Alcohols

★ Objectives:

- Biochemistry
- Ethanol
- Methanol
- Ethylene Glycol
- Isopropanol

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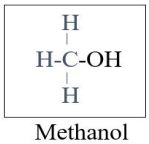
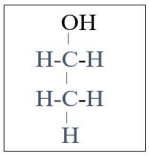
[Color index : Important | Notes | Extra | Editing file]

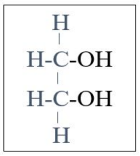
Alcohol

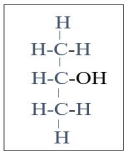
Alcohols Molecular structures: R-OH:

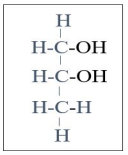
- What's the molecular structure of alcohols? Any additional OH group to hydrocarbon gonna be alcohol. So, we have many types of alcohol. For example, Methanol has(1C), Ethanol (2C), Ethylene glycol(2C &2OH group), Isopropanol (3C & 1OH), Propylene Glycol (3C & 2OH) and Benzyl Alcohol (1C & 1OH).. So mix anything with OH group you will have **ALCOHOL** at the end!

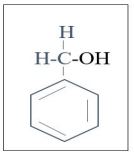

Methanol
1C


Ethanol
2C


Ethylene Glycol
2C


Isopropanol
3C

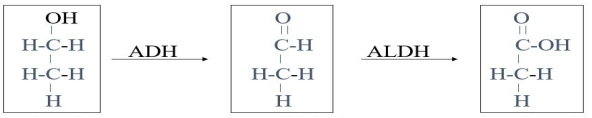

Propylene Glycol
3C


Benzyl Alcohol

1- Ethanol

Ethanol metabolism

- First when the **Ethanol** enters your body → converted into "**Acetaldehyde**" by "**ADH**" enzyme. Then, Finally into "**Acetic Acid**" by "**ALDH**" .. So, ALWAYS the 3rd compound is an **ACID**!!
- Just memorize the 3 compounds and the 2 enzymes in order.



Ethanol → Acetaldehyde → Acetic Acid

ADH = Alcohol Dehydrogenase
ALDH = Aldehyde Dehydrogenase

Ethanol toxicity:

- **Most commonly abused drug in the world**. "Including here in KSA"
- Majority of morbidity and mortality is due to **trauma** owing to impaired cognitive function. Because they get drunk and drive a car and then have an accident and die! or they walk and fall and have major injuries and bleeding in their brains. So, they don't have the complications b/c of the ethanol toxicity itself! it's b/c the **Trauma**! even some of them they fight in bar or something and end up with injuries.
- Rate of metabolism = **20 mg/dL/h.** Usually we're talking about up to 24hrs of the metabolism until the ethanol gets metabolized and leave the body completely.

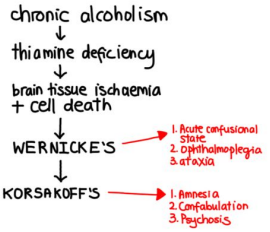
Which one will gets intoxicated from one can of beer? a young chronic drinker guy OR the older man who never drink a beer? People who drink a lot, one can or 2 they will have nothing b/c their liver rapidly metabolizes the ethanol. But someone who never drank alcohol for whole his life even one can of beer he will be intoxicated! This's called "**Tolerance**".. That's makes a difference when you dealing with ethanol toxicity patient in terms of assessing is he okay to go home or not? So, if the patient comes conscious in ER after drinking and he's known to be chronic drinker he will be okay after 2 hrs, but the other man he may stay for 1-2 days in the hospital!

So what are the other factors affect alcohol intoxication other than drinking habit?

Type of drink, the **percentage** of alcohol, **Food**. Is it on empty stomach or not?, The **weight** (Small child need small amount to be intoxicated), **Liver** disease and the **Gender**.
Blood ethanol levels correlate poorly with the degree of intoxication (Tolerance) = "The level may be high but the patient is walking, talking and okay, the level maybe low but the patient is completely unconscious!"

