تنظیم کننده: رستمی،حافظی زاده

درس: کنترل مسمویت

استاد: دکتر خدایار

جلسه: Comprehensive evaluation treatment

جلسه: ۱۶

هرشخصی که مسموم شده باشد جدای از اینکه با چه و چگونه مسموم شده ۴مورد زیر را بررسی میکنیم:

۱)Airway(راه هوایی فرد مسموم باز باشد)

۲)Breathing(عدم مشکل در حرکات تنفسی)

۳)Circulation(گردش خون برقرار باشد)

۴)D include | Dextrose

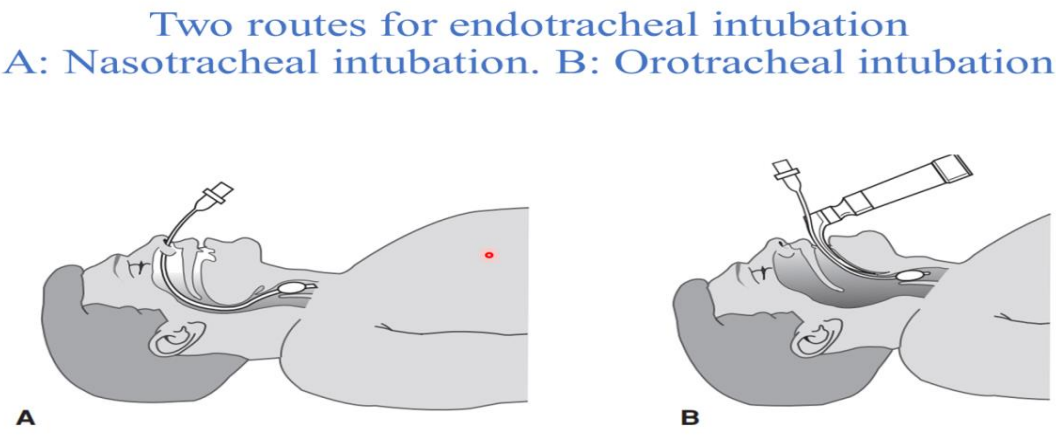
Don’t(coma cocktail)forget: Dextrose &Oxygen

Drugs(Antidotes)

Decontamination

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اولین قدم بررسی راه هوایی است ک باز باشد، درصورت بسته بودن از ۲مسیر میتوانیم فرد را انتوبه کنیم: ۱)مسیر بینی به نای ۲)مسیر دهان به نای



قدم دوم بررسی تنفس و نارسایی تنفس فرد مسموم است.

یکسری داروها و سموم سبب مشکل در تهویه هوای ریه میشوند و گاهی سبب فلج شدن عضلات تنفسی میشوند.

انواع فلج شدن عضلات تنفسی (parlysis)که سبب دپرشن تنفسی میشوند شامل: Flaccid(فلج شل) و Spatic فلج شل مثل سم بوتولینیوم و فلج اسپاتیک مثلNicotine،استریکینین و تتانوس

SELECTED DRUGS AND TOXINS CAUSING VENTILATORY FAILURE

Paralysis of ventilatory muscles	Depression of central respiratory drive
Botulinum toxin (botulism)	Antihistamines
Neuromuscular blockers	Barbiturates
Nicotine	Clonidine and other sympatholytic agents
Organophosphates and carbamates	Ethanol and alcohols
Saxitoxin ("red tide")	Gamma-hydroxybutyrate (GHB)
Snakebite (elapids and some vipers)	Opioids
Strychnine and tetanus (muscle rigidity)	Phenothiazines and other antipsychotics
Tetrodotoxin	Sedative-hypnotics
Warfare nerve gases	Tricyclic antidepressants

Adapted in part, with permission, from Olson KR, Pentel PR, Kelly MT. Physical assessment and differential diagnosis of the poisoned patient. *Med Toxicol.* 1987;2:52.

Paralysis: Flaccid vs Spastic

SELECTED CAUSES OF HYPOXIA

Inert gases	**Pneumonia or noncardiogenic pulmonary edema**
Carbon dioxide	Aspiration of gastric contents
Methane and propane	Aspiration of hydrocarbons
Nitrogen	Chlorine and other irritant gases
Cardiogenic pulmonary edema	Cocaine
Beta receptor antagonists	Ethchlorvynol (IV and oral)
Calcium channel blockers (eg, verapamil)	Ethylene glycol
Stimulant cardiomyopathy	Hydrogen sulfide
Quinidine, procainamide, and disopyramide	Mercury vapor
Tricyclic antidepressants	Metal fumes ("metal fumes fever")
Cellular hypoxia	Nitrogen dioxide
Carbon monoxide	Opioids
Cyanide	Paraquat
Hydrogen sulfide	Phosgene
Methemoglobinemia	Salicylates
Sulfhemoglobinemia	Sedative-hypnotic drugs
	Smoke inhalation

