


# ReviewFile

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# THINGS TO REMEMBER

"This file is **not** intended for studying, it is only for revision and if you really don't have time to check the lecture"

## This file includes:

-All important points
-Golden notes
-Doctor notes

GOOD LUCK! May Allah grant you success

A+

# Cocaine & Sympathomimetics

## CNS Stimulation

1-Increase release of neurotransmitters

- Cocaine
- Amphetamines

2-Inhibit reuptake

- Cocaine
- Flakka

## Effects

All signs and symptoms are due to the stimulation of sympathetic system; different stimulants cause similar effects but could differ in duration and severity

Most important effects:
-Hypertension (stroke and aortic dissection)
-Hyperthermia
-Cardiac dysrhythmias
-Seziures

Anticholinergics cause similar effects but you can differentiate them by Diaphoresis!!

## Management

-Rapid sedation with an IV Benzodiazepines is the key for most symptoms from stimulants
-Hyperthermia is a high risk sign, should be assesed early and reduced rapidly; 41.1 C° is associated with high mortality.
-Beta blockers are CONTRAINDICATED in all stimulants*

## Cocaine

-Release of dopamine, epinephrine,norepinephrine, and serotonin
-Inhibit reuptake of these stimulatory neurotransmitters
-Block Na channels in myocardial cells (Class IA antiarrhythmic effect), Nerve endings (local anesthetic effect)

**Metabolism:**
1-Liver → norcocaine (potentiates cocaine)
2-Plasma → ecgonine methyl ester via pseudocholinesterase. (Protective; vasodilator)

**Complications:**
Hyperthermia
Hypertensive emergency
Cardiac dysrhythmias
Others such as Cocaine washout

-Urine drug screening (Benzoylecgonine); Useful in (Abuse, psychiatry, ECG)
-Cardiac markers (Troponin I, troponin T)
-Persistent headache despite management (CT scan if negative >> Lumbar puncture)

-Wide complex tachycardia caused by Na channels blockade are treated first by IV NaHCO3, If didn't work then use lidocaine after a dose of benzodiazepines (seizures)
-Hypertension (First drug is Phentolamine; Beta blocker is contraindicated)
-Hyperthermia (must be reduced within 20 mins)

**Cocaine related chest pain:**
-Caused by either cardiac causes (Endocarditis "IV abuse"), non-cardiac (pneumothorax).
-Chest radiograph is used to identify (aspirated foreign body, pneumothorax or pneumomediastinum "inhalational barotrauma")

Disposition:
1 -Discharged after acute intoxication resolves
2-Complications → ICU.
3 -Intoxicated with chest pain and cardiac manifestations → admitted .
4-After 12-hour observation: benign clinical course + negative markers → discharged.
5-Body packers observed until all packets pass

Differential diagnosis:
-Heatstroke
-Sedative-hypnotic withdrawal
-Amphetamine and its derivatives
-Infection (Hyperthermia)

## Amphetamine

-Enhance release of catecholamines
-Effects nearly identical to Cocaine
-Do not block sodium channels
-Same management as cocaine, but with longer duration of toxicity

## Methamphetamine (crank)

-Fat-soluble, smokable amphetamine
-Paranoid delusions (15 hrs)
-Longer duration of action
-There is no Na channel blockade so wide complex tachycardia.

## Methylenedioxyamphetamine (Ecstasy)

-Life-threatening hyponatremia
-Alter release of vasopressin, high urine sodium similar to SIADH
-Treated by fluid restriction,if Hypovolemic —> hypertonic saline for Neurologic impairment
-Irreversible damage to serotonergic neurons.

## Body packer

-Ingest cocaine wrapped in latex. → On continuous cardiac monitoring.
-Evidence of cocaine toxicity → rapid transportation to the operating room.
-Large quantities of drugs

## Body stuffer

-Conceal evidence of cocaine possession by swallowing .
-Small quantity of drugs.

## Speedballing

-Naloxone may reveal cocaine intoxication in Speedballing (using IV heroin and cocaine).