Clinical presentation

- **Disinhibited behavior**. The 1st thing to remember is this Sx, they feel euphoric and happy! but once they drink more the end up with ↓
- Slurred speech.
- Impaired coordination.
- **Later**: Respiratory and (CNS) depression. "Altered LOC"

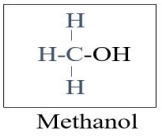
	- So, in terms of these symptomes, Which part of the brain gets affected most? **Cerebellum**!! - **Do they stop breathing?** The respiratory center affected but they usually don't stop breathing, only experience hypoventilation. They are at risk of **aspiration**!! - What intoxication lead to stop breathing? **Opioids**! they'll have respiratory arrest & stop breathing. but, with alcohol they have hypoventilation "B/c of affected respiratory center" shallow slow breathing only, no stopping! and become unconscious, vomit and aspirate it into their lungs and cause inflammatory processes > Pneumonia.
Ethanol toxicity work up:	• **Ethanol levels**. Very simple! just see how much ethanol in his blood. • **ABG/VBG** Why would you order it? To see the **Anion gap**, looking for **Acidosis**. Why would they get acidosis? **b/c of Acetic acid, ketone bodies & lactic acidosis.** WHY lactic? They **vomit** a lot after drinking → **dehydrated** (Low body volume) → **Hypoperfusion** → **Anaerobic metabolism** → **Lactate** goes up!**"Anion Gap/Metabolic Acidosis"** • Do they have ketone bodies? if they drink alcohol all the time without eating leads to **"Alcoholic Ketoacidosis"** • So, what after metabolic acidosis? **Hyperventilation**. And what Alcohol does to the brain? **Suppression**! Then, what will happen to their Hyperventilation? **Goes down!** And what will happen then to the CO2? **Goes up!!!** So, They will have both **"Respiratory & Metabolic Acidosis" = "Significantly Acidosis"** • **Renal profile/ElectroLytes.** • **Don't miss possible injuries (TRAUMA)** You have to examine him from head to toe!
Ethanol toxicity treatment:	• **ABC** "The most important thing" • Observation. • **IV Fluid Hydration** (Alcoholic Ketoacidosis) Usually we give IV b/c it's faster. "Ringer's lactate is better" • You can give Bicarb for acidosis, and you give them dextrose only if they're hypoglycemic. • **Thiamine**? Why? To prevent korsakoff & Wernicke syndromes. What are they? Wernicke is **ACUTE** confusional state. korsakoff is **CHRONIC**. What are the characteristics of korsakoff? **Confabulation =** يبهّر السوالف لأنه مايتذكر بالضبط وش صار يقوم يعبي فراغات مخه بتخريفات وتتغير القصة كل مرة. • Discharge the patient once sober. Usually stays up to **6-12** hrs under observation. 

2- Methanol

Definition:

What's it? the one we make from it "كولونيا"

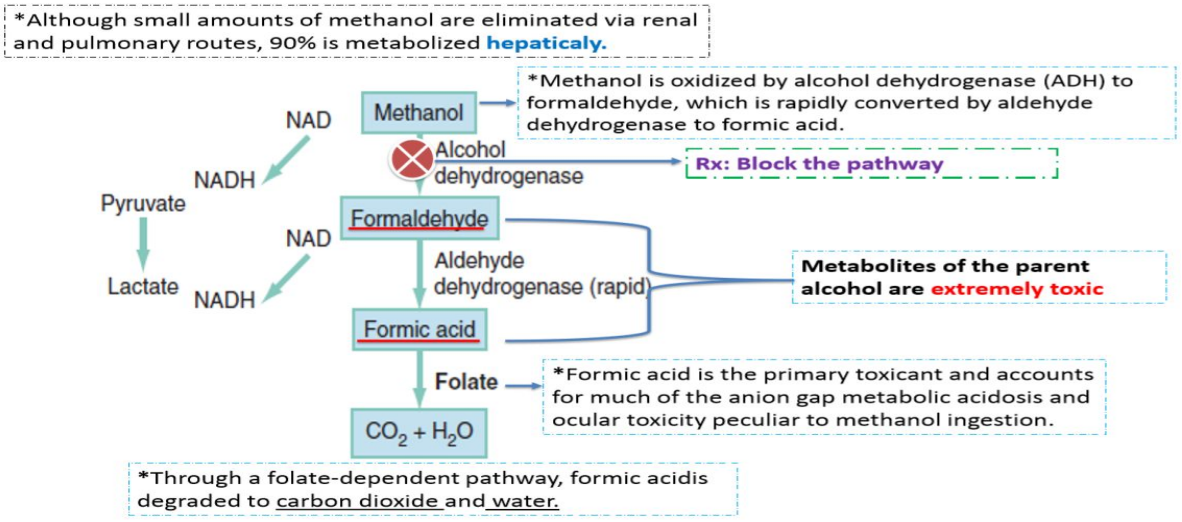
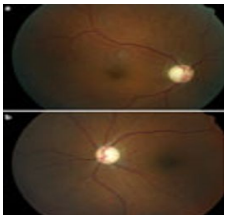
Methanol[1] is a colorless, volatile, slightly sweet-tasting alcohol.



