline, caffeine), salicylates, cellular asphyxiants (cyanide, carbon monoxide, hydrogen sulfide), **withdrawal** (ethanol, sedatives, clonidine, opioids), serotonin syndrome, neuroleptic malignant syndrome
Bradycardia	**β Blockers**, **calcium channel blockers**, **digoxin**, **clonidine**, organophosphates, opioids, sedative–hypnotics Most Antiarrhythmic medications cause bradycardia and hypotension
Hypertension	**Sympathomimetics**, **anticholinergics**, monoamine oxidase inhibitors, serotonin syndrome, neuroleptic malignant syndrome, clonidine withdrawal
Hypotension	**β Blockers**, **calcium channel blockers**, cyclic antidepressants, iron, antipsychotics, barbiturates, clonidine, opioids, arsenic, amatoxin mushrooms, cellular asphyxiants (cyanide, carbon monoxide, hydrogen sulfide), snake envenomation

RESPIRATORY SIGNS	
Depressed respirations	**Opioids**, sedative-hypnotics, alcohol, clonidine, barbiturates
Tachypnea	**Salicylates**, sympathomimetics, caffeine, metabolic acidosis, carbon monoxide, hydrocarbon aspiration
CENTRAL NERVOUS SYSTEM SIGNS	
Ataxia	Alcohols, anticonvulsants, sedative–hypnotics, lithium, dextromethorphan, carbon monoxide, inhalants
Coma	Opioids, sedative–hypnotics, anticonvulsants, antidepressants, antipsychotics, ethanol, anticholinergics, clonidine, GHB, alcohols, salicylates, barbiturates
Seizures	Sympathomimetics, anticholinergics, antidepressants (especially TCAs, bupropion, venlafaxine), cholinergics (organophosphates), isoniazid, camphor, lindane, salicylates, lead, nicotine, tramadol, water hemlock, withdrawal
Delirium/psychosis	Sympathomimetics, anticholinergics, LSD, PCP, hallucinogens, lithium, dextromethorphan, steroids, withdrawal
Peripheral neuropathy	Lead, arsenic, mercury, organophosphates

I added this table summarizing all physical findings that was **(underlined)** in the past slides, the ones that was mentioned by the dr are written in **(RED)**

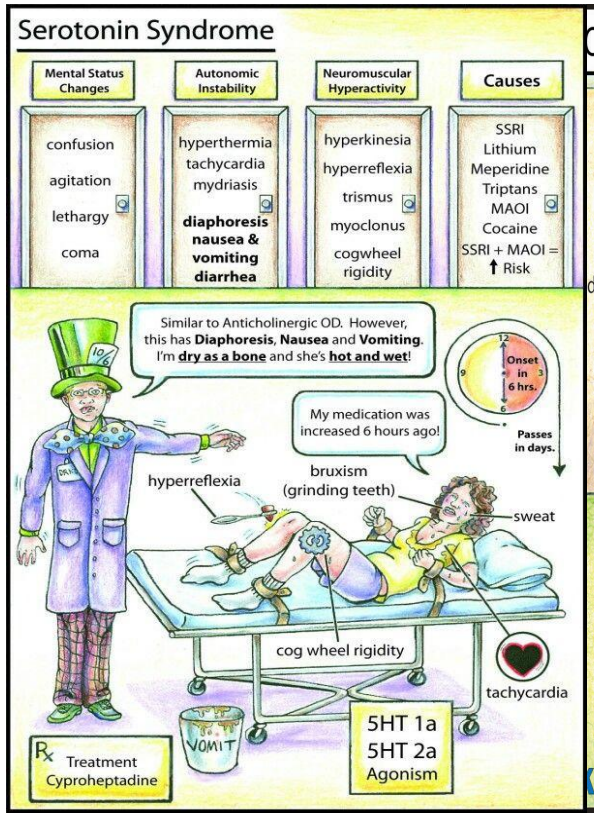
ODOR	Bitter almonds	Cyanide
	Garlic	Arsenic, **organophosphates**
OCULAR SIGNS	Miosis	**Opioids , organophosphates , cholinergics**
	Mydriasis	**Anticholinergics , sympathomimetics**
	Nystagmus	Anticonvulsants , ketamine
	Lacrimation	Organophosphates
CUTANEOUS SIGNS	Diaphoresis	**Cholinergics** (Organophosphates)
	Cyanosis	**Methemoglobinemia , amiodarone**
ORAL SIGNS	oral burns	Corrosive
GASTROINTESTINAL	Diarrhea	Opioids withdrawal
CARDIAC	Tachycardia	**Sympathomimetics, anticholinergics**, withdrawal (ethanol, sedatives, clonidine, opioids)
	Hypertension	**Sympathomimetics, anticholinergics**
	Bradycardia	**β Blockers, calcium channel blockers, digoxin**, clonidine
	Hypotension	**β Blockers, calcium channel blockers**
RESPIRATORY	Depressed respirations	Opioids
	Tachypnea	Salicylates