SELECTED DRUGS AND TOXINS CAUSING BRADYCARDIA OR ATRIOVENTRICULAR BLOCK

Cholinergic or vagotonic agents	**Sympatholytic agents**
Digitalis glycosides	Beta receptor antagonists
Organophosphates and carbamates	Clonidine
Physostigmine, neostigmine	Opioids
Membrane-depressant drugs	**Other**
Propranolol	Calcium antagonists
Encainide and flecainide	Carbamazepine
Quinidine and other Type Ia antidysrhythmics	Lithium
Tricyclic antidepressants	Phenylpropanolamine and other alpha-adrenergic agonists
	Propoxyphene

SELECTED DRUGS AND TOXINS CAUSING QRS INTERVAL PROLONGATION

Bupropion	Lamotrigine
Chloroquine and related agents	Phenothiazines (thioridazine)
Cocaine (high-dose)	Propoxyphene
Digitalis glycosides (complete heart block)	Propranolol
Diphenhydramine (high-dose)	Quinidine and other Type Ia antidysrhythmics
Encainide and flecainide	Tricyclic antidepressants
Hyperkalemia	Venlafaxine

SELECTED DRUGS AND TOXINS CAUSING TACHYCARDIA

Sympathomimetic agents	**Anticholinergic agents**
Amphetamines and derivatives	*Amanita muscaria* mushrooms
Caffeine	Antihistamines
Cocaine	Atropine and other anticholinergics
Ephedrine and pseudoephedrine	Phenothiazines
Phencyclidine (PCP)	Plants (many [p 375])
Theophylline	Tricyclic antidepressants
Agents causing cellular hypoxia	**Other**
Carbon monoxide	Ethanol or sedative-hypnotic drug withdrawal
Cyanide	Vasodilators (reflex tachycardia)
Hydrogen sulfide	Thyroid hormone
Oxidizing agents (methemoglobinemia)	

SELECTED CAUSES OF PUPIL SIZE CHANGES

TABLE I–19. SELECTED CAUSES OF PUPIL SIZE CHANGES[a]

CONSTRICTED PUPILS (MIOSIS)	**DILATED PUPILS (MYDRIASIS)**
Sympatholytic agents	**Sympathomimetic agents**
Clonidine	Amphetamines and derivatives
Opioids	Cocaine
Phenothiazines	Dopamine
Tetrahydrozoline and oxymetazoline	LSD (lysergic acid diethylamide)
Valproic acid	Monoamine oxidase inhibitors
Cholinergic agents	Nicotine[b]
Carbamate insecticides	**Anticholinergic agents**
Nicotine[b]	Antihistamines
Organophosphates	Atropine and other anticholinergics
Physostigmine	Carbamazepine
Pilocarpine	Glutethimide
Others	Tricyclic antidepressants
Heatstroke	**Retinal toxins (fixed, dilated pupils)**
Pontine infarct	Methanol
Subarachnoid hemorrhage	Quinine

SOME COMMON ODORS CAUSED BY TOXINS AND DRUGS

Odor	Drug or Toxin
Acetone	Acetone, isopropyl alcohol
Acrid or pearlike	Chloral hydrate, paraldehyde
Bitter almonds	Cyanide
Carrots	Cicutoxin (water hemlock)
Disinfectant	Phenol, pine oil-based cleaners, turpentine
Garlic	Arsenic (arsine), organophosphates, selenium, thallium
Hay (freshly mown)	Phosgene
Mothballs	Naphthalene, paradichlorobenzene, camphor
New Shower Curtains	Ethchlorvynol
Rotten eggs	Hydrogen sulfide, stibine, mercaptans, old sulfa drugs
Wintergreen	Methyl salicylate