# Caustics

| Acids PH<7 | Alkalis PH>7 |
| --- | --- |
| Proton donors;<br><br>The commonest Hydrochloric acid (HCl)<br>Sulfuric acid (H2SO4) | Accept protons;<br><br>Alkalis caustics include:<br>-Lye (NaOH and KOH).<br>-Ammonia (NH3) |
| Coagulation necrosis -> then eschar formation | Liquefaction necrosis, fat saponification, and protein disruption |
| Strong odor , immediate pain on contact | Colorless, odorless, no immediate pain on contact. Soapy taste |
| Resistance of squamous epithelium to coagulation necrosis -> systemic absorption ( metabolic acidosis ) burns of stomach will be augmented by the acidity of gastric secretions | -Typically involve the squamous epithelial cells of the oropharynx and esophagus<br>-Gastric necrosis and perforation.<br>-Burns below the pylorus carry a worse prognosis |

## Diagnosis:

-Check Hb if internal bleeding is suspected
-Check liver and renal functions
-Litmus test if the caustic nature is not known
-Chest x-ray to roll out aspiration pneumonia
-Abdominal x-ray for free air that may have been caused by proferaton
-Patients with S&S (vomiting, drooling, stridor, or dyspnea) should undergo endoscopy within 12 to 24 hours to define the extent of burn.(contraindicated in perforation).
-Hypoxia warrant immediate bronchoscopy.

## Degree of burn:

-**First Degree:** hyperemia and edema
-**Second Degree:** superficial ulcers, white membrane, exudates, friability and hemorrhage.assets by endoscopy
2A: Non circumferential (15 to 30% develop strictures)
2B: Circumferential (upto 75% develop strictures)
-**Third Degree:** Transmural involvement with deep injury, necrotic mucosa, or frank perforation of the stomach or esophagus. (90% result in stricture)

## Clinical Features:

-Laryngeal edema occurs over a matter of minutes to hours.(the worst)
-Burns can occur from spills or contamination after vomiting.
-Hypovolemic shock
-Metabolic acidosis
-Chest pain is common.

## Management:

Most of the times especially in children caustics is ingested by accident, the manifestations is usually mild ( no visible or very superficial burns, patient is active and cooperative, no drooling), in this case the patient should be asked to drink some water:
-If the patient is able to swallow it and doesn't have dysphagia (remember it has high sensitivity and specificity for deep burns) try to neutralize the burns by some milk, observe for several hours then discharge the patient.
-If dysphagia is definite and deeper burns are suspected we should arrange for endoscopy to assess the extent and depth of burns.
-Endoscopy will not help only in management it will also help to know the long term prognosis of an injury BUT it has to be done by the early window period if it is available, if not we should wait few days before performing it.
-Do not neutralize the ingested corrosive with weak acids or alkalis due to thermal reactions and worsening injury.
-Early and continuous respiratory and hemodynamic monitoring is essential.

## Special Cases:

-Ocular alkali (Immediate and aggressive lavage with at least 2 L of normal saline per eye except frank profration) >> NEVER irrigate with ACID OR ALKALINE
-Hydrofluoric acid (Hypocalcemia and dysrhythmias), (ECG monitoring and Ca replacement)
-Povidone-iodine "Betadine" (Ingestion is trouble, gastric irrigation with starch or milk >> less toxic iodide)
-Concentrated H2O2 (Gas emboli >> HBO*)

## Common Caustic:

1-**Liquid drain cleaners**(high concentrations of alkali (30% KOH) or acid (93% H 2 SO 4) ).
2-**Industrial and farms (dairy pipeline) cleaners**(Containing liquid NaOH and KOH (in concentrations of 8–25%) ).
3-**Swimming pool cleaners**(Contain caustics in high concentrations.).