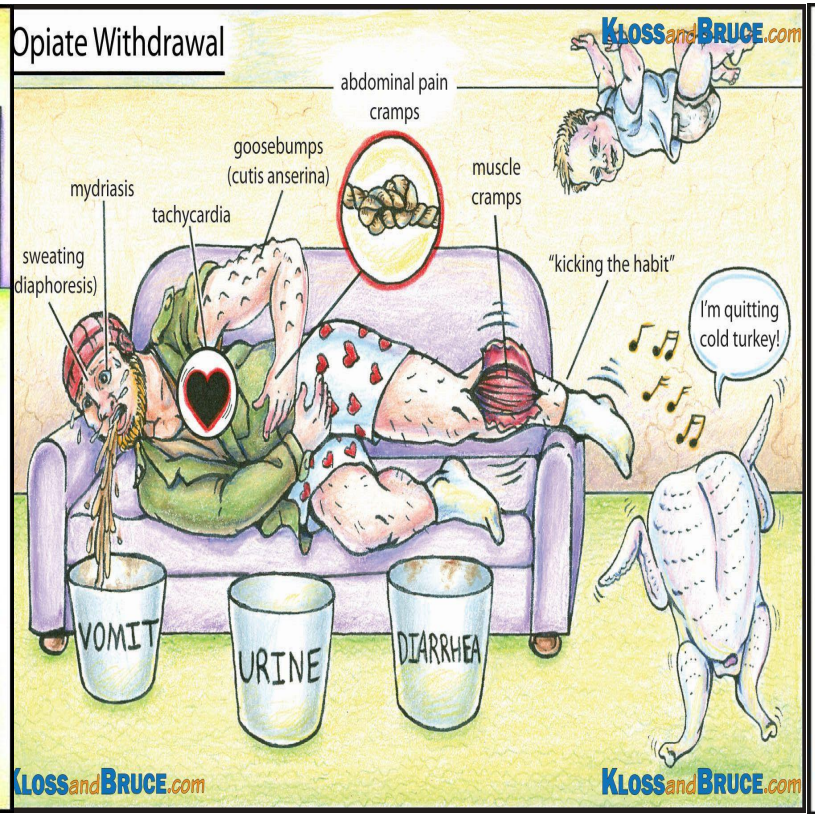
Toxidromes

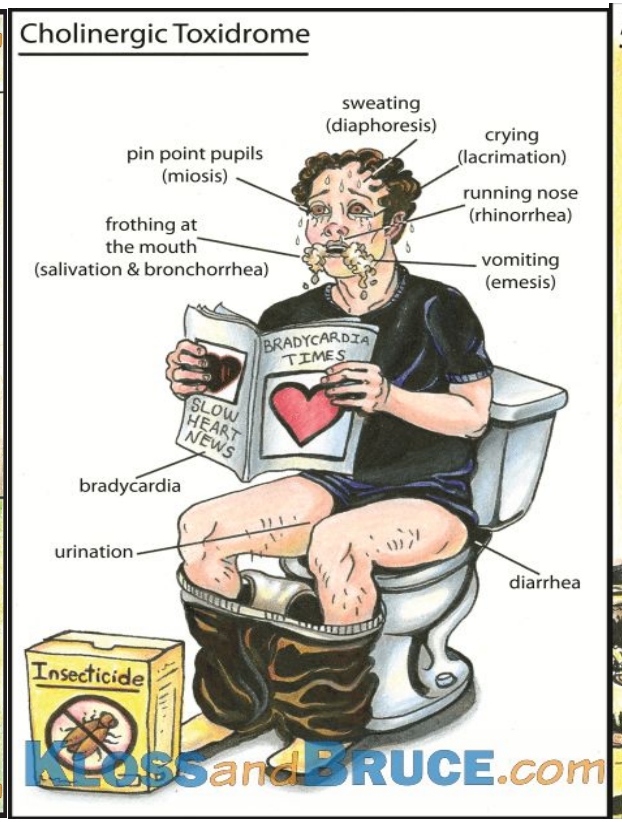
- Definition:

  A group of signs and symptoms constituting the basis for a diagnosis of poisoning.

this is book, but in real life most of the time the picture is not clear and we don't know what is the poison!







Sympathomimetic

Activation of sympathetic system, imagine that you are an a dark room and scared, so your pupil will be dilated, your heart rate will be high, you will be agitated, alert and sweating.

TOXIDROME	SIGNS						POSSIBLE TOXINS
	VITAL SIGNS	MENTAL STATUS	PUPILS	SKIN	BOWEL SOUNDS	OTHER	
Sympathomimetic	Hypertension, tachycardia, hyperthermia	Agitation, psychosis, delirium, violence	Dilated	Diaphoretic	Normal to increased	PCP = Phencyclidine, also known as angel dust	Amphetamines, cocaine, PCP, bath salts (cathinones), ADHD medication

Amphetamines, cocaine, PCP are all street drugs, what is most imp the ADHD medications like (Retanil)

Anticholinergic

Sympathomimetics toxidrome are similar to Anti-Cholenergic, so how to differentiate between them?

- Sympathomimetics = Sweating
- Anti-Cholenirgic = Only hot, there is no sweating (= dry)

By the way, it's very hard to differentiate between them in real life!

TOXIDROME	SIGNS						POSSIBLE TOXINS
	VITAL SIGNS	MENTAL STATUS	PUPILS	SKIN	BOWEL SOUNDS	OTHER	
Anticholinergic	Hypertension, tachycardia, hyperthermia	Agitated, delirium, coma, seizures	Dilated	Dry, hot	Diminished	Ileus urinary retention	Antihistamines, tricyclic antidepressants, atropine, jimson weed

Commonly used!

Cholinergic

Totally the opposite of anticholinergics

Although acute exposure to organophosphate and carbamate pesticides results in well-known acute syndromes, there is growing evidence that chronic exposure to these agents in early life can have adverse effects on neurodevelopment and behaviour. In addition, there is evidence associating some pesticides with an increased incidence of leukaemia and brain tumours.

TOXIDROME	SIGNS						POSSIBLE TOXINS
	VITAL SIGNS	MENTAL STATUS	PUPILS	SKIN	BOWEL SOUNDS	OTHER	
Cholinergic	Bradycardia BP and temp typically normal	Confusion, coma, fasciculations	Small	Diaphoretic	Hyperactive	Diarrhea, urination, bronchorrhea, bronchospasm, emesis, lacrimation, salivation	Organophosphates (insecticides, nerve agents), carbamates (physostigmine, neostigmine, pyridostigmine)

Organo phosphorus can be found in farms, pesticide, easily accessed here

Neostigmine used in Myasthenia gravis

Opioids

Everything is down, hypopnea bradycardia slow sleepy no bowel sounds might go to comma and develop hypotension and hypothermia

TOXIDROME	SIGNS						POSSIBLE TOXINS
	VITAL SIGNS	MENTAL STATUS	PUPILS	SKIN	BOWEL SOUNDS	OTHER	
Opioids	Respiratory depression bradycardia, hypotension, hypothermia	Depression, coma, euphoria	Pinpoint	Normal	Normal to decreased		Methadone, buprenorphine, morphine, oxycodone, heroin, (street drug) etc.

