

Environmental and Toxicologic Emergencies

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04 Important specific intoxications like: Paracetamol/ Aspirin/ TCA

05 Environmental emergencies: Hyperthermia & heat shock Hypothermia High altitude illness Drowning



Definitions :

Toxicology : the branch of science concerned with the nature , effects , detection of poison .



Medical Toxicology : a field of medicine dedicated to the evaluation and treatment of poisoned patients



General steps represent of the initial clinical encounter with a poisoned patients :

CLINICAL STABILIZATION:

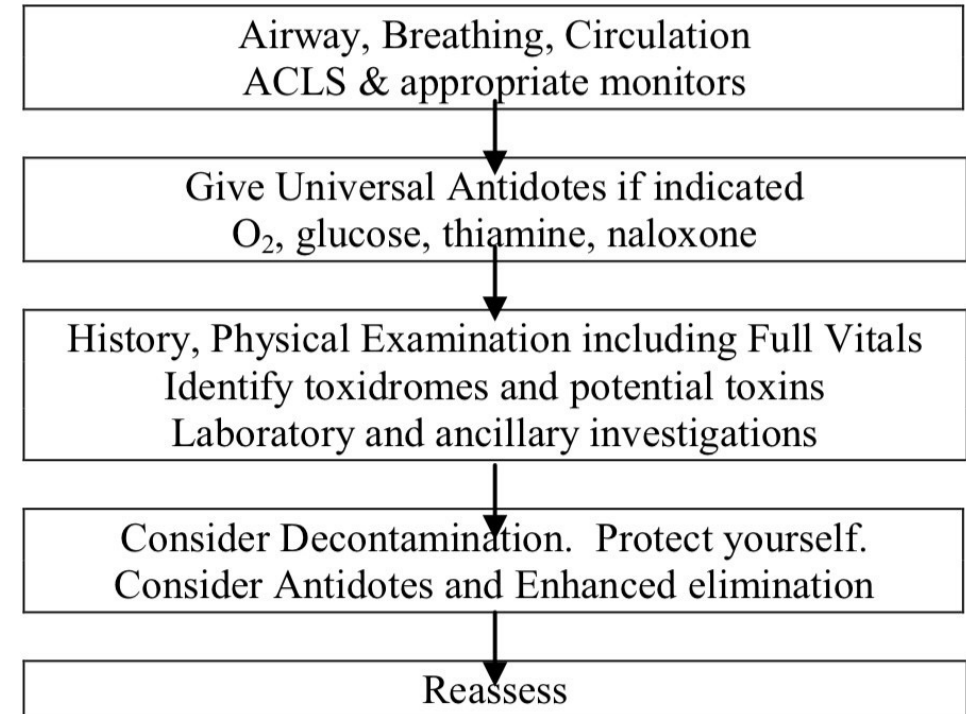
1. Stabilization of the patient .
2. Clinical evaluation (history , physical , laboratory ,and radiology)
3. Prevention of further toxin absorption .
4. Enhancement of toxin elimination .
5. Administration of antidote .
6. Supportive care and clinical follow - up .



³/₄ Clinical Stabilization : The first priority in treating a poisoned patient is stabilization . Initial focus is on assessing vital signs, respiration, and circulation. Some toxins or drugs may induce seizures early on in the presentation. Stabilizing a critically ill, poisoned patient often requires interventions such as supporting ventilation, circulation, and oxygenation. In severe cases, treatment may need to start before full stabilization is achieved.

INITIAL APPROACH

- $\frac{3}{4}$ First, treat the patient and not the poison.
- $\frac{3}{4}$ Second, remember that it is the dose, not the substance that makes the poison.
- $\frac{3}{4}$ Third, clinical manifestations of poisoning may be delayed for hours, even days after the exposure. When signs and symptoms do develop, their onset may be precipitous.



CLINICAL HISTORY IN THE POISONED PATIENT:

- ¾ The primary goal of history taking is to determine the substance ingested along with duration of exposure.
- ¾ It is very important to screen for a suicide setting. If he is not cooperative or provide incorrect details, we take information from his family members or from the pharmacist or ER technicians at the scene.

¾ HISTORY INCLUDE :

- ¾ 1. Time, route, duration.
- ¾ 2. Name, level of exposure (if unknown assume highest level), dose (refer to various information to estimate it).
- ¾ 3. Asses symptoms' time of onset, nature and severity.
- ¾ 4. Time of first aid measure it any
- ¾ 5. Family history of diseases and drug therapy.



PHYSICAL EXAM:

General of appearance : Level of consciousness , Posture, Odors

Vital Signs : Temperature , HR , BP , RR , SpO2

Neurologic : Pupils , Reflexes , Motor function , cranial nerves

CVS : Peripheral Perfusion , JVP

RS : Breath Sounds , Work of Breathing , Cyanosis

Skin : Sweating , Dry skin , Rashes

Biochemical Indecation

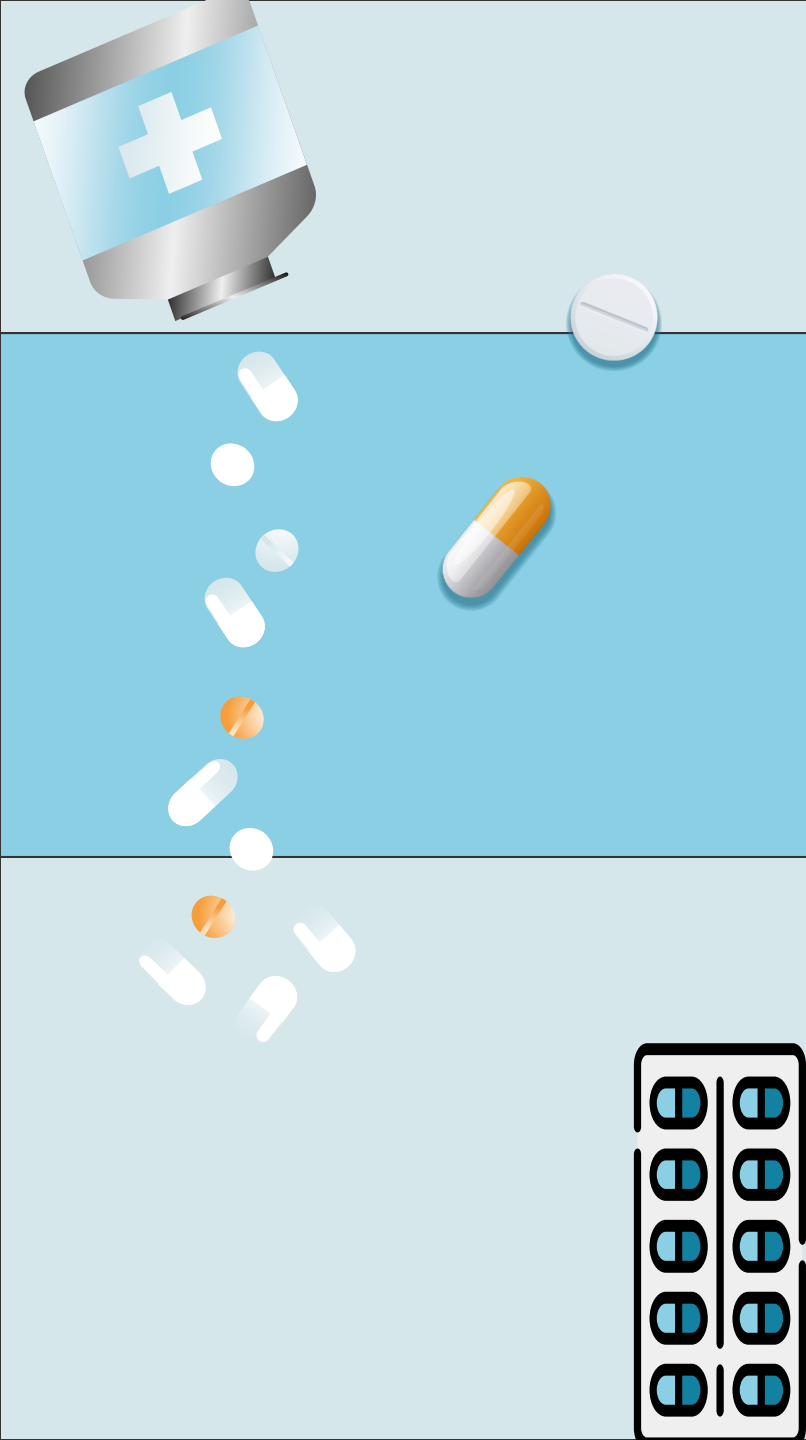


$\frac{3}{4}$ The poisoned patient may present with symptoms secondary to many systems' involvement. Classic presentations could include altered mental status (coma or seizures, hallucinations), dysrhythmias, altered vitals, altered saturations. There is an extensive toxicological differential for every symptom expressed.

$\frac{3}{4}$ Biochemical markers are laboratory values that provide additional evidence of poisoning and physiological effects.