*Hyperbaric Oxygen

# Alcohol

| Alcohol | Metabolism | Clinical Presentation | Toxicity Workup | Treatment | Adjunct Therapy |
|---|---|---|---|---|---|
| Ethanol | -Ethanol will be metabolized into acetaldehyde by ADH -Acetaldehyde will get metabolized into Acetic acid by ALDH | -Disinhibited behavior -Slurred speech -Impaired coordination -Later: Respiratory and (CNS) depression | ABG/VBG -Renal profile/Lytes -Don't miss possible injuries (TRAUMA) | -Observation -IV Fluid Hydration -Thiamine -Discharge the patient once sober -ABC | - |
| Methanol<br><br>**Methanol containing products: -Gas Line Antifreeze 100% as fluid in car radiator -Windshield washer fluid 30% -Varnish removers** | -ADH will act on Methanol to convert it into Formaldehyde -The latter will be metabolized into Formic Acid | -Delayed onset (8-12 hrs) Methanol<br><br>-CNS depression, Ataxia, Confusion -Abdominal pain -Multisystem organ failure -Visual complaints: 1-Retinol damage 2-"Snow storm" -Anion gap acidosis -Tachypnea | -ABG/VBG -Osmolality -Serum levels -Lactate -Renal profile -Ethanol level | When do you treat ?<br>If :<br>1 serum level > 25 mg/dL*<br>2-Any serum level with Acidosis<br><br>Treatment :<br><br>-Limit absorption: NG Tube Suction.<br><br>-Prevent metabolism of parent compound to toxic metabolite: ADH Inhibition:<br><br>Ethanol<br>Fomepizole<br><br>-enhance elimination of Parent and its Metabolites & Correct Derangements: Hemodialysis | Sodium bicarbonate<br><br>Folate administration |
| Ethylene Glycol<br><br>**Ethylene Glycol containing products: Solvents Coolant\ Antifreeze** | metabolized into Glycolaldehyde by ADH. then will be metabolized to Glycolic acid by the action of ALDH and it will be converted into Glyoxylic acid by LDH. The formation of Glyoxylic acid will lead to the production of Oxalic acid -Glyoxylic acid itself will go through another metabolic reaction: 1-B6 actions will form Glycine+Benzoic acid 2-B1,Mg2+ actions will form **α**-OH-**β** Ketoadipic Acid | -Onset 4-6 hours -Low Volatility Ethylene Glycol -Low freezing point -High boiling point -Anion gap acidosis -Tachypnea -Abdominal pain -Hypocalcemia -Calcium oxalate crystals in urine -Renal failure (Calcium Oxalate) -It has sweet taste!! | -Limited utility of fluorescence of urine<br><br>-May note crystals in urine using Wood's lamp | | Thiamine (B1)<br><br>-Pyridoxine (B6) |
| Isopropranolol | Metabolized to : Acetone Osmol Gap: YES NO Acidosis Ketosis without Acidosis (characteristic) | -It has bitter taste!! | - | Treated by: Supportive care | |

# Aspirin

| | |
|---|---|
| Basic Knowledge | • Rapidly absorbed from GI tract within 30 minutes<br>• eliminated by renal excretion<br>• Therapeutic peak level within 2-4h , pathological peak within 18-24h<br>• At therapeutic dose, elimination follows first order kinetics (elimination rate depends on drug concentration)<br>• At toxic dose (>30 mg\dL), elimination follows zero order kinetics (excretion does not depend on drug concentration, but depends on the metabolic rate)<br>• It crosses BBB (blood-brain barrier) |
| Toxicity | • Toxic dose is 200-300mg/Kg<br>• Chronic toxicity associated with higher mortality<br>• Respiratory alkalosis FIRST , followed by metabolic acidosis with high anion gap<br>• Hypokalemia<br>• In children, primarily = metabolic acidosis and acidemia<br><br>**In Adults:**<br>1-Mild (less than 150mg/kg)->mild CNS symptoms: Tinnitus,dizziness and nausea<br>2-Moderate (150-300mg/kg)-> high vitals (fever & respiration) , ataxia and polyuria<br>3-Severe (more than 300mg/kg)-> organs failure, consciousness loss, seizure&arrhythmias |
| Diagnosis | Serum salicylate concentrations<br>(initially every 1 to 2 hours until level declined then every 4-6 hrs, don't use single level !) |
| Treatment | Signs of end organ damage or failure of alkalization or >100mg in acute ingestion or >40 in chronic-> HEMODIALYSIS<br><br>Other symptoms management ->• Treat dehydration<br>• Alkalinize urine with goal urine pH of 7.5 to 8.0<br>• Correct serum potassium to 4.5 mEq and IV 40 mEq potassium chloride (KCL)<br>• Consider activated charcoal if tolerated |

# Chemical & Radiation Exposure

## Asphyxiants

-When a gas replaces the oxygen, either by 1. Liquified gas 2. An Apparatus like divers 3. Working in confined place.
-The oxygen percentage in the room is 21% if it drops down we'll have symptoms. It causes symptoms by lowering FiO2
-At 15% > cardiac, autonomic, cerebral hypoxia (manifest by confusion, dizziness, incoordination, and seizure sometimes)
-Patient die from hypoxia not hypercarbia
-It's really important to understand that hypoxemia is the main principle of causing mortality
-If O2 sat falls below 10% patient will have cerebral edema
-If O2 sat falls below 6% patient will have immediate cardiac arrest
-The identification of the gas is not important because the treatment is supportive (Oxygen is the antidote!!)
-High risk patients (elderly, seizure, cardiac) are kept for observation
-Carbon dioxide is simple asphyxiant but it causes systemic toxicity
-Sign and symptoms Varies according to the levels of Fio2