Codeine
Fentanyl (synthetic)

Opioids Withdrawal

Totally the opposite of opioids toxidrome
seizure, agitated, diarrhea, hypertensive

TOXIDROME	SIGNS						POSSIBLE TOXINS
	VITAL SIGNS	MENTAL STATUS	PUPILS	SKIN	BOWEL SOUNDS	OTHER	
Withdrawal (opioid)	Tachycardia	Restlessness, anxiety	Dilated	diaphoretic	Hyperactive	Nausea, vomiting, diarrhea	Lack of access to opioids or excessive use of naloxone

What is Withdrawal? symptoms appear after stopping the medication

What is tolerance? lets say you are using morphine chronically, after 5 or 7 days your body recognizes actually that something inhibiting the receptors, so upregulate the receptors (more expression of that protein on the surface) so the body is trying to fight this medication, so this result in needing a higher dose to reach the same effect for example

- if you have 100 receptors, you need 70 to be occupied by the drug in order to have the effect
- if the body upregulate them to 150, if only 70 of them are occupied you will not have the effect of the drug, you need more

Serotonin Syndrome

Happens with SSRI, very hard to differentiate from other toxidromes, so we usually we treat them empirically

TOXIDROME	SIGNS						POSSIBLE TOXINS
	VITAL SIGNS	MENTAL STATUS	PUPILS	SKIN	BOWEL SOUNDS	OTHER	
Serotonin syndrome	Hyperthermia, tachycardia, hypertension or hypotension (autonomic instability)	Agitation, confusion, coma	Dilated	Diaphoretic	Increased	Neuromuscular hyperexcitability: clonus, hyperreflexia (lower extremities > upper extremities)	SSRIs, lithium, MAOIs, linezolid, tramadol, meperidine, dextromethorphan

I added this table summarizing some toxidromes that was mentioned in the past few slides

Toxidrome	Toxins	Signs				
		Pupil	Skin	Mental status	Vitals (HR, RR, Tem)	Bowel sounds
Opioids everything down	illegal: Heroin Medical: Morphine, oxycodone...	Constricted	Normal	Coma	Low	Low
Opioids withdrawal like sympath...	Lack of opiods	Dilated	Sweating	Agitated	High	High
Serotonin syndrome almost like sympath...	SSRIs	Dilated	Sweating	Agitated	High or Low	High
Sympathomimetics everything up	illegal: Amphetamines, cocaine, PCP Medical: for ADHD	Dilated	Sweating	Agitated	High	High
Anticholinergics remember (dry)	Antihistamines, tricyclic antidepressants, atropine	Dilated	Dry	Agitated	High	Low
Cholinergic opposite anti chole	Organophosphate Neostagmine	Constricted	Sweating	Coma	only RR low	High

Case scenario from Illustrated:

Jemima, a 14-year-old girl, is brought to the emergency department in the morning by her mother as she has been vomiting and complaining of severe abdominal pain. On examination she has a generally tender abdomen. Blood tests reveal an extremely high alanine transaminase concentration, well above normal for her age. Her clotting is also deranged with a prothrombin time of 17 seconds. An initial diagnosis of hepatitis is considered but on discussion with the consultant the lack of jaundice is considered atypical. On further direct questioning, Jemima admits to having taken 22 para- cetamol tablets (500 mg) the previous afternoon following an altercation with another girl at school. Jemima is commenced on N-acetylcysteine and makes a full recovery.
This case highlights the need to consider a toxicological cause when the history, examination findings, and investigation results do not fit together.

Lead poisoning is one of the most important chronic environmental toxins affecting children worldwide. Although it is now uncommon in the UK and other developed countries, in some developing countries contamination of water supplies and the home envi- ronment by mining processes and factories remains a significant problem.

The symptoms of chronic lead exposure are nonspecific but include:
- behavioural changes
- hyperactivity or decreased activity
- developmental delay or loss of developmental milestones
- chronic lead nephropathy.

More significant exposure may result in:
- abdominal pain, vomiting, constipation
- headache and ataxia
- lethargy, seizures, and coma.

The most important treatment is:
- to prevent further exposure to lead

Lab/Diagnostic Testing

- Tox-Screen Details in next slides
- Drug Levels Details in next slides
- U&E
- Blood Gas
- LFTs (Acetaminophen)
- renal function
- Serum Osmolality (Alcohol)
- CK (prolonged down time)
- ECG (Dig, Amiodaron, SSRI)

ECG is very imp, because certain drugs causes ECG changes like in beta blockers look for bradycardia and heart block

KUB

A kidney, ureter, and bladder X-ray study

you might see the medications if they are radio opaque

RADIOPAQUE SUBSTANCE ON KUB (MNEMONIC = CHIPPED)
•**C** hloral hydrate, calcium carbonate •**H** eavy metals (lead, zinc, barium, arsenic, lithium, bismuth) •**I** ron •**P** henothiazines •**P** lay-Doh, potassium chloride •**E** nteric-coated pills •**D** ental amalgam, drug packets

Tox-Screen

- Varey from lab to lab :
  - What it tests for?
  - Cutoff for +
  - **False + /Fales –**
  - Eg **Synthetic opioid are not detected** with urine tox-screen
- Helpful in patients with altered mental status of unknown etiology, persistent, unexplained tachycardia, acute myocardial ischemia or stroke at a young age

What is Tox screen?