$\frac{3}{4}$ Common examples include:

$\frac{3}{4}$ Blood glucose levels (hypoglycemia or hyperglycemia can indicate insulin, oral hypoglycemics, toxic alcohols) Electrolytes and acid-base status (e.g., metabolic acidosis in salicylate or lactic acid poisoning) Serum drug levels (e.g., acetaminophen, salicylate, lithium levels) Liver function tests (elevated in hepatotoxic substances like acetaminophen) Creatine kinase (CK) (elevated in muscle damage, such as in rhabdomyolysis due to toxins) Arterial blood gases (ABG) (for oxygenation and acid-base balance) Carboxyhemoglobin (for carbon monoxide poisoning) Cholinesterase levels (for organophosphate poisoning)



DECONTAMINATION

- **Definition:** Processes used to remove or neutralize contaminants.
- **Routes of exposure to contamination:** toxins can enter through various routes (skin, hair, nails, ..) **And most commonly is through GI tract.**
- The **sooner** the decontamination is initiated the more it limits the amount of the drug absorbed into circulation leading to **better** potential outcomes.



TYPES OF DECONTAMINATION:

Gastric lavage

Activated charcoals

Whole bowel irrigation

Cathartics



ACTIVATED CHARCOAL:



- **MOA:** bind(adsorbe) to many xenobiotics(Toxins) in the GI tract to prevent absorption into Bloodstream
- **Indications:** within **1 hour** of ingestion for toxins absorbed through GI tract and beyond 1 hour for substance that delay GI emptying or form solids masse
- **Contraindications:** not effective for alcohols, heavy metals, acids and any substances don't bind well to charcoal

CATHARTICS:

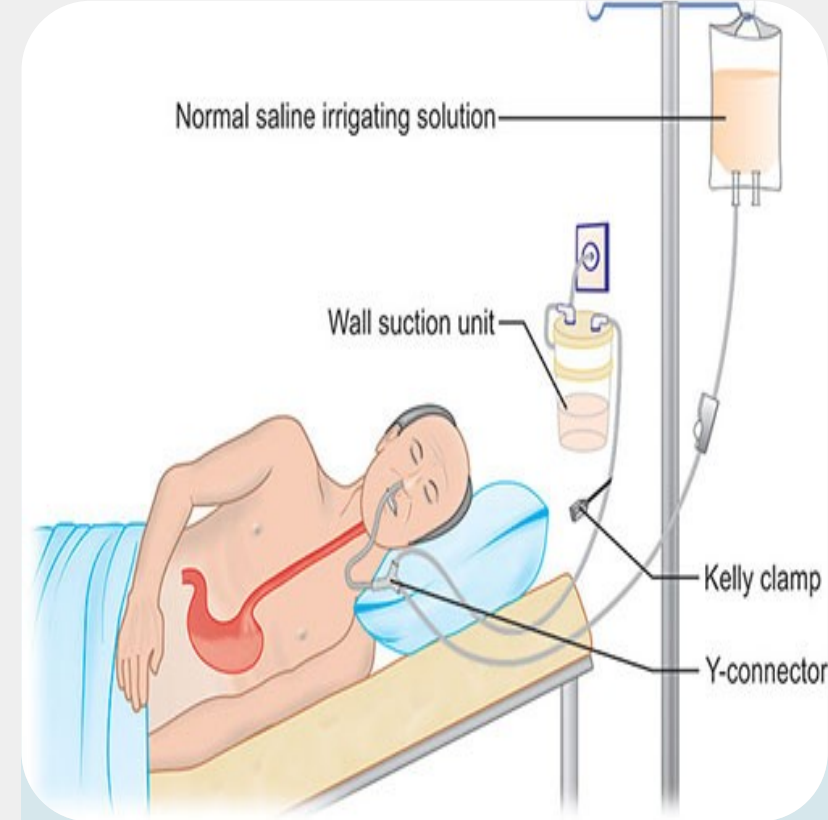
Cathartic of choice are succharides e.g. sorbitol

- **MOA:** they stimulate the peristaltic movement and accelerate the removal of toxins or contaminations.
- They can be used alongside activated charcoals. While activated charcoal prevents absorption, cathartics promote elimination of any unabsorbed toxins ,thus reducing the overall toxic load.
- **Contraindications:** in renal impairment (can cause electrolytes imbalance),bowel obstructions , sever dehydration and corrosive ingestion



GASTRIC LAVAGE (STOMACH PUMPING):

- **MOA:** inserting large orogastric tube into stomach; room temperature fluid then removed by suction until the toxic stomach contents removed.
- **Indications:** rarely recommended, may be considered in certain severe intoxications within **1-2 hours** of ingestion.
- **Contraindications:** ingestion of caustic substance (acids, alkalis) or hydrocarbons or lithium, patients who are not able to protect their airway, patients who are at high risk of perforation and hemorrhage.



WHOLE BOWEL IRRIGATION(WBI)

- **MOA:** administer polyethylene glycol(PEG) via NG tube at rates of 1-2L /hr in adults or 500 ml/hr in infants until rectal effluent is clear.
- **Indications:** considered for cases with sustained release or slow release medications(diltiazem, verapamil), certain metals(iron) and drugs carried by body packers or stuffers.
- **Contraindications:** should be avoided in patients with active diarrhea, bowel obstruction, unprotected airway.



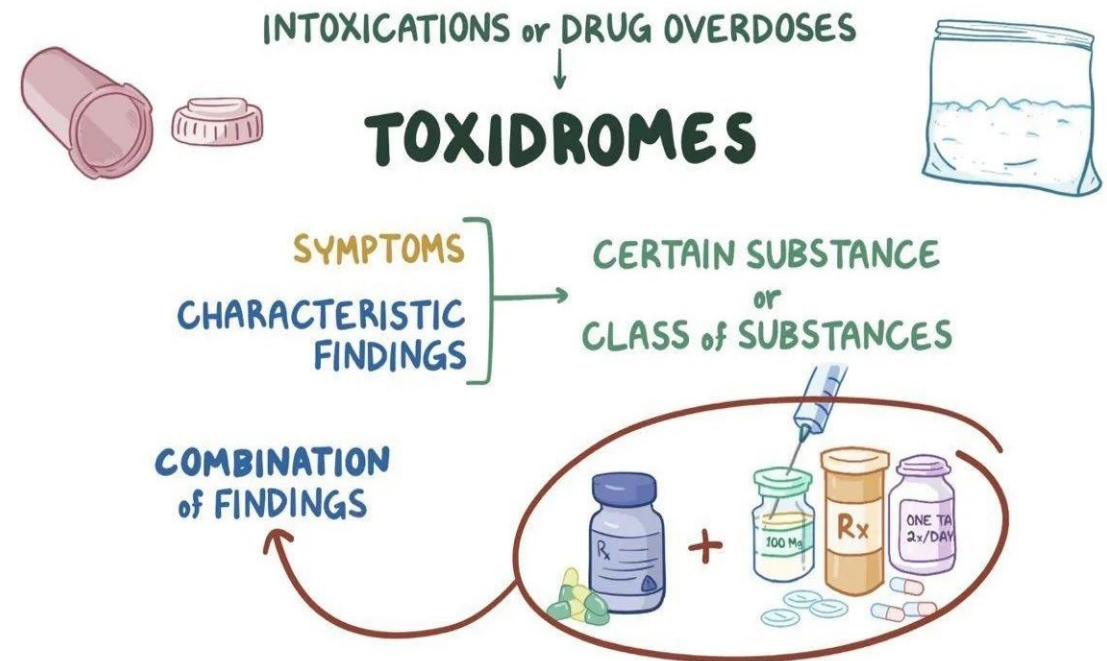
Toxidromes

Definition:

A collection of signs and symptoms commonly associated with a substance or class of substances that has reached toxic levels in the body.

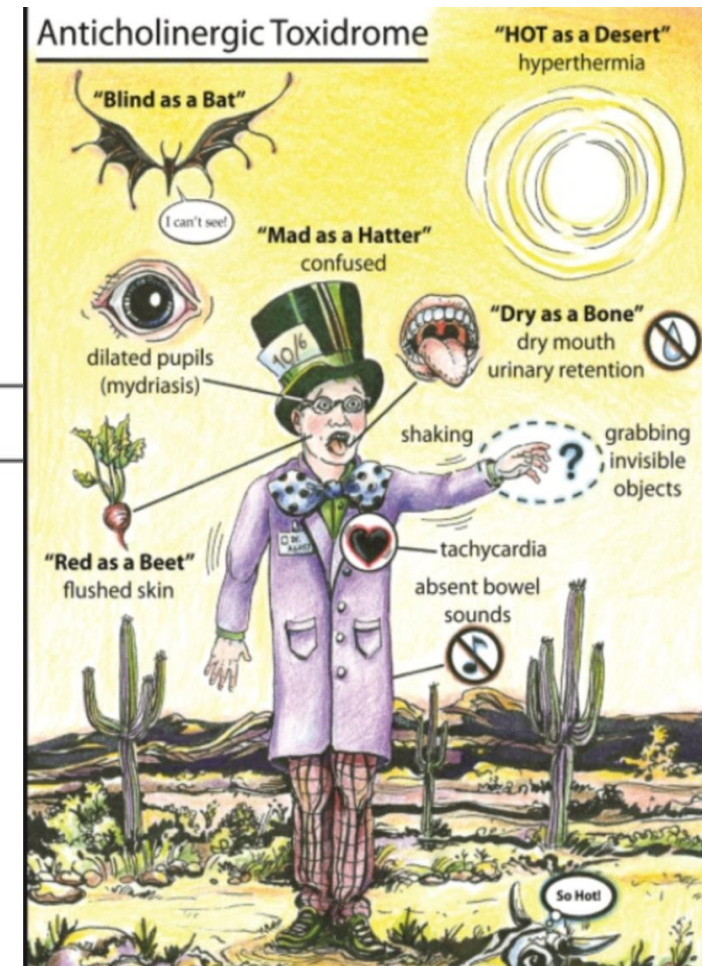
Classic toxidromes

- Anticholinergic
- Sympathomimetics
- Cholinergic
- Opioid



Toxidrome	Overdose Signs and Symptoms	Examples of Drugs
Cholinergics	"DUMBELS" Diaphoresis, D iarrrhea, D ecreased blood pressure U rination M iosis B ronchospasm, B ronchorrhea, B radycardia E mesis, E xcitation of skeletal muscle L acrimation S alivation, S eizures	Natural plants: mushrooms, trumpet flower Anticholinesterases: physostigmine, Insecticides (organophosphates, carbamates) Nerve gases