Methanol

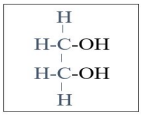
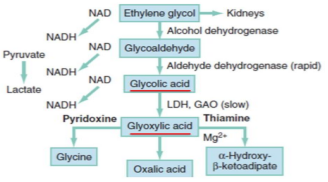
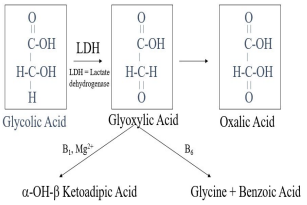
- Molecular weight 32.
- Low freezing point. الناس اللي عندهم شتاء أغلب الوقت يضيفونه لقطع السيارة عشان ما تتجمد بسرعة
- Highly volatility. People may get intoxicated because of just **smelling** it!!!
- It's absorbed in the alveoli and lung then to the body.

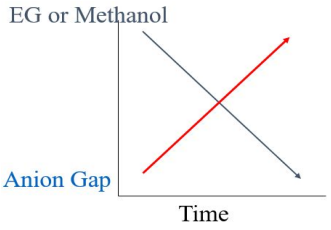
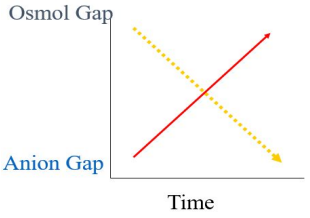
[1] Methanol acquired the name **wood alcohol** because it was once produced chiefly as a byproduct of the destructive distillation of wood.

Methanol containing products	• **Gas Line Antifreeze 100%** • Windshield washer fluid 30% • Varnish removers • Fuel for food warming 3-70% • Industrial uses. • Carburetor fluid; duplicator fluid; hobby engine fuel; gasohol; dry gas; sterno; glass cleaners; and thinners for shellacs, lacquers, adhesives, and inks may contain high concentrations of methanol. 
Methanol Pharmacology and metabolism:	- **Methanol** first metabolized into "**Formaldehyde**" by "**ADH**" and then into the final compound which is "**Formic acid**" by "**ALDH**" - Which one of them causes Blindness? The **Formic Acid.** B/c it accumulates in the retina and causes irreversible damage to the eyes.  • **Prolonged half-life (24 to 30 hours)** • Rapid absorption from the GIT, and blood levels peak 30 to 60 min after ingestion. "Gastric decontamination has a limited role and it's not useful" • **Inhalational** abuse of methanol is a recent trend that can result in toxic serum levels. • Certain **occupations**, including painting, glazing, varnishing, lithography, and printing, are at high risk for inhalational exposure to methanol. • **Transdermal** and, respiratory tract absorption also has resulted in toxicity, especially in **infants**.
Methanol toxicity and clinical presentation	- They have the same feelings like ethanol, euphoria and happiness. • Delayed onset (8-12 hrs) If they drink it they won't get the happy feeling within an hour, it takes 8-12 hrs to start work in the body. • **CNS depression**, Ataxia, Confusion. Also mainly **cerebellar** signs! • Abdominal pain. • Multisystem organ failure. B/c of severe toxicity!  1. **Visual complaints: "Unique about Methanol = Blindness"** Retinal metabolism. **"Snow storm"** Cloudy, blurred, indistinct, or misty vision or may note yellow spots or, rarely, photophobia. Pic: this's the vision when they get in the beginning. **"The earliest sign of eye blindness"** So, you have to fix that before this happens otherwise they may end up with irreversible blindness.  2. **Anion gap acidosis:** Why again? b/c Formic acid and the rest like ethanol, but ketones not as common here as Ethanol! Mostly is the **Formic & Lactic Acids.** a. **Tachypnea**. B/c of respiratory acidosis and depression. As a compensatory mechanism for acidosis.