- It is screening for the common abused drugs, but the issue is that each lab and hospital has different list of drugs, but most of them test for benzo, morphine, amphetamine, PCP and not necessarily includes the others

Unfortunately it is not that helpful, why?

- As we mentioned it covers only common drugs, so if you send tox screen and it comes -ve this doesn't roll out poisoning
- It is usually QuaLitative not QuaNtitative just to know if the drug is there or not there

Drug levels

- Tox screen is not that helpful but what's imp is to do is **suspected drug level,** if the pt has certain drugs at home
- **Imp drugs that can be measured:** alcohol, paracetamol, salicylic acid, most anti-epileptic like phenytoin
- **Why it is imo to know the drug level?** to know if it reaches to toxicity or no and some drugs like paracetamol there is a antidote giving depending on which the drug level is

- For some drugs quantitative blood concentrations are integral to confirming the diagnosis and formulating a treatment plan.
  - SAS, Acetaminophen, dig, iron and methanol.

- For most exposures, quantitative measurement is not readily available and is not likely to alter management.

The drug level itself doesn't indicate poisoning! it should be integrated with history

- All intoxicant levels must be interpreted in conjunction with the history.
  - Chronic vs acute use
  - Time of ingestion
  - Co-ongestion

Acetaminophen Level

- ➔ usually intentionally ingestion by the patient
- ➔ very nasty and dangerous medication and I'm insisting on that because mothers nowadays are giving it to their children like a candy while they should be cautious. It has multiple names (adon, panadol) so bc of the different names you might find mothers give their children 3 medication to lower their child fever then he becomes overdosed! they don't know that they are all paracetamol derivatives

- Very helpful.

- Acetaminophen is a widely available medication and a commonly detected co-ingestant with the potential for severe toxicity. it is the most com abused drug for suicide because its commonly available and it does kill, the most common cause of fatality in poisoning attempt suicide and the most com cause of pediatrics liver transplant

- There is an effective antidote to acetaminophen poisoning that is time-dependent. we have an effective antidote N acetyl Cysteine = NAC , you need to give it in certain time and dose depemding on time of ingestion

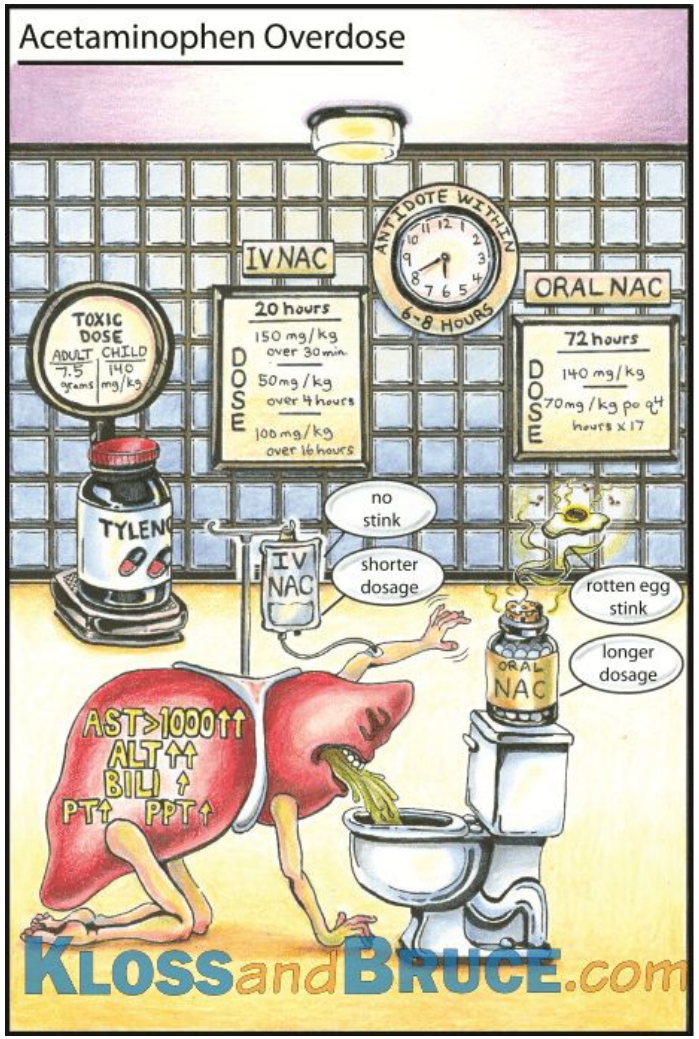
- Patients might initially be asymptomatic and might not report acetaminophen as a coingestant, an acetaminophen level should be checked in all patients who present after an intentional exposure or ingestion usually are asymptomatic, if not treated it progresse , GI upset vomiting , signs of liver failure, decreased LOC, coagulopathy, upper and lower GI bleeding

Acetaminophen

- The most common cause of acute liver failure in USA the most com cause of liver transplant in teenager
- **The single acute toxic dose of acetaminophen is generally considered to be >200 mg/kg in children and >7.5-10 g in adolescents and adults.**

★ One pill of paracetamol contains 500 mg = 0.5 g
★ How many bills you take if you have headache? 2 pills = 1 gram
★ so you need 20 pills to reach toxic dose
:) By the way, there is no evidence that 1 g is more effective than 500 mg !