Toxidrome	Overdose Signs and Symptoms		Examples of Drugs
Anticholinergics	Hyperthermia	"Hot as a hare"	Antidepressants (e.g. TCAs)
	Dilated pupils	"Blind as a bat"	Cyclobenzaprine (Flexeril®)
	Dry skin	"Dry as a bone"	Carbamazepine
	Vasodilation	"Red as a beet"	Antihistamines (e.g. diphenhydramine)
	Agitation/hallucinations	"Mad as a hatter"	Antiparkinsonians
	Ileus	"The bowel and bladder lose their tone and the heart goes on alone"	Antipsychotics
	Urinary retention		Antispasmodics
	Tachycardia		Belladonna alkaloids (e.g. atropine)



Toxidrome	Overdose Signs and Symptoms	Examples of Drugs
Sympathomimetics	Increased temperature CNS excitation (including seizures) Tachycardia, hypertension Nausea and vomiting Diaphoresis Dilated pupils	Amphetamines, caffeine, cocaine, LSD, PCP Ephedrine and other decongestants Thyroid hormone Sedatives, EtOH withdrawal

Sympathomimetic Toxidrome

Mnemonic: **"MATHS"**

M : Mydriasis
A : Agitation, arrhythmia, angina
T : Tachycardia
H : Hypertension, hyperthermia
S : Seizure, sweating



Management:

1. Treat agitation, HTN, and seizures with benzodiazepines
2. Avoid pure β -blockers due to unopposed alpha agonism

Toxidrome	Overdose Signs and Symptoms	Examples of Drugs
Narcotics, Sedatives/Hypnotics, EtOH	Hypothermia Hypotension Respiratory depression Dilated or constricted pupils (pinpoint in opiate OD) CNS depression	EtOH Benzodiazepines Opiates (morphine, heroin, etc.) Barbiturates GHB

Narcotic (Opioid) Toxidrome

Mnemonic: **"CPR-3H"**

C : Coma
P : Pinpoint pupils
R : Respiratory depression
H : Hypotension
H : Hypothermia
H : Hyporeflexia

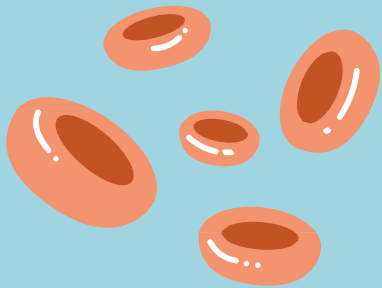
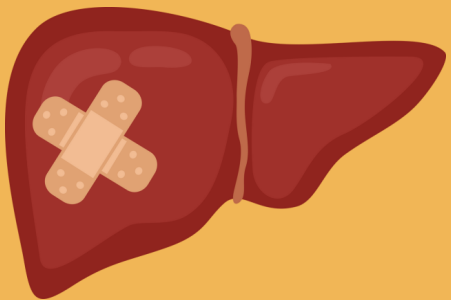


NOTE: Meperidine (*Demerol*) will not cause miosis

Antidote: Naloxone

Start with **0.04 mg** and titrate up q 2-3 min as need for ventilation to 0.5 mg, 2 mg, 5 mg, up to max 10-15 mg

PARACETAMOL TOXICITY



Nehaya shareef

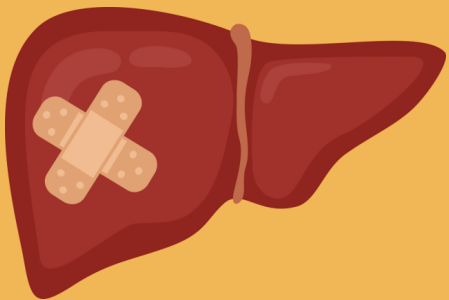
paracetamol or N-ACETYL-PARA-AMINOPHENOL (APAP) or acetaminophen

**Toxic exposure occurs
when an adult ingests:**

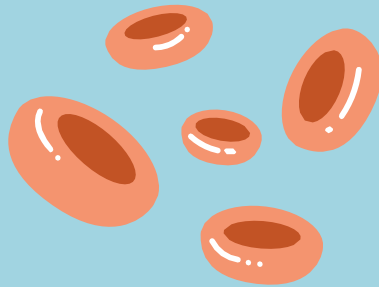
- 10 grams or 200 mg/kg
in a single dose or over
24 hours.
- 6 grams or 150 mg/kg
per day for 2 days
(20 pills a day or 16,8 a day)

Ex. if a 60kg teen wanted
attempted suicide by 500mg
panadol pills how many pills does
it need for it to be toxic ?

Most common cause of liver failure



**Serum level
(Rumack-Matthew nomogram)**



Has an antidote



**$200\text{mg/kg} \times 60\text{kg} = 12,000\text{mg}$
 $12,000\text{ mg} / 500\text{ mg per pill} =$
24 tablets**

Acetaminophen

10%

90%

Pathway saturated in overdose

To be water soluble and easily excreted in urine

CYP2E1

Therefore a larger proportion is oxidized by the CYP450 to NAPQ

NAPQI



N-acetyl-p-benzoquinone imine

Glucoronide and sulfate metabolites (Non toxic)



hepatic glutathione stores are decreased

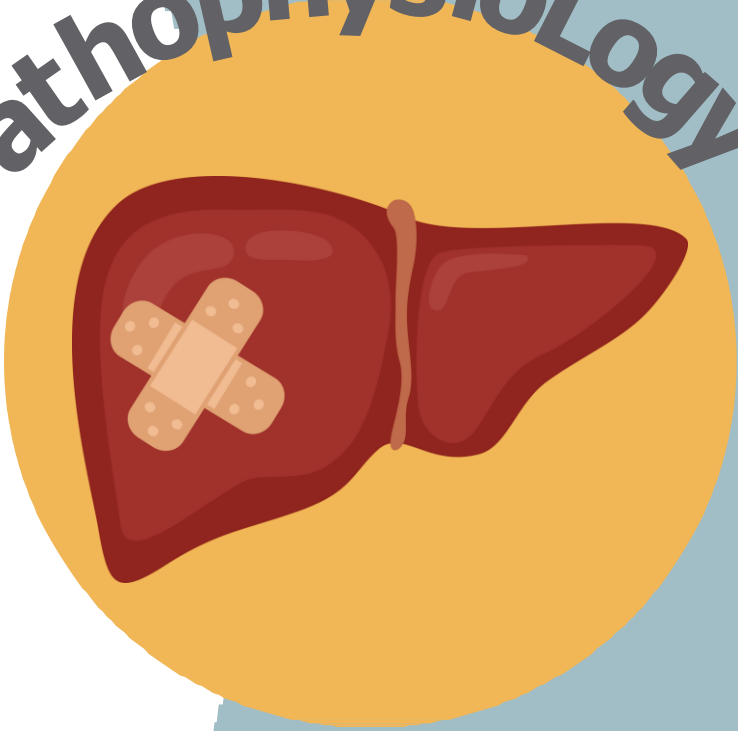
Glutathione

Hepatocyte necrosis

Mercapturic acid and cysteine Non toxic metabolites

NAPQI binds to cellular proteins (adduct formation). (especially in mitochondria) oxidative stress and mitochondrial dysfunction (e.g. cessation of ATP formation, and mitochondrial membrane rupture) resulting in cellular necrosis

Pathophysiology



nausea, vomiting, pain in upper right quadrant, confusion, bleeding

Clinical features

1

Confirm toxicity :

serum
acetaminophen
concentration

2

Confirm toxicity :

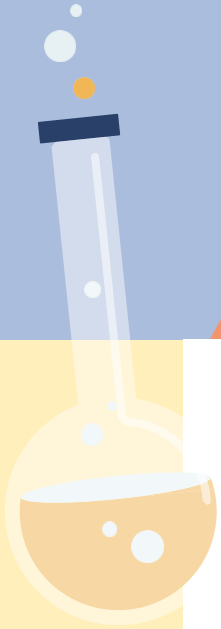
the time of
ingestion

3

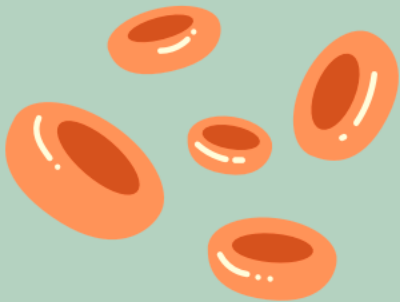
Additional lab

1. **Electrolytes**: renal function and metabolic status.
2. **Glucose**: hypoglycemia; relevant in altered mental status.
3. **BUN and Creatinine**: elevated levels indicate potential kidney injury.
4. **Transaminases (ALT/AST)**: elevated levels indicate liver injury.
5. **(CBC)**
6. **Prothrombin Time (PT)**: prolonged PT suggests liver dysfunction.
7. **Acetylsalicylic Acid (ASA)**: aspirin overdose.
8. **Urine Toxicology Screen**: poly-drug overdose.*
9. **(ECG)**