## Pulmonary Irritants

You inhale them, they go to the lung, react and form new compounds e.g.
1.Chlorine: (used in swimming pools) if aspired in large amounts, it forms hydrochloric acid in lungs, which is very corrosive to the respiratory tract.
2. Ammonia: it forms ammonium hydroxide (base)
3.Sulfur dioxide: forms sulfurous acid which reacts and form acid or alkaline which are very irritating and cause a lot of damage
4.Phosgene: A gas used in chemical wars, very very injurious
Pulmonary irritants mechanism:
1. Dissolve in the mucus of bronchial tree 2. Induce very intense inflammation 3. Form new acid or alkaline 4. Form new oxygen radicals. All these mechanisms cause severe bronchospasm and bronchial inflammation
Signs and symptoms vary according to water solubility:
1-High water solubility (Affect upper airway, fast effect)
2-Intermediate (Upper and lower, takes hours)
3-Low (Affects lower airway more than upper >> high concentrations affect both. Delayed effect)
-If Standard management didn't work, we should give the patient Steroids

## Carbon Monoxide

Pathophysiology:
-Carboxy-Hb → decrease transport & release of Oxygen to the tissues since CO has higher affinity to Hb than O2 does.
-CO binds to cytochrome oxidase in mitochondria inhibiting O2 utilization and aerobic metabolism. Affect ATP production
-Lipid peroxidation, particularly in the hippocampus and corpus striatum (neurological sequelae!!)
Symptoms:
-CO is known to cause wide range of symptoms that are non-specific but headache is common. History is essential in diagnosing, specially history of camping and similar symptoms in people living in the same area.
Investigation: (COHb level; Most useful)
Management:
Removal from site of exposure, then:
-ABCD
-O2 (100% ASAP) Mainstay of treatment (antidote)
-Supportive care ( IV fluid for hypotension, standard ACLS PRN)
-Hyperbaric Oxygen (HBO)

## Cyanide

Pathophysiology:
-Cytochrome oxidase inhibition (inhibits other enzymes but this is the most important)

Symptoms:
-Most characteristic to CN toxicity:
Unconscious, hypotensive, and high lactate

Investigation: (History of smoke exposure is important)

Management:
-ABCDE
-Antidote (almost always empirical):
1-Hydroxocobalamin (5 g IV during 15 minutes for adults and 70 mg/kg IV for children, up to an adult dose). Cyanide + hydroxocobalamin = Cyanocobalamin
2-Cyanide kit (Amyl nitrite, Sodium nitrite, Sodium thiosulfate)
-Supportive care

## Other Chemicals

**-Anhydrous Ammonia** (Can freeze any tissue, liquefaction necrosis, water irrigation)
**-Phenol** (Coagulative necrosis, stimulation, seizures, irrigated with large volume of water)
**-Nitrate and Nitrites** (Symptoms depends on Methemoglobin conc "40-50% Acidosis, 70% death", Methylene blue!)
**-Elemental Metals** (harmless unless come in contact with water "exothermic reaction", Use mineral oil before water)
**-Nerve Agents** (Atropine,Pralidoxime) (**Most lethal is VX**)
**-Blistering Agents** (Hydrotherapy,Moist dressing on blisters,Supportive care)

Weapon of war
Management:
Removal from the scene, Supportive treatment (oxygenation), Observation until you reach the hospital
Signs of respiratory distress:
1. Cyanosis 2. Tachypnea 3. Tachycardia 4. Pulsus paradoxus 5. Using accessory muscles 6. CNS manifestations 7.Coma