There is two formulation of paracetamol in the market :
1- High concentration: 1-2 ml for small babies
2- Not concentrated = 10 ml
sometimes family confuses the two forms and uses the highly concentrated with the regular dose (almost x10 the right dose)
Inform your family about this **:(**

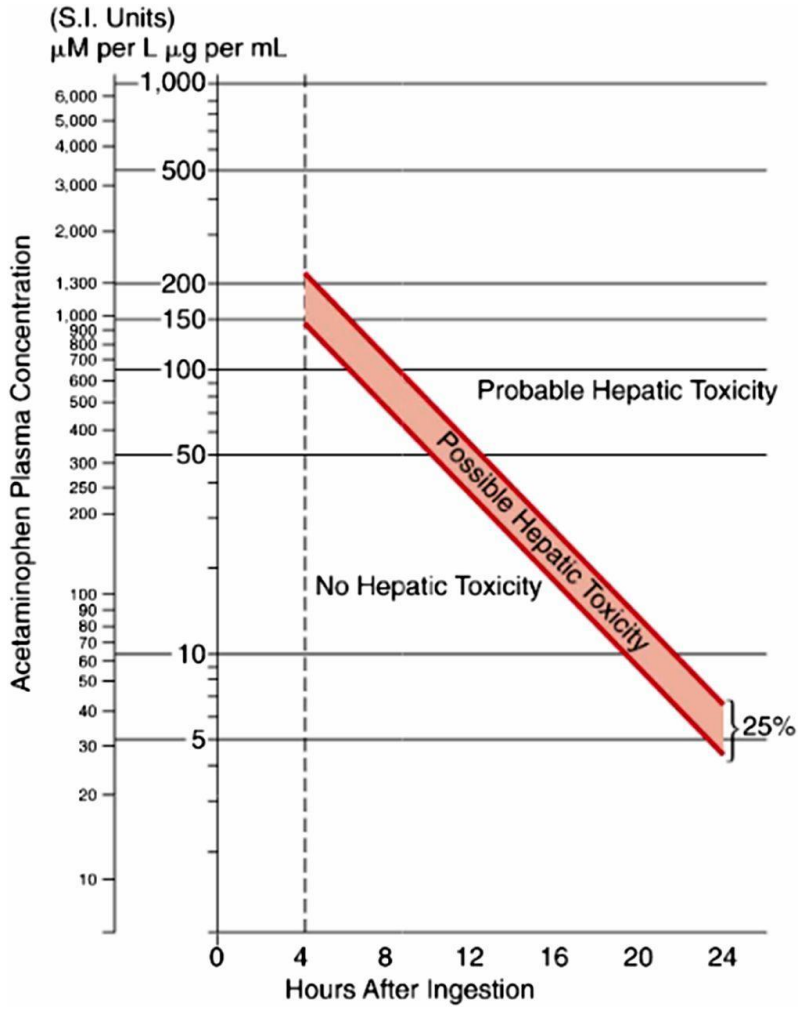


Classical signs and symptoms

STAGE	T after Ingestion	CHARACTERISTICS
I asymptomatic or mild symptoms	0.5–24 hr	Anorexia, nausea, vomiting, malaise, pallor, diaphoresis
II usually takes 2-3 days fulfilled the symptoms?	24–48 hr	Resolution of earlier symptoms; right upper quadrant abdominal pain and tenderness; elevated bilirubin, prothrombin time, hepatic enzymes; oliguria
III labs show high ALT AST INR	72–96 hr	Peak liver fxn abnormalities; anorexia, nausea, vomiting, and malaise may reappear
IV	4 days–2 wk	Resolution of hepatic dysfunction or complete liver failure

Acetaminophen overdose Rx

- After ABCs
- Hx
  - Time
  - Quantity
  - Regular vs extended release
  - Intention
- LFTs, Lytes, renal function
- Obtain a level 4 hours after ingestion
- Use Rumack-Matthew nomogram



- only valid after 4 hours of ingestion, if you take level after 2 hrs for example this is meaningless, why? you have to wait until 4 hours to reach the dose
- Interpretation:
- if it was above the line = toxic dose = give antidote
- below the line = not toxic = dont give antidote
- So you measure it after 4 hs then you measure it again after 8 Hs, then 16 Hs … hours until 24-48 hours

Principles of Management

Details in next slides

Young children who have been exposed to agents of low toxicity and are asymptomatic can typically be discharged with advice to return if symptoms develop. The circumstances surrounding the exposure need to be considered to determine if there are social issues such as inadequate supervision that need to be addressed.

1. ABCDs (supportive care)
2. Antidots most poisoning treatment is supportive unless there is antidote
3. Decontamination removing active exposure either from skin or from the gut like by activated charcot
4. Enhanced elimination enhance how the body is eliminating the drug like by doing dialysis

Summary from Illustrated:

- Accidental poisoning in children is common in toddlers and young children
- most substances do not cause serious illness
- when an ingestion has occurred, identify the agent and assess its toxicity to plan management
- poisons potentially harmful in children include alcohol, acids and alkalis, bleach, digoxin, batteries, iron, paracetamol, petroleum distillates, salicylates, and tricyclic antidepressants
- assess the social circumstances behind why it happened.

Antidots

- Definition = a medicine taken or given to counteract a particular poison.
- Only a small proportion of poisoned patients are amenable to antidotal therapy
- Only a few poisoning is antidotal therapy urgent (e.g., CO, cyanide, organophosphate and opioid intoxication)

More limitations:

- most the time we don't know the poison
- not all of antidotes are available
- not all antidotes are effective
- It should be given at certain time

ABCDs (supportive care)

- Airway: Patent and maintainable
- Breathing: Spontaneous with GABL ,RR, SpO2 and WOB

  what do we check in pulse? rate, strong or weak, regular or irregular, while the rhythm is seen in ECG to know if it's sinus or no
- Circulation: HR, BP, Cap refill, Rhythm and Liver edge in infants

  children who are 4 years old and younger, the liver is a good sign for venous congestion in case of heart failure, because you can't see JVP
- Disability: Pupils ?equal and reactive . GCS . Glucocheck

  Some people add D for Dextrose to remember to check the glucose / some people add D for Decontamination as well
- Exposure , check the skin for any contamination or any source of bleed