*coingestion in the suicidal patient



EVALUATE & MONITOR
SERUM LEVEL



RUMACK MATTHEW NOMOGRAM



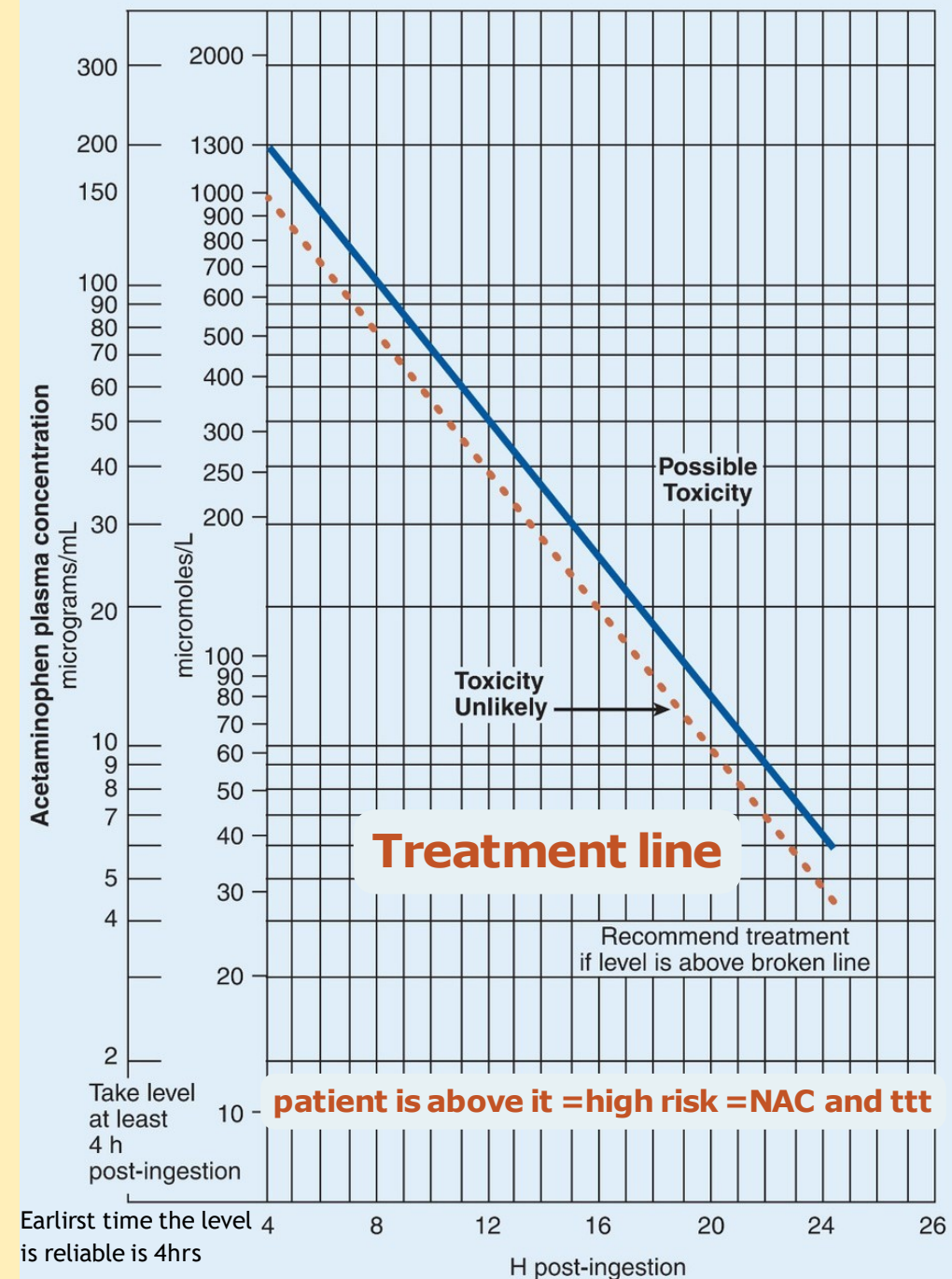
Single exposure



Window **4hrs-24hrs**
post ingestion

Not applicable for chronic overdose or
unkown timing

normally decreases over time



Acetaminophen

10%

90%

Pathway
saturated in
overdose

CYP2E1

Fomepizole

NAPQI



Glucoronide
and sulfate
metabolites
(Non toxic)

Glutathione

Hepatocyte
necrosis

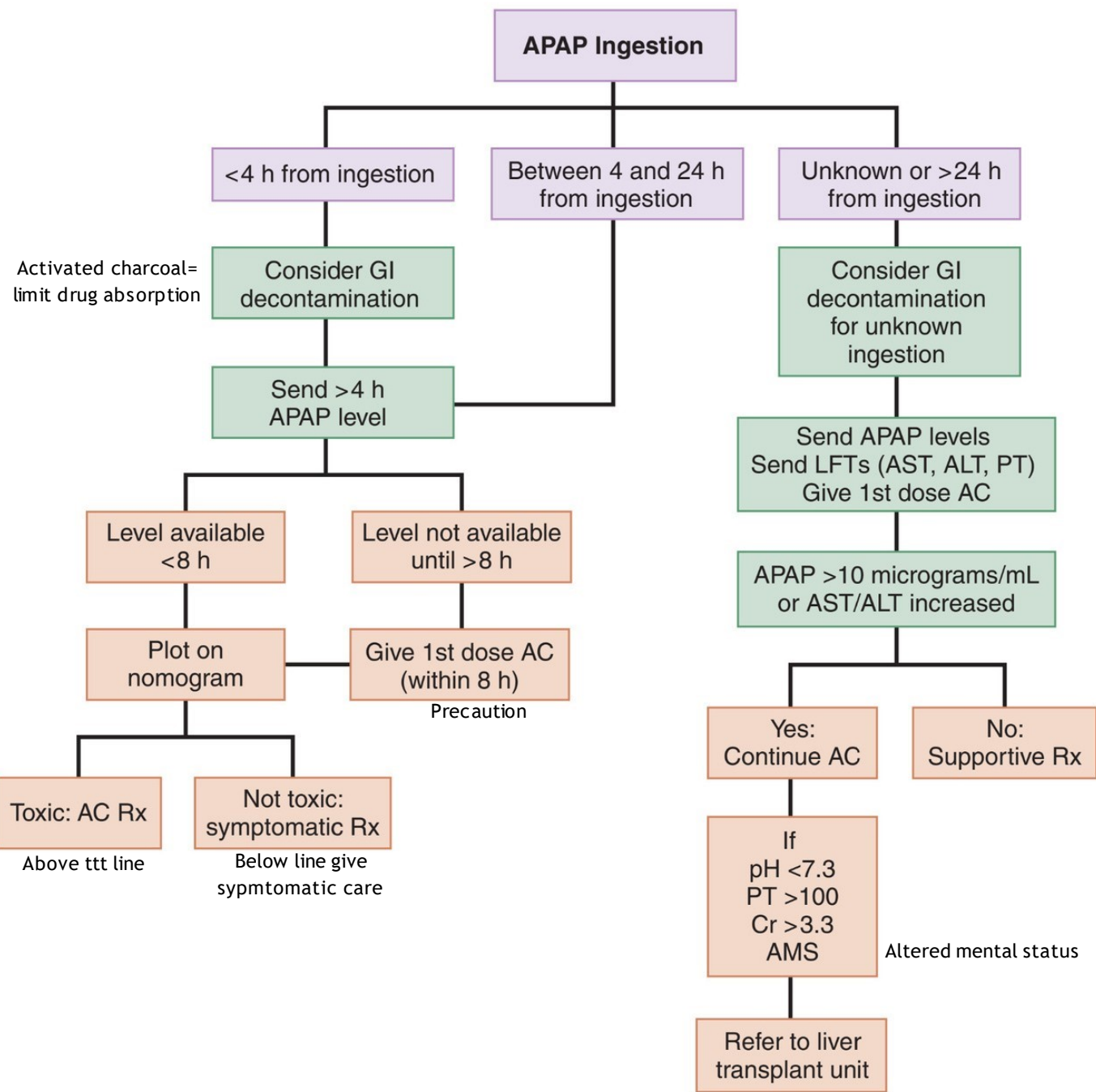
Mercapturic
acid and
cysteine
Non toxic
metabolites

NAC

Provides cysteine
for glutathione
synthesis and
possibly
Form adduct
directly with it.