SELECTED CAUSES OF ALTERATIONS IN SERUM GLUCOSE

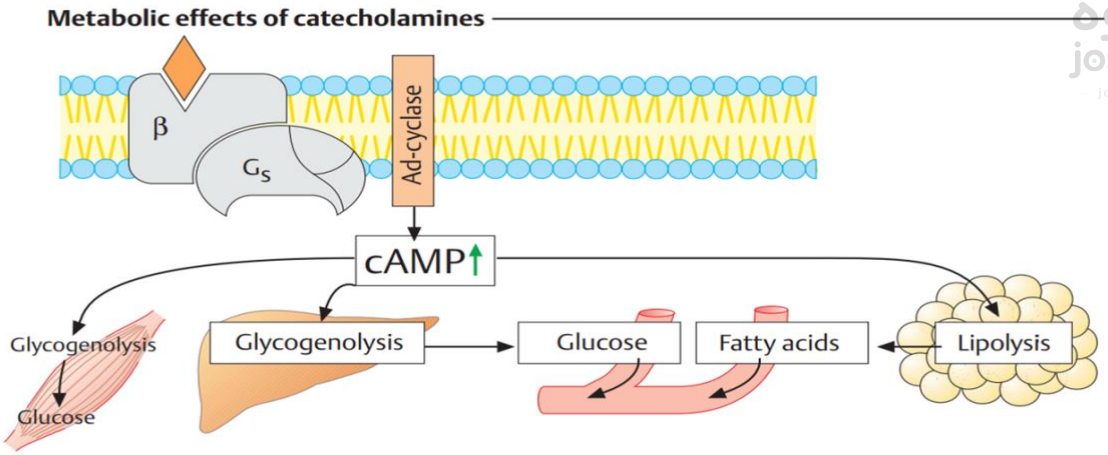
Hyperglycemia	Hypoglycemia
$Beta_2$-adrenergic receptor agonists	Ackee or lychee fruit (unripe)
Caffeine intoxication	Endocrine disorders (hypopituitarism, Addison disease, myxedema)
Corticosteroids	Ethanol intoxication (especially pediatric)
Dextrose administration	Fasting
Diabetes mellitus	Hepatic failure
Diazoxide	Insulin
Excessive circulating epinephrine	Oral sulfonylurea hypoglycemic agents
Glucagon	Pentamidine
Iron poisoning	Propranolol intoxication
Theophylline intoxication	Renal failure
Thiazide diuretics	Salicylate intoxication
Vacor	Streptozocin
	Valproic acid intoxication

کاتکول امین ها یاNEهنگام نشستن روی گیرنده بتا۲ سبب افزایشCAMP میشوند و از ۳طریق سبب افزایش انرژی میگردند:

۱)گلیکوژنولیز در عضلات ⟵ افزایش گلوکز در خون

۲)لیپولیز دربافت چربی ⟵ افزایش گلوکز در خون

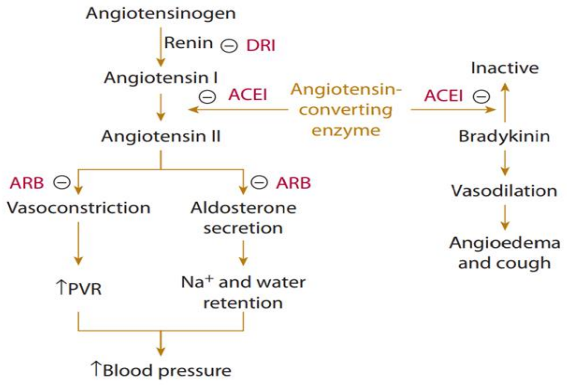
۳)گلیکوژنولیز در کبد ⟵ افزایش گلوکز در خون



SELECTED DRUGS AND TOXINS AND OTHER CAUSES OF ALTERED SERUM POTASSIUM

Hyperkalemia	Hypokalemia
Acidosis	Alkalosis
Adrenal insufficiency (chronic steroid use)	Barium
Angiotensin-converting enzyme (ACE) inhibitors	Beta-adrenergic drugs
Beta receptor antagonists	Caffeine
Digitalis glycosides	Cesium
Fluoride	Chloroquine
Lithium	Diuretics (chronic)
Potassium	Epinephrine
Renal failure	Hypomagnesemia
Rhabdomyolysis	Salicylate poisoning (with dehydration)
	Theophylline
	Toluene (chronic)

An overview of the normal function of the renin–angiotensin–aldosterone system (RAAS) and the mechanisms of action of angiotensin converting enzyme inhibitors (ACEIs), angiotensin II receptor blockers (ARBs), and direct renin inhibitors (DRIs) on that system. PVR = peripheral vascular resistance



در هایپرکالمی ،زمانی که برادیکاردی رخ دهد و موج QRS وP قابل مشاهده نباشد از درمان زیر استفاده میکنیم:

۱۰٪ **کلسیم کلرید** ۱۰-۵۰ میلی لیتر یا ۱۰٪**کلسیم گلوکونات** ۱۰-۲۰میلی لیتر

تجویز همزمان **گلوکز و انسولین** همچنین استفاده **از فراورده های استنشاقی اگونیست β۲ آدنرژیک ها مثل البوترول** سبب شیفت پتاسیم به درون سلول میشود.