## Radiation

-The early indicator for a significant radiation exposure is the absolute decreased lymphocyte count. Prognosis according to the Lymphocyte Count within the First 48 Hours after Acute Exposure; Lymphocyte count of 100 and below carries high incidence of death even with hematopoietic stimulation.
**Acute Radiation Syndrome**
1-Nausea, Vomiting, Diarrhea "NVD" (Occurs at 1-2 Gy, Symptomatic treatment)
2-Hematopoietic (Occurs at 2-6 Gy, Loss of cellularity in the hematopoietic organs, death occurs in 10-30 days if not treated, Stem cell therapy and bone marrow transplant)
3-Gastrointestinal (Occurs at 8-15 Gy, "damage to intestinal crypt cells, weight loss, low blood pressure, electrolytes imbalance", death happens in 3-5 days without treatment, "replacement of fluids and electrolytes, antiemetic and stem cell therapy")
4-Central Nervous System (Occurs at >25 Gy, irreversible neurological symptoms "epileptic type fits", death within 48 hrs, NO TREATMENT)
-Amount of radioactivity (Ci/Bq): The amount of radioactivity in a radionuclide.
-Amount of radiation emitted (Roentgens): When Gamma or X-rays ionize the air surrounding a source, an electrostatic charge is produced. (Radiation emitted during decay!)
-Amount absorbed by tissue (Rad/Gy): Only the fraction of particles that contacts and is absorbed by tissue can cause cellular damage.
-Degree of damage (Rem/Sv): To predict the degree of damage that a given particle will cause, the dose in Gy or Rad is multiplied by the particle-specific biological effectiveness coefficient (Q) to calculate rem or Sv.
-Alpha and Beta particles have weak penetration ability (alpha being the weakest) but if they were ingested then their effect is severe and lead to death. Lead is the best to protect from Gamma rays.
-Externally Contaminated patients are "radioactive"; irradiated patients are not.
-No danger to medical personnel from contaminated patients exists with proper precautions and decontamination procedures.
-Decontamination should not delay or impede the stabilization of the patient in radiation emergencies.
-Three principal risks to a fetus following radiation exposure: Congenital abnormalities, Mental retardation, Later Development of neoplasm. Risk is decreased at 16th week

# Heavy Metals

## Iron (Fe)

-Normal serum iron levels : 50 to 150 micg/dL. Serum iron level isn't a great test because it is bound.
-Ingestions of less than 20 mg/kg = no symptoms.
-Ingestion of 20 to 60 mg/kg = mild to moderate symptoms. Toxicity begins at level above 20 mg/kg
-Ingestion of more than 60 mg/kg = severe morbidity.
-50% mortality (LD50) is reported to be 200 to 250 mg/kg. 50% die no matter what you do
-A serum iron level: 3 to 5 hours (best) after ingestion, is the most useful laboratory test to evaluate the potential severity of an iron overdose.
-Pediatric toxicity more(susceptible)than adults and die in a lesser dose
Treatment:
-Whole-Bowel Irrigation(WBI): is routinely recommended, contraindicated in the presence of bowel obstruction, perforation, or ileus.
-Deferoxamine(Combined with WBI at the same time): -Pregnancy is not a contraindication to deferoxamine. The presence of ferrioxamine turns the urine a "vin ros?" Red wine color, which reflects the excretion of chelated iron
-Exchange transfusions have been recommended for severely symptomatic patients with serum iron levels exceeding 1000 micg/dL.
-Asymptomatic less than 20 mg/kg only observed, if remains asymptomatic after 6 hours of observation, discharge is recommended.

## Lead (Pb)

-Lead poisoning is a disease of industrialization.
-Its toxic effects are most **prominent** in:
1-Hematopoietic (ANEMIA "Normo or hypochromic", Basophilic stippling, Severity correlates with BLL!)
2-Neurologic (Peripheral neuropathies, wrist drop and foot drop in adults, Low IQ in children, Acute Lead Encephalopathy)
3-Renal systems (Lead nephropathy "Fanconi syndrome" is fibrosis of proximal tubules with sparing of glomeruli)
-The most informative biomarker is BLL (Blood lead levels), presence of lead in blood is abnormal, for a child level of 10 is toxic and causes permanent sequelae but for an adult carries nothing. A child dies at levels of 100 while an adult will develop Acute Lead Encephalopathy.
Treatment:
1-Whole bowel irrigation
2-Chelation therapy: (Must read it from lecture >_<)
A)Severe Poisoning "> 70 micg/dL or signs suggestive of encephalopathy" >> FIRST choice is Dimercaprol (avoid peanut allergic patients) followed by CaNa2EDTA "YOU SHOULD USE BOTH"
B) Less toxic "45-69 micg/dL without GI or CNS symptoms" >> DMSA if not well tolerated then use oral d-Penicillamine (Avoid penicillin allergic patients)
-No need for chelation for children with a BLL lower than 45 micg/dL.
-Acute Lead Encephalopathy: Standard measures to control cerebral edema, including intubation and neurosurgical consultation for invasive monitoring of ICP (intracranial pressure) are indicated.
-A BLL between 20 and 44 micg/dL in a patient who is asymptomatic or minimally symptomatic requires a more aggressive medical and environmental evaluation. (Screening)
-The treatment of adults with chronic poisoning is less aggressive than for children.