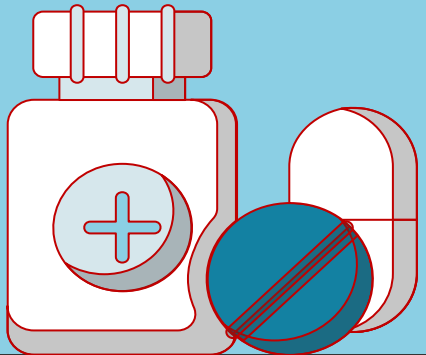
N-ACETYL CYSTEINE





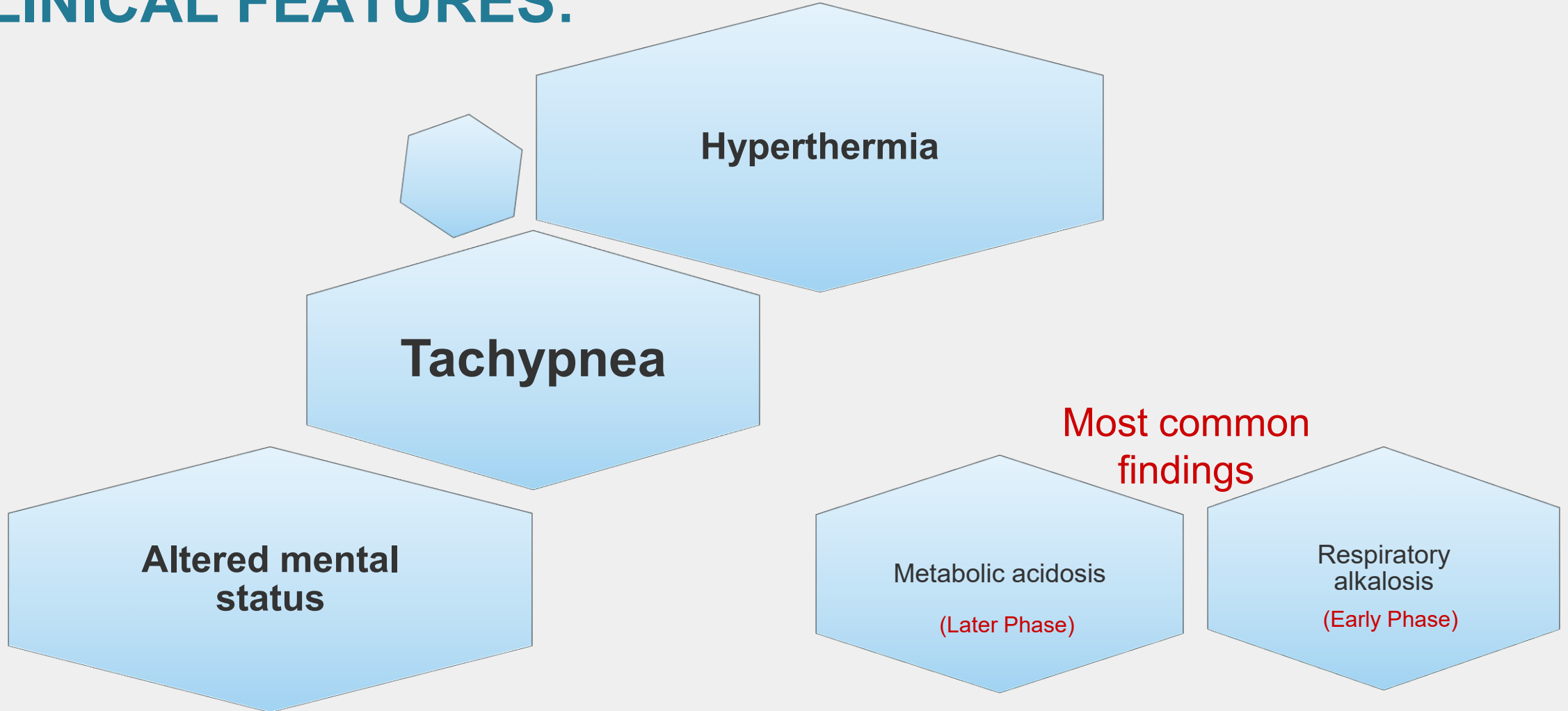


Aspirin and salicylate toxicity



Aspirin and salicylate toxicity

CLINICAL FEATURES:



Aspirin and salicylate toxicity

Severity Grading of Salicylate toxicity in adult:

	Mild	Moderate	Severe
Acute ingestion (dose)	<150 mg/kg	150-300 mg/kg	>300 mg/kg
End-organ toxicity	Tinnitus Hearing loss Dizziness Nausea/Vomiting	Tachypnea Hyperpyrexia Diaphoresis Ataxia Anxiety	Abnormal mental status Seizures Acute lung injury Renal failure Cardiac arrhythmias Shock



Aspirin and salicylate toxicity

CLINICAL FEATURES:

- **Chronic or "therapeutic" (repeated dose) poisonings** are generally **more serious and associated with higher mortality** than **acute overdoses** and are typically encountered in **elderly patients with multiple medical problems**



Aspirin and salicylate toxicity

Management and Treatment:

- **ABC**
- **Activated charcoal to reduce absorption**
- **Enhancing elimination:**
 1. **Alkalization of urine:** administering sodium bicarbonate, enhance ASA (aspirin) protein binding and urinary elimination.
 2. **Hemodialysis:**
 - All cases with ASA level in excess of 100 mg/dl.
 - Chronic toxicity + (60-80 mg/dl)
 - Clinical deterioration, Renal insufficiency or failure, Severe acid-base disturbance, Altered mental status, Adult respiratory distress syndrome.





TCA Toxicity

TCA Toxicity Presentation

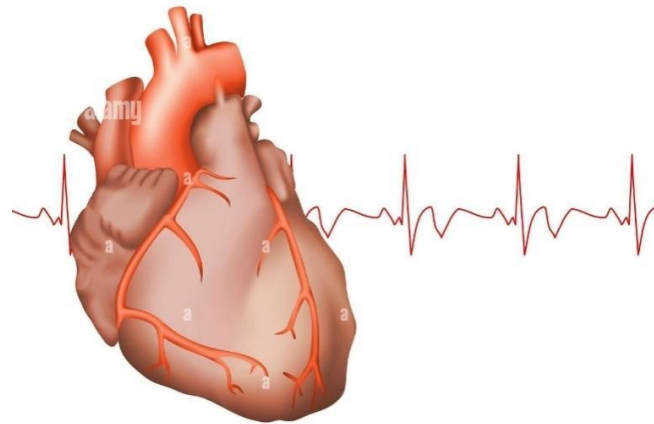
ECG Findings

Treatment With Bicarbonate

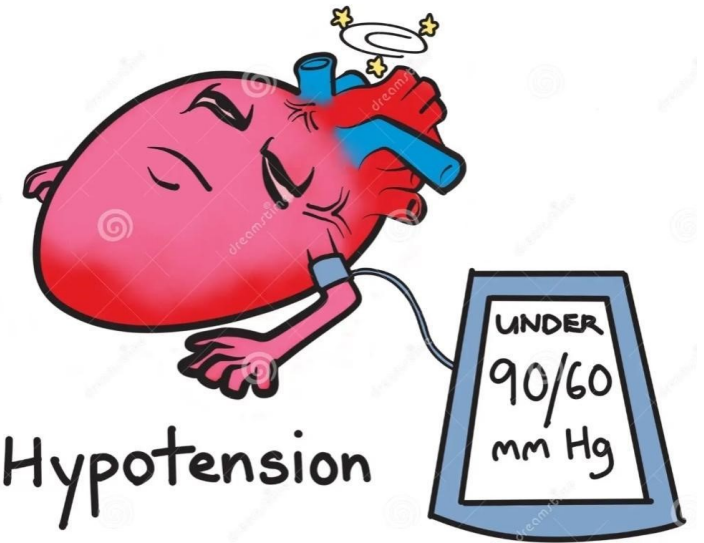
Seizure



Arrhythmia



Hypotension



Anticholinergic Toxidrome

"HOT as a Desert"
hyperthermia

"Blind as a Bat"



I can't see!

"Mad as a Hatter"
confused



dilated pupils
(mydriasis)