همودیالیز سبب کاهش پتاسیم سرم میشود.

تجویز **آنتی بادی های حاوی دیگوکسین** درصورت بروز هایپرکالمی ناشی از فراورده های دارویی خاص مثل دیگوکسین میتواند موثر باشد.

رزین های خوراکی هم از طریق باند پروتیین به خود سبب درمان هایپرکالمی میشوند مثل **سدیم پلی استایرن سولفات**

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- Ventricular arrhythmia
- Hypotension
- Hypertension
- Hypothermai
- Hyperthermia

Important antidotes

Antidote	Poison(s)
Acetylcysteine	Acetaminophen; best given within 8–10 h of overdose
Atropine	Cholinesterase inhibitors, rapid-onset mushroom poisoning with muscarinic effects
Bicarbonate, sodium	Membrane-depressant cardiotoxic drugs (eg, quinidine, tricyclic antidepressants)
Calcium	Fluoride; calcium channel blockers
Deferoxamine	Iron salts
Digoxin antibodies	Digoxin and related cardiac glycosides
Esmolol	Caffeine, theophylline, sympathomimetics
Ethanol	Methanol, ethylene glycol (fomepizole is better tolerated)
Flumazenil	Benzodiazepines, zolpidem (note: flumazenil can trigger seizures)
Fomepizole	Methanol, ethylene glycol
Glucagon	Beta-adrenoceptor blockers
Glucose	Hypoglycemics
Hydroxocobalamin	Cyanide
Naloxone	Opioid analgesics
Oxygen	Carbon monoxide
Physostigmine	Suggested antidote for muscarinic receptor blockers when effect needed in CNS, NOT as antidote for tricyclics
Pralidoxime (2-PAM)	Organophosphate cholinesterase inhibitors. Most effective if used within 24 hours of exposure

Coma cocktail: DON'T forget

- Dextrose (D-Glucose)
- Oxygen
- Naloxone
- Thiamine

در شرایط کما مهمترین کار رساندن انرژی به بیمار است.

درمان هایپوگلایسمی:

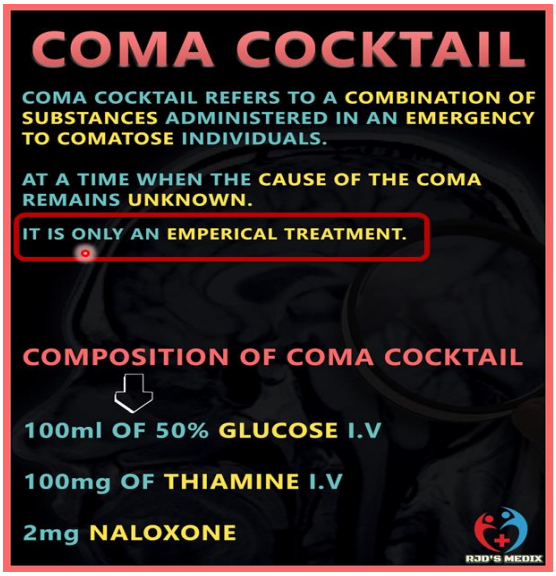
MEDICAL EDUCATION

"coma cocktail"

- The **"coma cocktail"** consists of :

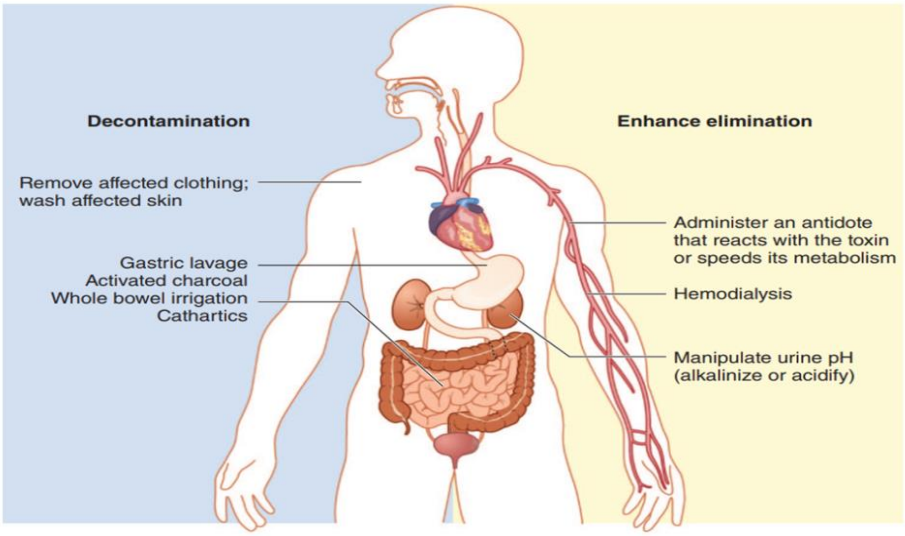
1) intravenous dextrose to treat hypoglycemia, a possible toxicological cause of altered mental status,
2) along with naloxone to treat possible opioid or *clonidine* toxicity,
3) and thiamine for ethanol-induced Wernicke encephalopathy.