Methanol work up:	• **Methanol level.** Keep in mind methanol level takes one to 2 days and comes back with the results. • Is there any indirect way to measure the methanol level? → **Osmolality** Which is normally fixed but if they have methanol in their blood it will be **increased** compared to the normal person. We know that the acidosis measures the **"Metabolites compounds not the parents"** so, early on the acidosis will be normal b/c the parents are there but no metabolites yet! but after 12 hrs you will see the metabolites and **acidosis goes up!** • **The onset of acidosis may be delayed 12 to 24 hours, the presence of a normal anion gap does not rule out methanol exposure.** • **Ethanol level.** Why? b/c sometimes they used to co-drink both. • ABG/VBG, Lactate, Renal profile.

3- Ethylene Glycol (EG)

Ethylene Glycol (EG)	• Molecular Weight 62 • Low Volatility. So, the opposite of methanol you don't get intoxicated b/c of inhalation only. • High boiling point. that's why we use it here in hot countries. • **Ethylene Glycol containing products:** - Most ethylene glycol poisonings occur with antifreeze. - Airplane deicing solutions, hydraulic brake fluids, and industrial solvents/precursors. - It is in certain paints, lacquers, and cosmetics.  Ethylene Glycol
Ethylene Glycol metabolism	- **EG** is first metabolite into "**Glycolaldehyde**" by "**ADH**" and then into "**Glycolic acid**" by "**ALDH**" - Then, the "**Glycolic acid**" will be metabolized into "**Glyoxylic acid**" by "**LDH**" and finally into "**Oxalic acid**" 
Ethylene Glycol toxicity	• **Onset 4-6 hours.** • **Anion gap acidosis:** B/c Oxalic acid. - Tachypnea. As compensatory response again. • Abdominal pain. • **Hypocalcemia**? B/c of the combination of Ca with Oxalic acid the Ca will be low in the blood. • **Calcium oxalate crystals in urine.** - What the other metabolites after Oxalic acid? What combines with OXalic acid? **Ca-Oxalate!!** the one we call it **Crystals** "Not Stones" >Accumulates in the renal tubules> Block it> End up with **Acute renal failure and Kidney injury!** - So, The EG toxicity is in the "**KIDNEY**"! - Keep in mind those Crystals **NOT** always there with EG toxicity! 40-50% of pts only have them. So, if you don't see the Ca-Oxalate you **cannot** Rule out EG toxicity!! • **Renal failure (Calcium Oxalate)** • How to avoid the Oxalic acid then? Here you need the **B1"Thiamine", Mg & B6"Pyridoxine"** to end up with other metabolites like **a-OH-b Ketoadipic Acid and Glycine + Benzoic Acid** respectively.  
Toxicity tests	**Ethylene glycol:** • Limited utility of fluorescence of urine. You have to do it!! For both EG And Methanol. • May note **crystals** in urine using Woods lamp. **Methanol:** Hyperemia **retina or visual** complaints.