"Dry as a Bone"
dry mouth
urinary retention



shaking



"Red as a Beet"
flushed skin

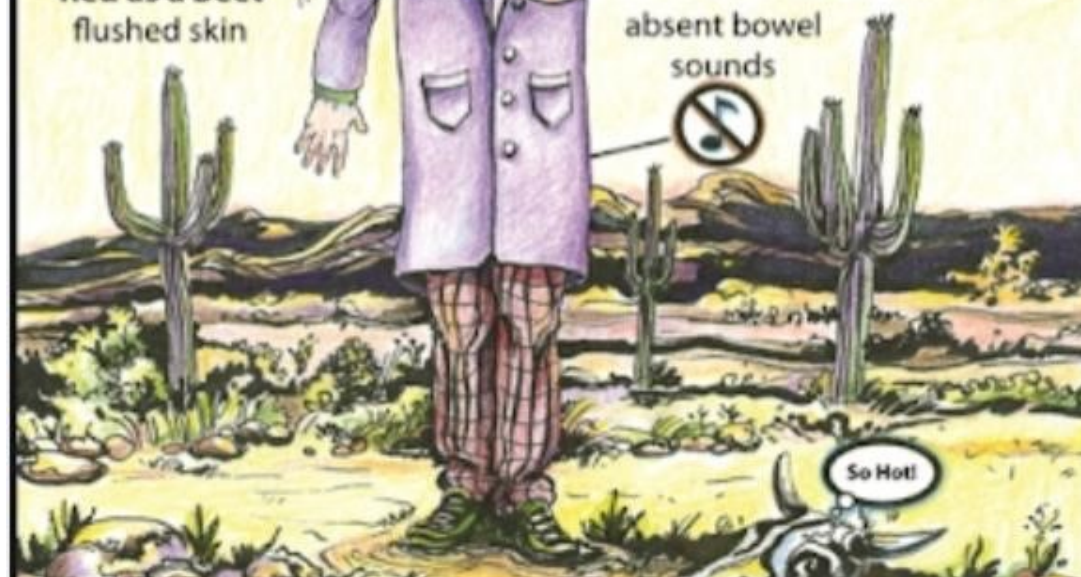
grabbing
invisible
objects

tachycardia

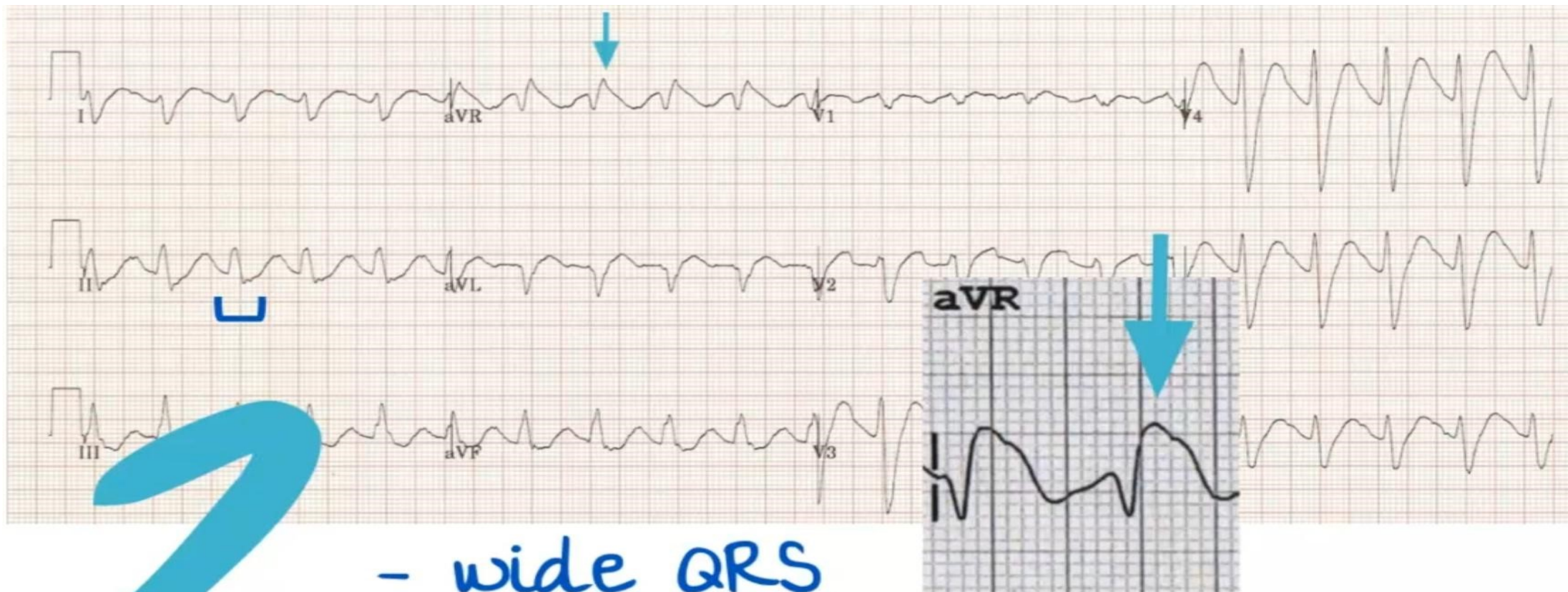
absent bowel
sounds



So Hot!



ECG



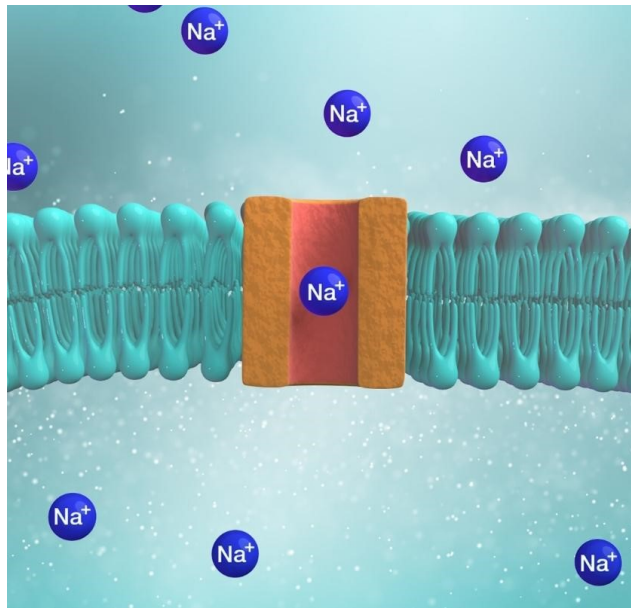
- wide QRS

- dominant R wave in aVR

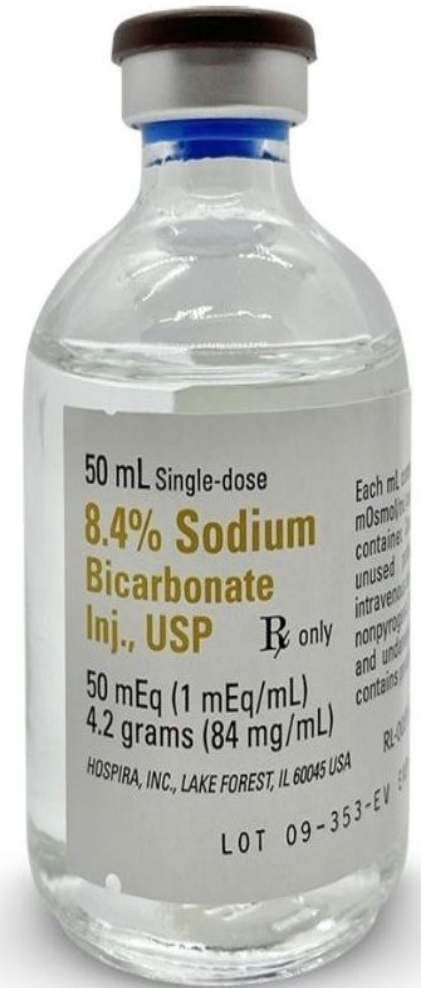
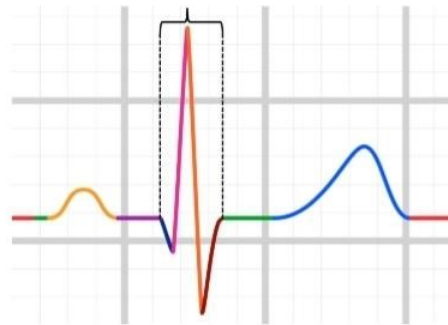
(>3mm)



PH SCALE



QRS Complex



Dose ?!

Supportive

- * Very low BP $\Rightarrow$ Pressor "NE"
- * Seizure / Need for sedation $\Rightarrow$ Benzodiazepine

Stabilise + Prevent Complications

→ ABC, ECG, IV Fluid

→ Decontamination

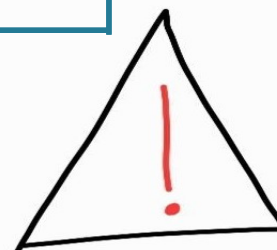
In First 2h

→ Bicarbonate

TPQRS
> 100

Titrate

Lipid Emulsion Therapy



* Physostigmine

* Na^+ , K^+ , Ca^{+2} , Beta
Channels Blockers

* Flumazenil

A decorative vertical bar on the left side of the slide, composed of light blue and white segments. It features several 3D-rendered pills: two yellow and white capsules at the top, a white round tablet with a score line in the middle, and several red and white capsules at the bottom.

Dose?!

Stabilize and prevent complications
ABC, decontamination, bicarbonate

Supportive
Pressor ,benzodiazepine

Lipid emulsion therapy

Contraindication
Physostigmine , Ca/K/Na/beta blockers
,Flumazenil

TCA =tricyclic antidepressants

Ex: amitriptyline , nortriptylin , imipramine , doxepin

**Narrow therapeutic index

Toxicity Presentation

Symptoms:

CVS , CNS , Anticholinergic

CVS - hypotension / arrhythmias "most dangerous "

Hypotension -> blocking α adrenergic receptors

Arrhythmia / tachycardia mainly -> blocking sodium channels , that cause slowing down cardiac conduction (wide QRS)