## Arsenic (As)

-Agent of homicide.
-Arsenic is still used for purposes in the treatment of trypanosomiasis, amebiasis, and leukemia.
-Of the two inorganic forms, trivalent arsenite (very toxic gas) (As3+ ) is highly lipid soluble is 5 to 10 times more toxic than the pentavalent arsenate (As5+ ) form.
-Arsine (AsH3 ), a colorless and almost odorless gas, is extremely toxic. It is immediately lethal at 250 ppm. Acute exposure to arsine gas is characterized by severe(massive) hemolysis that is associated with renal tubular injury.
-It disrupts oxidative phosphorylation by replacing phosphorus in the formation of phosphate bonds (arsenolysis).
-Within 30 to 60 minutes of exposure, patients complain of a metallic or garlicky taste.(While Cyanide causes bitter almond odor)
-Encephalopathy with seizures and coma, respiratory failure associated with ARDS and dysrhythmias associated with cardiac conduction disturbances. (Multi organ dysfunction)
-Less common complication includes unilateral facial nerve palsy
-Arsenic poisoning should also be considered in any patient with a history of severe or recurrent gastroenteritis or abdominal pain and unexplained dermatologic lesions associated with peripheral neuropathy (New undiagnosed rash; think Arsenic chronic toxicity)
-Weeks to Months later on : Characteristic lines in the nails (Mees' lines), sensorimotor neuropathy hyperkeratosis of the palms and soles (binds to sulfhydryl groups in keratin)
-(Gold standard) less than 50 micg/day in a (24-hour urine)
-**Management**:
1- supportive management of shock, dysrhythmias,and seizures.
2- Hemodialysis removes arsenic in the setting of acute renal failure.
3- Intramuscular dimercaprol(BAL) is the preferred chelator in patients who are critically ill, DMSA is a water-soluble analogue of dimercaprol that can be given orally.

## Mercury (Hg)

-The most familiar form of mercury is elemental or metallic mercury, also known as "quicksilver."
-Inorganic causes mad hatter's disease and organic
-**A common route**: inhalation of volatilized vapor. After inhalation, 74% of the metallic mercury is retained in the lungs. This can result in severe pneumonitis and ARDS.
-Aspiration of elemental mercury results in primary pulmonary toxicity, in addition to CNS and renal toxicities.
-Blood level more than 35 micg/L needs Rx. (not helpful)
-24-hour urine mercury levels is the most helpful test in confirming exposure and monitoring the effectiveness of chelation.

**Management:**
-Chelation Therapy:
1-BAL(succimer):(don't give to organic(found in fish) causes neuronal dysfunction) is used for clinically significant acute inorganic mercury intoxication.
2-DMSA: Used for both acute and chronic mercury poisoning and may be the best chelator for methylmercury.
3-d-Penicillamine: -It should be administered **only after thorough gastrointestinal decontamination** because mercury absorption from the intestinal lumen is **enhanced by the penicillamine**.
-Ingested metallic mercury is generally harmless unless its passage is impaired by entrapment in a diverticulum or the appendix.
-There is no role for prophylactic antibiotics or steroids.

# Acid-Base Disorders


**1**

First, we determine the change in pH:
pH <7.38 (Acidemia)
pH >7.42 (Alkalemia)

**2**

Now, Is it metabolic or respiratory?
Just look at pH and PCO2 and start to compare:
Both values are increasing/decreasing >> Metabolic
Both values are not the same >> Respiratory

**3**

Metabolic (We need to know if we are compensating or not!):
-Acidosis (PCO2 = [1.5 x HCO3]+8)
-Alkalosis (↑PCO2 = 0.6 x ↑HCO3)
If patient PCO2 and calculated PCO2 are the same then there is an appropriate compensation but if they are ar not then there is another disorder!