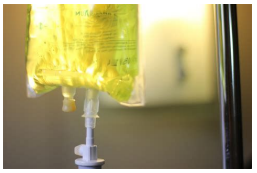
Identifying Patients for Treatment: Methanol/EG	• Serum ethylene glycol or methanol level. As we said it takes 2 days or more to have the results. • **Action level for treatment:** When you start Treatment? - Methanol / EG > 25 mg/dL. - Any level **with acidosis**. Even if it was low you have to treat immediately!! Why would u expect acidosis with low level? b/c Methanol may be already metabolized, and the only compounds left are metabolites. That means the Methanol decrease and the metabolites "Formic acid" increase with time! which is bad!! b/c the 1st metabolite not as toxic as the **Metabolites "The baddest part of toxicity".** **Limits of Serum Levels:** • Useful prior to onset of acidosis or in massive overdoses. • Parent compound not directly toxic. • Levels not universally available. - Graph: The EG or Methanol in the beginning will be high and the pH is normal! NO Acidosis. With time the parent compounds with go down and the Anion Gap b/c of the metabolites that has been left will go up! Causes Acidosis!!  **Arterial Blood Gas/Lactate:** • **Acidosis indicates advanced poisoning.** • Lactate usually **low**. Usually low at the beginning unless they're severely dehydrated. • Patients with acidosis should receive treatment. **ASAP!**
Osmol gap	- It is the difference between measured serum osmolality and calculated serum osmolality. ➢ **Osmol Gap = Measured serum osmolality-Calculated Osmoles.** ➢ **Measured:** **Done in the lab.** ➢ **Calculated: (2 Na + BUN + Glucose + 1.25 Ethanol)** As we said before the Ethanol will increase the osmolality in the blood so, you have to include it here. Do you remember? We said it's the **Indirect way** to measure the alcohol levels in the blood!! • Lab Measurement: Must use freezing point depression. Not imp.  **Limitation:** • Normal Osmol gap in between **(-14 to +10)or** Roughly the gap should be <10 • Normal Osmol Gap in setting of poisoning **does not rule out** a treatable level! • Osmol Gap diminishes as parent compound is metabolized.
Treatment:	❖ **Methanol and ethylene glycol ingestions are treated essentially the same.** ❖ Decontamination is not useful. **To keep it in categories:** 1. Limit absorption ⇒ **NG tube.** How? What's the decontamination here? Not Charcoal it won't bind to it. Just insert NG tube and suction the liquid, usually it take 30 min from the first ingestion until you get chance to suction something out. But they come too late > make the NG tube unuseful. 2. Prevent metabolism of parent compound to toxic metabolite ⇒ **ADH inhibition. Like Ethanol and Fomepizole (Antizol)** "Those are the true antidotes" 3. Enhance elimination of Parent and its Metabolites & Correct Derangements ⇒ **Hemodialysis**.

Adjuncts for Methanol/EG Poisoning:

1. **Sodium bicarbonate:** pH < 7.30 - As it Can trap formic acid in urine and enhance elimination. Instead of going to the brain and eyes it goes to the kidney.
2. **Folate administration:** Facilitates conversion of one carbon fragments to CO2.

Adjuncts for Ethylene Glycol Poisoning:

- **Thiamine (B1) & Pyridoxine (B6):** To enhance metabolism away from oxalates.
- **What's the "Banana bag" in th ER?** A **banana bag** (or **rally pack**) is a bag of IV fluids containing vitamins and minerals. The bags typically contain **thiamine**, folic acid, and magnesium sulfate, and are usually used to correct nutritional deficiencies or chemical imbalances in the human body. The solution has a yellow color, hence the term "banana bag"



Antidotal Therapy: Ethanol! Make them drink more? So why?

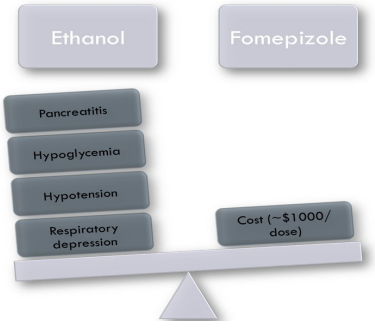
- Serum ethanol **inhibits metabolism of EG and Methanol**. Ethanol has high affinity to ADH compared to EG and Methanol. It has 6-8x more affinity than ethylene glycol and 4x more affinity than methanol.
- Onset of toxicity EG/Methanol may be **delayed**.