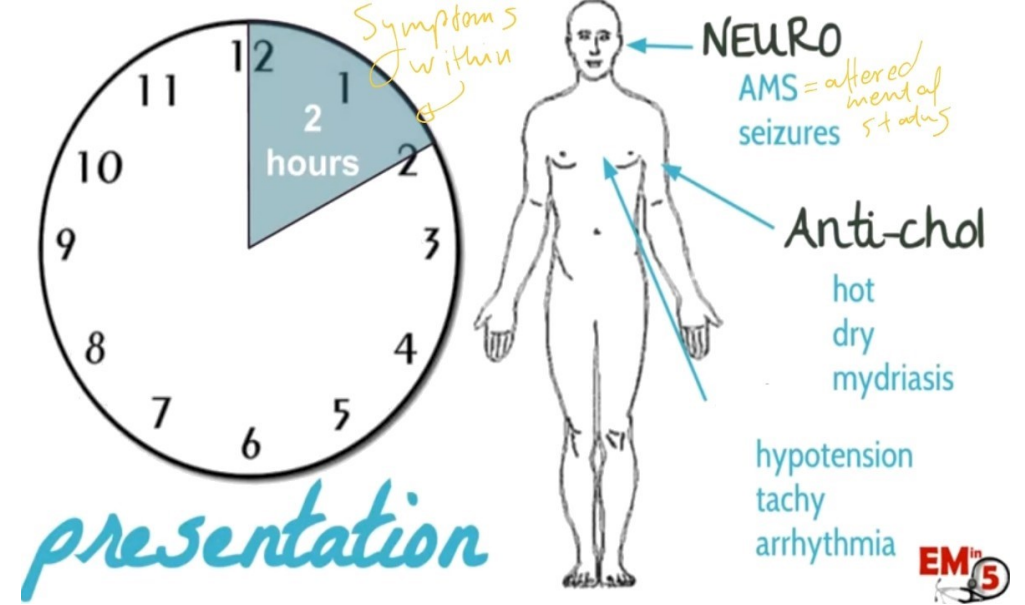
CNS - seizures , confusion , agitation , hallucination , coma if severe

-> serotonin , NE reuptake inhibition with antagonising GABA and serotonin receptors

Anticholinergic - dry mouth , urinary retention , blurred vision , pupil dilation , hyperthermia , flushing

-> blocking cholinergic receptor

In severe cases - respiratory depression and coma



ECG

**Any patient comes altered , you're not sure what they took -> do ECG

**QRS > 100 = seizure , QRS > 160 = arrhythmia

ECG in TCA toxicity

- Wide QRS
- Prominent R wave in aVR (> 3mm)
- prolonged PR , QT intervals / Right axis deviation (right bundle branch blocking) / Sinus tachycardia

Treatment

1) Dose ? To know if significant and what management to consider

2) Stabilizing and preventing complication

-ABC , airway protection / oxygenation /IV fluid if hypotensive + do ecg

-Decontamination during the first 2 h , aftering assuring the safety of the airway and patient is awake to tolerate it . Give active charcoal (1g/kg)

-If QRS > 100, give bicarbonate (100mEq)

** If prolonged and delayed cardiotoxicity -> ICU

** Repeat and Titrate : repeat the ECG over and over again until narrowing the QRS and improving the BP

* NaHCO_3

↳ Counteract Na^+ Channels Blockade

=
↳ QRS duration

=
↳ risk of arrhythmias

↳ Alkalinization of Blood : $\uparrow \text{pH}$ = $\downarrow$ TCA Binding

=
↳ Correcting Metabolic Acidosis

"less active"

**If patient was asymptomatic with no clinical signs of intoxication and normal ECG -> observation for 6 hours with proper decontamination

**If very severe and other interventions failed to reach stabilization Use -> Lipid emulsion therapy

**Supportive:

Very low BP -> pressor (NE)

Seizure or to achieve sedation -> Benzodiazepine

**Contraindications

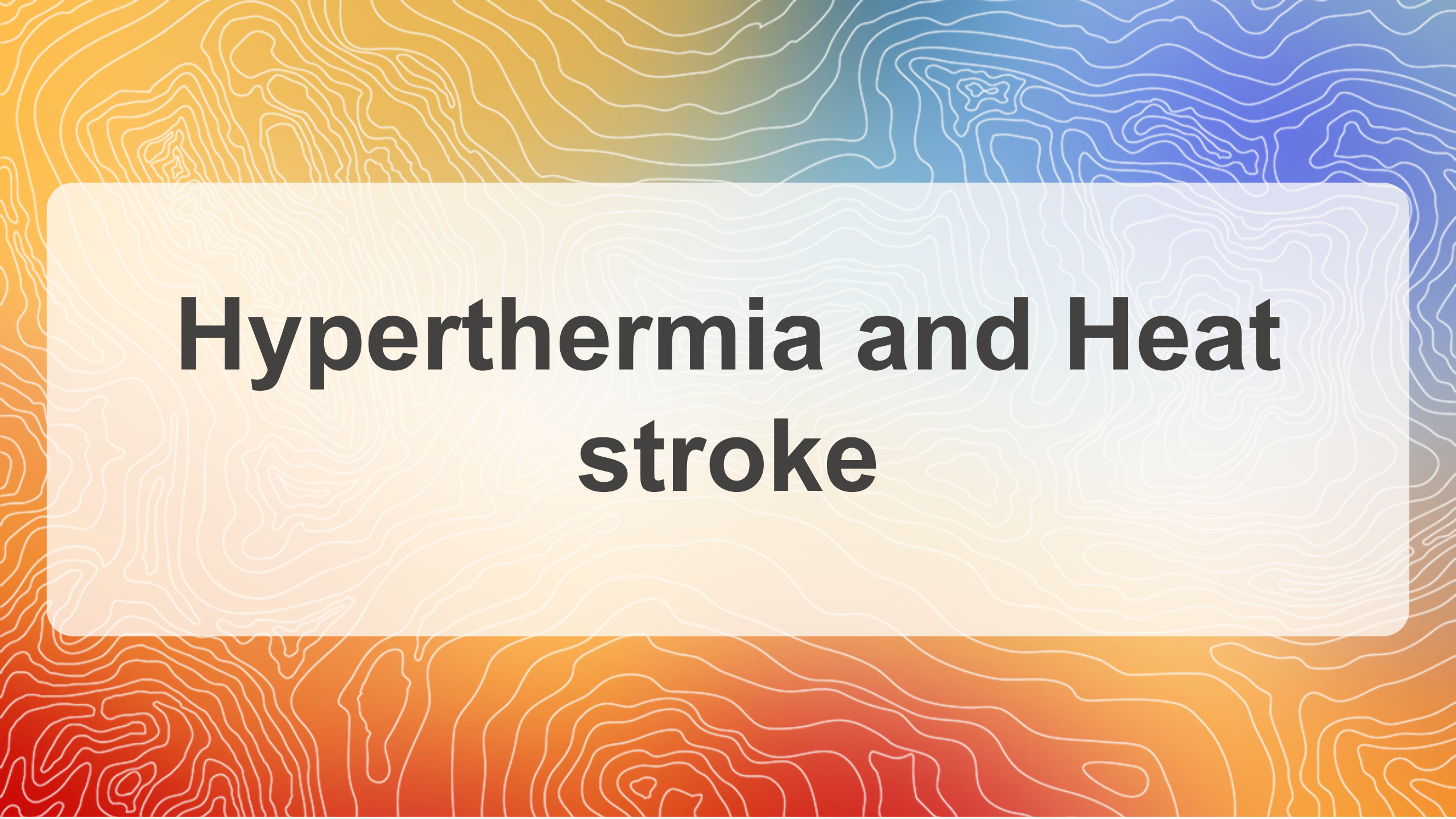
Never use these drugs in case of TCA toxicity:

Physostigmine / Beta, Na, K, Ca channels blockers

Also never use Flumazenil , when TCA is combined with benzodiazepine

ENVIRONMENTAL EMERGENCY

- 1-Hyperthermia and heat shock
- 2- Hypothermia
- 3- High altitude illness
- 4- Drowning

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Hyperthermia and Heat stroke

What is Hyperthermia?

- Hyperthermia is the term used to describe the failure of the body's thermoregulatory mechanisms (hypothalamus) to stabilize the temperature of the body leading to an elevation in the body's temperature. (unlike fever which is regulated)

What is Heat stroke?

- Is a life-threatening situation due to the failure of the normal heat shedding mechanisms of the body resulting in a body temperature greater than 40 degrees Celsius. This elevation in the temperature leads to many symptoms such as red skin, headaches hyperventilation and more dangerously altered mental status confusion and sometimes can result in end organ damage.

Types of heat strokes

- **Classical heat strokes:** which normally result from a setting of high ambient temperature (environment), often the patients presenting with this type are more likely to be old, immobile and leading a sedentary lifestyle
- **Exertional heat strokes:** which normally results from high endogenous heat production and overwhelmed homeostatic mechanisms, often the patients presenting with this type are more likely to be young and athletic