POISON	ANTIDOTE	DOSAGE	ROUTE	ADVERSE EFFECTS, WARNINGS, COMMENTS
Acetaminophen The most common one	*N* -Acetylcysteine (Mucomyst)	140 mg/kg loading, followed by 70 mg/kg q4h	PO	Vomiting (patient-tailored regimens are the norm)
	N -Acetylcysteine (Acetadote)	150 mg/kg over 1 hr, followed by 50 mg/kg over 4 hr, followed by 100 mg/kg over 16 hr	IV	Anaphylactoid reactions (most commonly seen with loading dose) (Higher doses of the infusion are often recommended depending upon the acetaminophen level and the degree of injury)
Anticholinergics	Physostigmine	0.02 mg/kg over 5 min; may repeat q5-10min to 2 mg max	IV/IM	Bradycardia, seizures, bronchospasm *Note:* Do not use if conduction delays on ECG
Benzodiazepines	Flumazenil	0.2 mg over 30 sec; if response is inadequate, repeat q1min to 1 mg max	IV	Agitation, seizures; **do not use for unknown ingestions**
β Blockers	Glucagon	0.15 mg/kg bolus followed by infusion of 0.05-0.15 mg/kg/hr	IV	Hyperglycemia, vomiting

POISON	ANTIDOTE	DOSAGE	ROUTE	ADVERSE EFFECTS, WARNINGS, COMMENTS
Carbon monoxide	Oxygen	100% F_{io_2} via non–rebreather mask (or ET if intubated)	Inhalational	Some patients may benefit from hyperbaric oxygen (see text)
Cyanide	Cyanide kit:			
	Amyl nitrate	1 crushable ampule; inhale 30 sec of each min	Inhalation	Methemoglobinemia
	Sodium nitrate	0.33 mL/kg of 3% solution if hemoglobin level is not known; otherwise, based on tables with product	IV	Methemoglobinemia Hypotension
	Sodium thiosulfate	1.6 mL/kg of 25% solution; may be repeated q30-60min to max of 50 mL	IV	If inducing methemoglobinemia is contraindicated; consider only using the thiosulfate component of the kit
	Hydroxocobalamin (Cyanokit)	70 mg/kg (adults: 5 g) given over 15 min	IV	Flushing/erythema, nausea, rash, chromaturia, hypertension, headache
Digitalis	Digoxin-specific Fab antibodies (Digibind; DigiFab)	1 vial binds 0.6 mg of digitalis glycoside; #vials = digitalis level × weight in kg/100	IV	Allergic reactions (rare), return of condition being treated with digitalis glycoside

why Carbon monoxide (CO) poisoning happen?
The hemoglobin has higher affinity to CO more than the Oxygene so the oxyhemoglobin dissociation curve shifted to the left so the cell will not deliver enough oxygen

What about saturation?
not all devices pick up CO, so some if them will read it as normal saturation

POISON	ANTIDOTE	DOSAGE	ROUTE	ADVERSE EFFECTS, WARNINGS, COMMENTS
Opioids	Naloxone You need to know that Naloxone saves lives in both hospitals and street	0.01-0.1 mg/kg; adolescents/adults: 0.04-2 mg, repeated as needed; may give continuous infusion	IV	Acute withdrawal symptoms if given to addicted patients May also be useful for clonidine ingestions (inconsistent response)
Organophosphates	Atropine	0.05-0.1 mg/kg repeated q5-10min as needed	IV/ET	Tachycardia, dry mouth, blurred vision, urinary retention
	Pralidoxime (2-PAM)	25-50 mg/kg over 5-10 min (max: 200 mg/min); can be repeated after 1-2 hr, then q10-12hr as needed	IV/IM	Nausea, dizziness, headache, tachycardia, muscle rigidity, bronchospasm (rapid administration)

POISON	ANTIDOTE	DOSAGE	ROUTE	ADVERSE EFFECTS, WARNINGS, COMMENTS
Ethylene glycol, methanol	Fomepizole	15 mg/kg load; 10 mg/kg q12h × 4 doses; 15 mg/kg q12h until EG level is <20 mg/dL	IV	Infuse slowly over 30 min; If fomepizole is not available, can treat with oral ethanol (80 proof)
Iron	Deferoxamine	Infusion of 5-15 mg/kg/hr (max: 6 g/24 hr)	IV	Hypotension (minimized by avoiding rapid infusion rates)
Isoniazid (INH)	Pyridoxine	Empirical dosing: 70 mg/kg (max dose = 5 g) If ingested dose is known: 1 g per gram of INH	IV	May also be used for *Gyromitra* mushroom ingestions

I added this table summarizing the antidotes that was mentioned in the past slides, the ones that was mentioned by the Dr in **RED**

Poison	Antidote
Acetaminophen	*N* -Acetylcysteine (Mucomyst) (Acetadote)
Anticholinergics	Physostigmine
Benzodiazepines	Flumazenil
β Blockers	Glucagon
Carbon monoxide	Oxygen
Cyanide	Cyanide kit: Amyl nitrate / Sodium nitrate / Sodium thiosulfate / Hydroxocobalamin (Cyanokit)
Digitalis	Digoxin-specific Fab antibodies (Digibind; DigiFab)
Opioids	Naloxone
Organophosphates	Atropine / Pralidoxime (2-PAM)
Ethylene glycol, methanol	Fomepizole
Iron	Deferoxamine
Isoniazid (INH)	Pyridoxine

Decontamination

- The goal of decontamination is to minimize absorption of the toxic substance.
- Decontamination should not be routinely employed for every poisoned patient.
- **Dermal and ocular decontamination** remove any contaminated clothing and particulate matter, followed by flushing of the affected area with tepid water or normal saline.
  - 10-20 min of washing is recommended for most exposures.
  - Dermal decontamination, especially after exposure to **adherent or lipophilic** (e.g., organophosphates) agents, should include thorough cleansing with soap and water .clean him very well
  - Avoid water with highly reactive agents. avoid alkaline soaps that could agitate