- Note: Hypoglycemia may be caused by oral hypoglycemics, *insulin*, ackee plant, and ethanol.



The Coma Cocktail: Do DON'T, or don't DON'T?

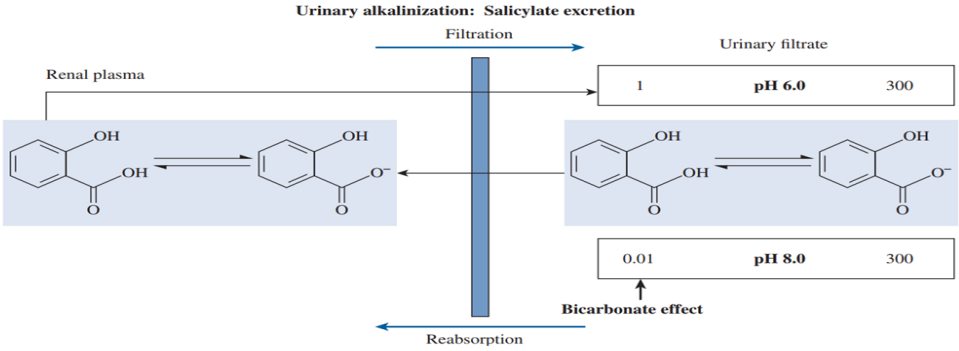
- **Dextrose**
  - Only if glucose < 4
  - 1 amp (25g) D50
- **Oxygen**
- **Narcan**
  - Reasonable if any clinical/historical suspicion of narcotic use
  - 0.4-2mg IV/SC
    - Start small, increase. Full dose in code.
  - Response? Ongoing boluses vs infusion (2/3 effective dose)
- **Thiamine**
  - If worried about nutritional deficiency
  - Q- Before or after glucose?
  - A- In the ED, who cares
- **No Flumazenil**
  - Risk of seizure induction



Acidification of urine: NH_4Cl, vitamin C, or cranberry juice

Alkalinization: sodium bicarbonate

Ion-trapping schematic for salicylate in the renal tubule



Anion gap

- The difference between the serum concentrations of the major cations (Na+/K+) and anions (HCO٣−/CI−); an increased anion gap indicates the presence of extra anions and is most commonly caused by metabolic acidosis.
- This gap is normally ١٢–١۶ mEq/L
- A significant increase can be produced by diabetic ketoacidosis, renal failure, or drug induced metabolic acidosis.

$$\text{Anion gap} = (Na^+ + K^+) - (HCO_3^- + Cl^-)$$

Osmolal gap

- The difference between the measured serum osmolality and the osmolality that is calculated from serum concentrations of sodium, glucose, and BUN; an increased osmolal gap is produced by high serum concentrations of intoxicants of low molecular weight such as ethanol, methanol, and ethylene glycol.

- This gap is normally zero

Osmolal Gap = [Measured Osmolality] – [Calculated Osmolality]

Normal Osmolality: 280 mosmol/kg

Calculated Osmolality = $2[Na] + ({}^{[glucose]}/_{18}) + ({}^{[BUN]}/_{2.8})$

Reasons for Increased Osmolal Gap:
- The presence of an additional solute or solutes (i.e. Ethanol, Methanol, or Ethylene Glycol
- The measured sodium concentration may be spuriously reduced (called pseudohyponatremia) with marked hyperlipidemia or hyperproteinemia.

The values ١٨, ٢.٨ convert mg/dL into mosmol/kg

Agents that increase the osmolal gap

ME DIE
- **M**ethanol
- **E**thylene glycol
- **D**iuretics (osmotic diuretics like mannitol)
- **I**sopropyl alcohol
- **E**thanol

* استاد مطالب هراسلاید را تیتروار فقط مرور کردند و نکته بیشتری بیان نکردند. پس اسلایدها را با دقت تر مطالعه کنید.☺