Respiratory (We need to know if it is acute or chronic!):
-Acidosis (↑PCO2 10, ↑HCO3 1 "Acute" 4 "Chronic")
-Alkalosis (↓PCO2 10, ↓HCO3 2 "Acute" 5 "Chronic")
If your patient is chronic (COPD or Smoker) and the calculated value was lower than chronic >> Acute on Chronic!

**4**

Anion Gap is your concern at this step it is either:
-Your patient is with primary disorder
-No primary disorder based on 1-3
To Calculate Anion Gap:
(Na-[HCO3+Cl]) , >12 means AGMA is present.

**5**

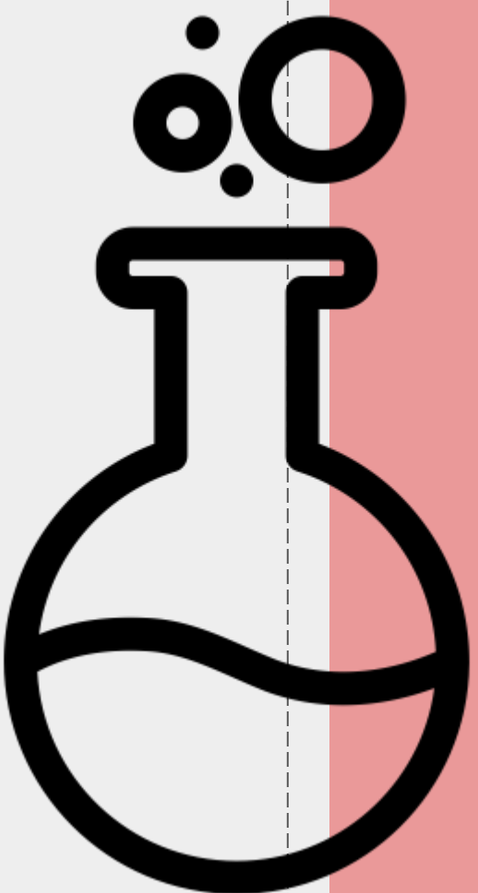
We only do this step if AGMA is present because we want to know if it associated with other disorder:
ΔGap = ΔAG-ΔHCO3 =(AG-12) - (24-HCO3)

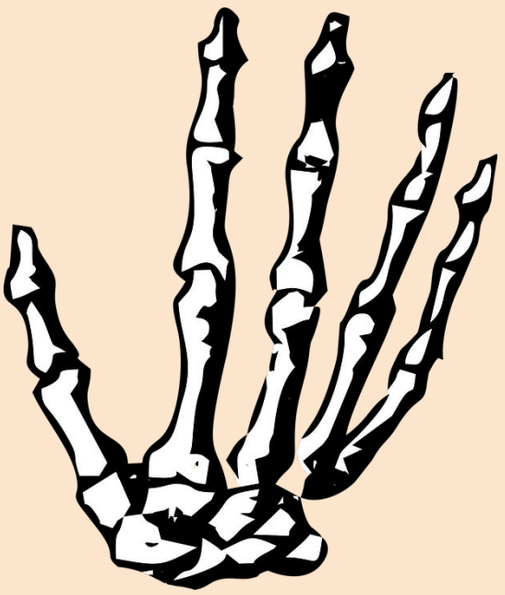
What to except?
-If Gap is >6 then AGMA + Metabolic Alkalosis
-If Gap is <-6 then AGMA + NAGMA
-If the value is between 6 and -6 then you are ONLY dealing with AGMA!!

Be aware!! Normal findings at these steps doesn't exclude an Acid-Base Disorder! Proceed for Step 4, if it is normal then your patient is safe to be discharged, otherwise, you should treat.

Low albumin must be dealt with differently, these patients may have a normal "low" AG so how to avoid misdiagnosis of AGMA in these pts? Simply, for every 1 g drop in albumin, the AG decreases by 2.5! For example, if the drop in albumin was 7 then multiply 7 into 2.5 = 17.5. Ok, the calculated value add it to their AG! Then judge.

# Thank you and good luck!





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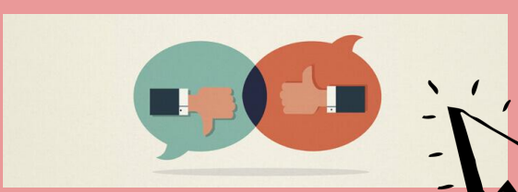

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