GI Decontamination:

- In GI decontamination activated charcoal is the only thing that we can actually now do
- what is it? فحم in a serum form, that has very micro bores that can absorb and bind to medications
- the idea of it is if patient took 100 pill and came within an hour this means that the medication is still in the stomach so the activated charcoal can bind with it and get excreted instead of going to the body
- no body with his right mind will drink such thing , it is sour black and disgusting, so if the child is resisting to take it so don't force it
- if the pt is unconscious, would you put NGT to give it? NO PLEASE DON'T DO IT , the patient should be conscious and cooperative, the only exception is if the patient is intubated and having NGT so you can give it through NGT

- GI decontamination strategies are most likely to be effective **in the 1st hour after an acute ingestion.**
- GI decontamination at more than 1 hr after ingestion may be considered in patients who ingest toxic substances with these properties:
  - GI absorption may be delayed after ingestion of agents that slow GI motility (anticholinergic medications, opioids or TCA).
  - massive pill ingestions.
  - sustained-release preparations.
  - ingestions of agents that can form pharmacologic bezoars (e.g., enteric-coated salicylates).

- no body with his right mind will drink such thing , it is sour black and disgusting, so if the child is resisting to take it so don't force it
- if the pt is unconscious, would you put NGT to give it? NO PLEASE DON'T DO IT , the patient should be conscious and cooperative, the only exception is if the patient is intubated and having NGT so you can give it through NGT

Methods of GI Decontamination:

- Syrup Ipecac: By induce vomiting
  - Risk is more than benefit (no evidence)

- Gastric Lavage:
  - in most clinical scenarios, the use of gastric lavage is no longer recommended (no evidence)

Single dose Activated Charcoal:

- It has an extensive network of pores that provides a very large adsorptive surface area.
- 1g/kg or 50-100 g in adolescents and adults this is the dose
- A repeat dose of activated charcoal may be warranted in the cases of ingestion of an extended release product or, more commonly, with a significant salicylate poisoning as a result of its delayed and erratic absorption pattern. the only exception of using it after 1 hour if we have extended release medication or if there was a massive pills ingestion
- Not effective in :
  - Charged molecules (i.e., heavy metals lithium, iron)
  - liquids do not bind well to activated charcoal
  - Caustic agents

Table 63-9	Substances Poorly Adsorbed By Activated Charcoal
Alcohols Caustics: alkalis and acids Cyanide Heavy metals (e.g., lead) Hydrocarbons Iron Lithium	

Single dose Activated Charcoal:

- 20% of pt will vomit → must ensure that the patient's airway is intact or protected and that the patient has a benign abdominal exam.

- In the awake, uncooperative adolescent or child who refuses to drink the activated charcoal, there is relatively little utility and potential morbidity associated with forcing activated charcoal down a nasogastric tube, and such practice should be avoided.

- Constipation is a common side effect. (consider lactulose)

Enhanced Elimination:

- Urinary Alkalinization
- Hemodialysis
- Multiple-Dose Activated Charcoal
- Intralipid Emulsion Therapy

Urinary Alkalinization

- Making a molecule charged and hydrophilic ⟶ difficult to be absorbed through fat membrane Thus, the molecule is trapped within the renal tubules

- Accomplished via a continuous infusion of sodium bicarbonate–containing intravenous fluids, with a goal urine pH of 7.5-8. 8 actually is very alkaline we try not to make it reach to that

- Alkalinization of the urine is most useful in managing **salicylate and methotrexate toxicity.**

Hemodialysis

- Enhance the elimination of the toxin itself
- Also be useful to correct severe electrolyte disturbances and acid–base derangement
- Not all medications are dialysable, Toxins that are amenable to dialysis have the following properties:
  - low volume of distribution (<1 L/kg).
  - low molecular weight.
  - low degree of protein binding.
  - high degree of water solubility.
- **eg methanol, ethylene glycol, salicylates, theophylline, bromide, lithium, and, potentially, valproic acid.**