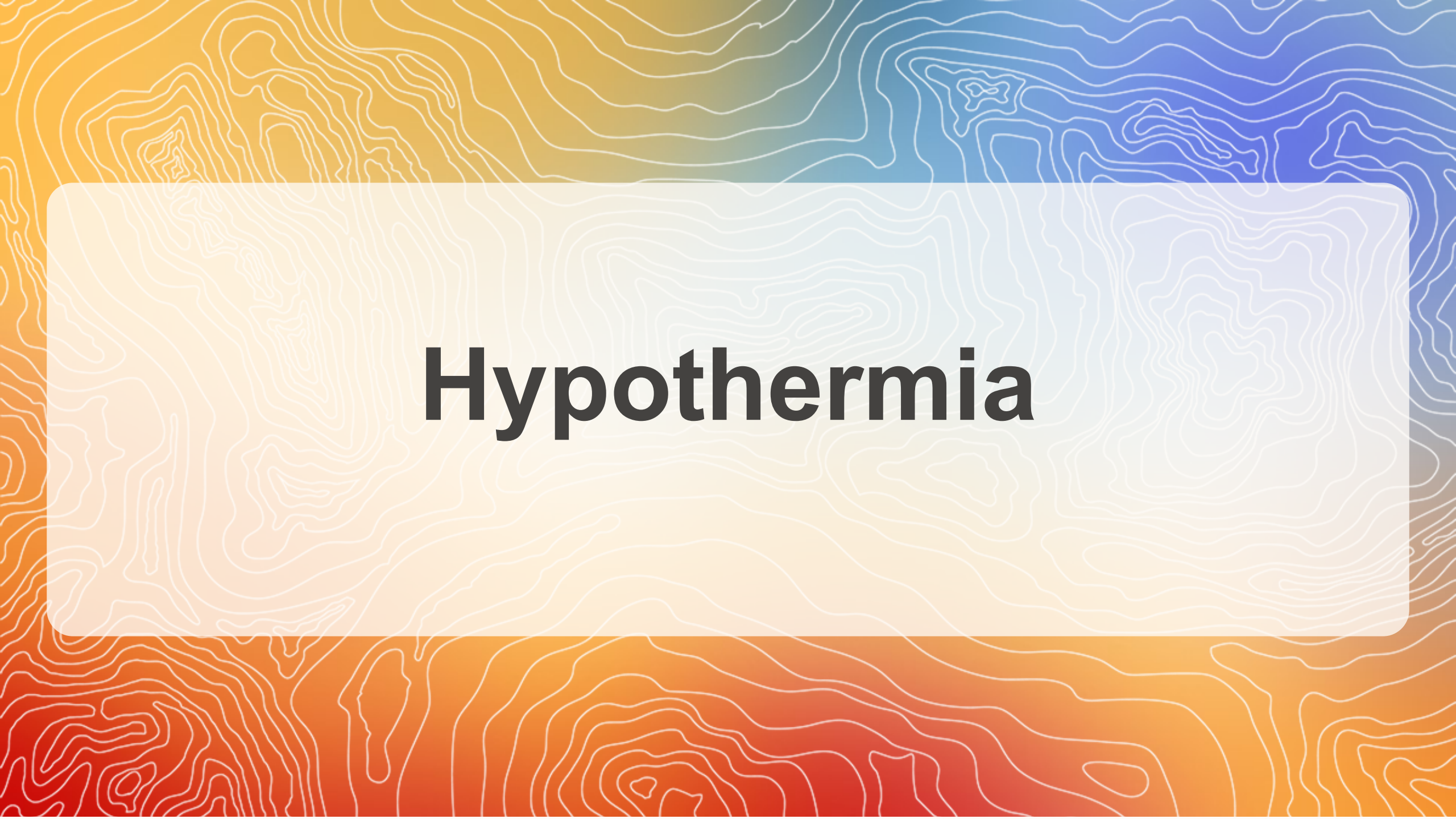
Treatment and Prevention

Prevention:

- Avoid long duration of exposure directly to the sun or other heat sources
- Always keep yourself hydrated
- Wear lightweight, light colored clothes especially while exercising
- Avoid drinking alcohol or caffeine.

Treatment:

- Evaporative cooling: Place fans near the completely disrobed patient and spray the patient with tepid water(avoid icy water due to the risk of hypothermia)
- Secure airway in case of seizures and aspiration
- Avoid alpha agonists ,peripheral vasoconstrictors and antipyretics

The background of the slide is a topographic map with white contour lines. The map is color-coded with a gradient: the top half transitions from yellow to blue, while the bottom half transitions from orange to red. A semi-transparent white rectangle is centered on the slide, containing the title text.

Hypothermia

What is Hypothermia?

- Hypothermia is a medical emergency in which there is a decrease in the core body temperature below 35 degree Celsius which occurs due to heat loss resulting from conduction, convection, radiation and evaporation.
- **The most common causes of hypothermia include:** cold water immersion, exposure to a cold environment
- **Complications include:** cardiac and respiratory complications such as coagulopathy, ventricular arrhythmias, asystole, volume and electrolyte depletion, pulmonary edema and pneumonia

- The symptoms of the patient will worsen depending on the degree of hypothermia but mainly they will present with shivering, slow shallow breathing, bradycardia(in later stages) lack of coordination, confusion and eventually loss of consciousness.
- **Risk factors include:** the extremes of age(old or very young), people with mental illnesses, hypothyroidism, alcohol and drug usage.

Treatment and Prevention

Prevention:

- Wear adequate clothing for cold environments
- Stay as dry as possible in cold weather
- Avoid drinking alcohol
- Avoid outdoor exercises

Treatment:

the main goal is to rewarm the patient back to regular temperature methods include placing the patient in a warm room, remove wet clothes, dry the patient and provide supportive care for any cardiac or respiratory complications.

The background of the slide is a topographic map with white contour lines. The map is color-coded with a gradient: the top half transitions from yellow to blue, while the bottom half transitions from orange to red. A semi-transparent white rectangle is centered on the slide, containing the title text.

High-Altitude Illness

Present.



Headache
Nausea
Anorexia

Oliguria
Dyspnea

Postural hypotension
Peripheral edema

Tx: History of rapid ascent.

Comp. Edema

Pulmonary [2nd -> 4th day]

- R.F. @ cold

② Pulmonary hypertension

③ child with ARDS

④ exertion

- **dx:** History, dry cough, ↓ exercise

- **Tx** - $\text{SaO}_2 \geq 90\%$
- immediate descent

or hyperbaric chamber

- sildenafil, tadalafil, Nifedipine if intubated monitor ABG - pCO_2 Not < 30

Cerebral

history + Neurologic manifestations

- $\text{SaO}_2 \geq 90\%$

- immediate descent
or hyperbaric chamber

- dexamethasone

- pCO₂ Not < 30

Mild

- Acetaminophen
- NSAID +
dexamethasone
4x 4mg

Moderate (ataxia, awareness)

- immediate descent/
hyperbaric chamber

- Acetazolamide till
resolved then dexamethasone

[Contra indicated in
sulfa drugs]

- incl.

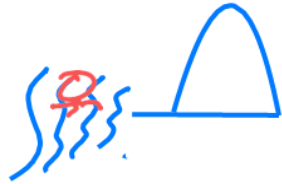
① previous Hx ② Ascent to 3000m

③ symptomatic periodic
breathing while asleep

The background of the image is a topographic map with white contour lines. The map is color-coded with a gradient: the top half is in shades of blue and teal, while the bottom half is in shades of orange and red. A semi-transparent white rectangular box with rounded corners is centered horizontally and vertically, containing the word "Drowning" in a bold, black, sans-serif font.

Drowning

{Drowning and Diving}



Patients who aspirate water into their lungs have washout of surfactant, resulting in diminished alveolar gas transfer, atelectasis, ventilation perfusion mismatch, and hypoxia. Noncardiogenic pulmonary edema results from moderate to severe aspiration.

⚠ Risk for hypothermia even in warm water



⚠ On decompression

⚠ clinically

- dx : dive history

- Gas filled space [contraction/expansion]

- Barotrauma → Middle ear squeeze [Most com. during descent. - due to inability to equalize pressure.]

→ inner ear trauma with forceful Valsalva occurs in oval or round window

Cerebral arterial gas embolism

Type 1

- Joint pain

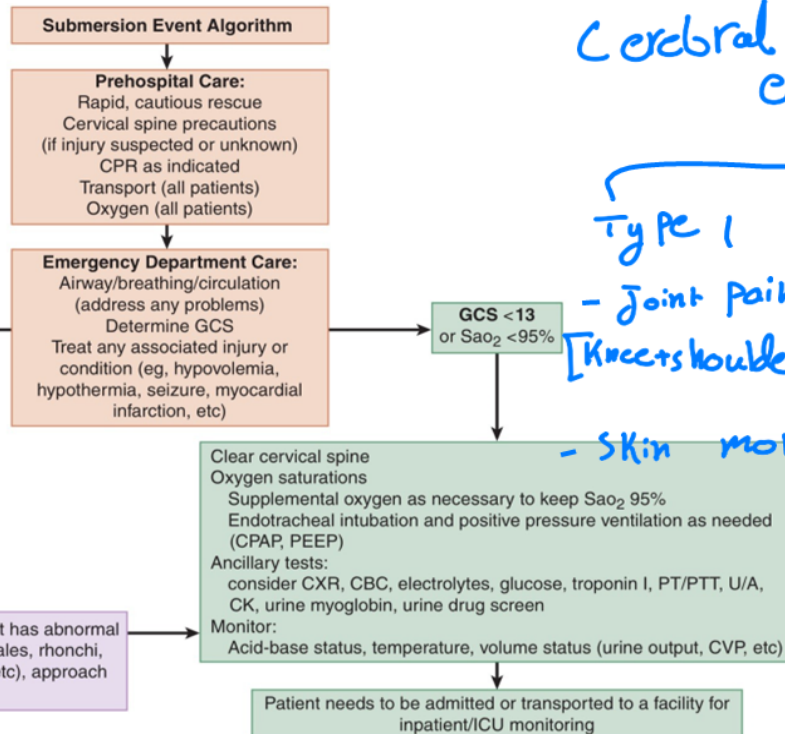
[Knees + shoulder ☹]

- Skin mottling

Type 2

Nervous sys. (spine)

Cardio pulmonary chokes/
Vestibular staggers



Thanks!