Management of a poisoned child or young person

Outline of management

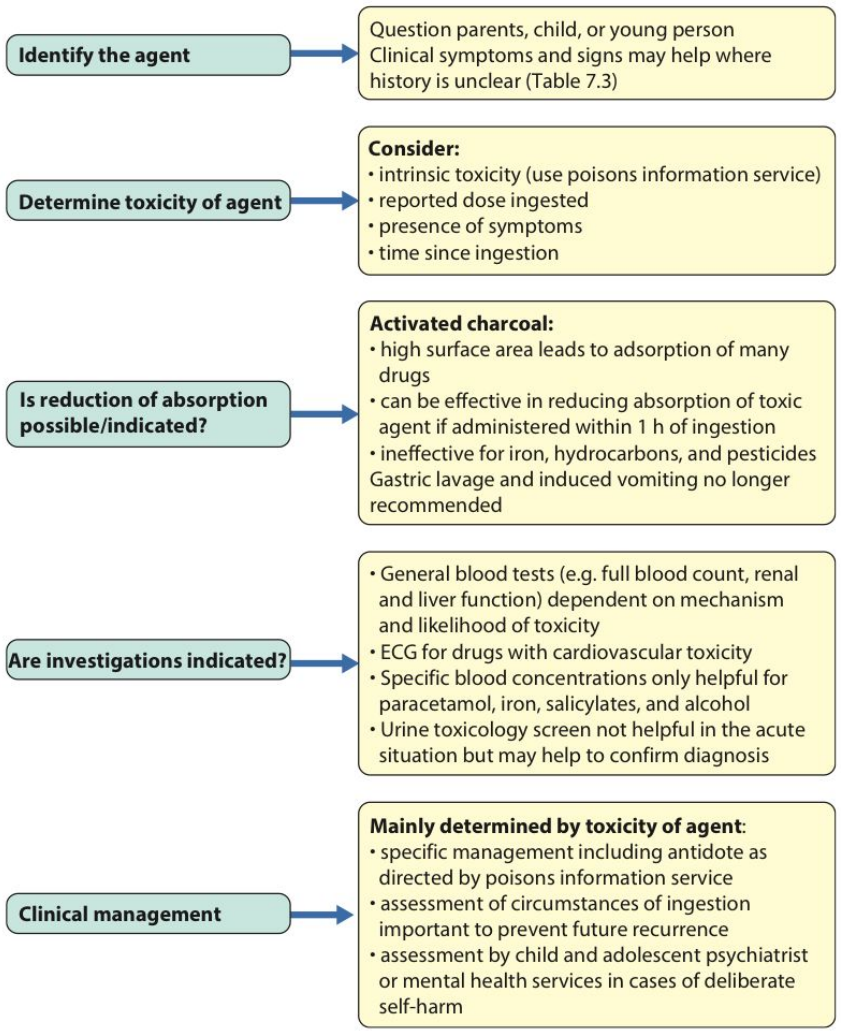


Figure 7.10 Outline of management of poisoning.

Table 7.2 Some poisons and their treatment

Agent	Clinical symptoms	Mechanism	Management
Paracetamol	Early: • abdominal pain, vomiting Later (12 h to 24 h): • liver failure	Initial gastric irritation Toxic metabolite (NAPQI) produced by saturation of liver metabolism	Risk assessed by measuring plasma paracetamol concentration Treat with intravenous acetylcysteine if concentration is high or liver function abnormal
Button batteries	Abdominal pain Gut perforation and stricture formation	Leakage: corrosion of gut wall due to electrical circuit production	X-ray of chest and abdomen to confirm ingestion and identify position Endoscopic removal is recommended if in the oesophagus, the object fails to pass, or symptoms are present (e.g. abdominal pain or melaena)
Carbon monoxide	Early: • headache, nausea Later: • confusion, drowsiness leading to coma	Binds to haemoglobin causing tissue hypoxia	High-flow oxygen to hasten dissociation of carbon monoxide The role of hyperbaric oxygen therapy is unclear
Salicylates	Early: • vomiting, tinnitus Later: • respiratory alkalosis followed by metabolic acidosis	Direct stimulation of respiratory centre Uncouples oxidative phosphorylation leading to metabolic acidosis and hypoglycaemia	Plasma salicylate concentration 2–4 h after ingestion helps to estimate toxicity Alkalinization of urine increases excretion of salicylates. Haemodialysis also effectively removes salicylate
Tricyclic antidepressants	Early: • tachycardia, drowsiness, dry mouth Later: • arrhythmias, seizures	Anti-cholinergic effects, interference with cardiac conduction pathways	Treatment of arrhythmias with sodium bicarbonate Support ventilation
Ethylene glycol (anti-freeze)	Early: • intoxication Later: • tachycardia, metabolic acidosis leading to renal failure	Production of toxic metabolites that interfere with intracellular energy production	Fomepizole inhibits the production of toxic metabolites; alcohol may also be used but has more adverse effects Haemodialysis to remove toxic metabolites in severe cases
Alcohol (accidental or experimenting by older children)	Hypoglycaemia Coma Respiratory failure	Direct inhibitory effect on glycolysis in the liver and neurotransmission in the brain	Monitor blood glucose and correct if necessary. Support ventilation if required Blood alcohol levels may help to predict severity

Table 7.2 Some poisons and their treatment—cont'd

Agent	Clinical symptoms	Mechanism	Management
Iron	Initial: vomiting, diarrhoea, haematemesis, melaena, acute gastric ulceration Latent period of improvement 6–12 h later: drowsiness, coma, shock, liver failure with hypoglycaemia, and convulsions Long term: gut strictures	Local corrosive effect on gut mucosa Disruption of oxidative phosphorylation in mitochondria leads to free radical production, lipid peroxidation, and metabolic acidosis	Serious toxicity if >75 mg/kg elemental iron ingested Serum iron level 4 h after ingestion is the best laboratory measure of severity Intravenous desferoxamine chelates iron and should be administered in cases of moderate-to-severe toxicity
Hydrocarbons (e.g. paraffin, kerosene)	Pneumonitis Coma	Low viscosity and high volatility makes aspiration easy, resulting in direct lung toxicity Direct inhibitory effect on neurotransmission in the brain	No specific antidote – supportive treatment only
Organophosphorus pesticides	Cholinergic effects: • salivation, lacrimation, urination, diarrhoea and vomiting, muscle weakness, cramps and paralysis, bradycardia. and hypotension Central nervous system effects: • seizures and coma	Inhibition of acetylcholinesterase resulting in accumulation of acetylcholine throughout the nervous system	Supportive care Atropine (often in large doses) as an anticholinergic agent Pralidoxime to reactivate acetylcholinesterase

NAPQI, *N*-acetyl-*p*-benzoquinone imine.

Table 7.3 Physical findings that may help identify different classes of drugs in overdose

Type of effect	Heart rate and blood pressure	Respiratory rate	Temperature	Pupils	Sweating
Anticholinergic (e.g. tricyclic antidepressants, antihistamines)	Increased	No effect	Increased	Dilated	Reduced
Opioid (e.g. morphine, codeine)	Reduced	Reduced	Reduced	Constricted	Reduced
Sympathomimetic (e.g. cocaine, amphetamines)	Increased	Increased	Increased	Dilated	Increased
Sedative-hypnotic (e.g. anticonvulsants, benzodiazepines)	Reduced	Reduced	Reduced	No effect	Reduced