Ethanol Infusion: Management:

- How we give Ethanol? IV. and what are the problems if you give it IV? CNS depression and toxicity! So you have to monitor the patient.
- Serial ethanol levels.
- Watch glucose* and sodium*
- Observe for respiratory status*
- (*)Especially in **children**!

Fomepizole (Anotizle):

- A **blocker of alcohol dehydrogenase**.
- Has replaced ethanol as the agent of choice in known or suspected exposures.
- Minimal adverse effects.
- Fomepizole is a pregnancy category C drug.
- S/E: inflammation at the site of infusion, rash.
- **What's the down side of Fomepizole? COST!!**



Hemodialysis indications:

- Consult nephrology early in **acidemic** patients. **"Metabolic Acidosis"** PH <7.1
- Levels toxic alcohol **> 25 mg/dL**
- Renal compromise. Acute Renal failure in EG toxicity.
- Visual symptoms(methanol)
- Deterioration despite intensive supportive care.
- Electrolyte imbalances.
- Unresponsive to conventional therapy.

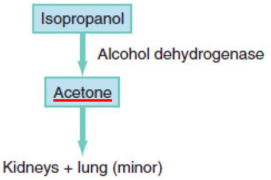
Summary:

For Methanol/EG Poisoning:

- Early Level, ABG, Lactate, **Ethanol level** "For every patient"
- Caution in using osmol gap.
- **Antidote: 1st line Fomepizole.**
- Hemodialysis.

Isopropanol

- Is not as bad as EG/Methanol.
- Is a clear, colorless liquid with a slightly bitter taste.
- It is the second most commonly ingested alcohol after ethanol.



➢ Metabolized to **acetone** > **CNS depression.** When do we usually use it? **Alcohol Swab! Is an Antiseptic.**

➢ **Acidosis: NO** B/c Acetone is not an Acid!

➢ **Osmol Gap: YES** B/c most of the alcohol will increase the osmolality in the blood.

➢ **The finding of ketosis without acidosis is characteristic of isopropyl ingestion.**

➢ Tx: Supportive care. "they rarely come to the ER"

433 Notes: "Just go through it!

- Two main complications of severe **methanol** poisoning: **Optic neuropathy & Putamen necrosis.**
- **The characteristic finding of bilateral putaminal lesions suggests methanol poisoning.**
- The primary sites of ocular injury are the retrolaminar **optic nerve and retina.**
- **Methanol** adversely affects other areas of the CNS, specifically the **basal ganglia.**
- Long-term morbidity takes the form of visual impairment, including **blindness**, and **parkinsonian** motor dysfunction, characterized by **hypokinesis** and **rigidity**.
- The typical 12- to 24-hour latency may be shorter when large amounts are consumed or longer when ethanol is co-ingested (range 40 min to 72 hr).
- In patients who present early, **formic acid accumulation may be ongoing,** with risk for significant toxicity despite being **asymptomatic**.
- Pallor and cupping, indicative of optic atrophy, are late findings suggesting a **poor prognosis** for visual recovery.
- Patients surviving the acute phase of toxicity may be left with **permanent** blindness or neurologic deficits.
- Anion gap is due primarily to the presence of **formic acid**, with a variable contribution from lactic acid.
- **CT** may be indicated in an intoxicated patient with altered mental status.
- **MRI** may also detect putaminal aberrations or optic neuropathy from methanol intoxication.
- Serum toxic alcohol level is **necessary** if **not** readily available, empirical treatment is warranted.
- Characteristically, **isopropanol** does not cause an increased anion gap.
- Majority of **isopropanol** are 70% metabolized in the liver and 30% excreted unchanged in urine.
- in **EG** they have **(acidotic breathing).**
- **Methanol may cause a "double gap" (i.e., an osmol gap in addition to the anion gap).**
- Methanol ,Ethylene glycol & Isopropyl alcohol: Very toxic.
- Ethanol : non-toxic (Long-term use causes damage)
- Alcohol intoxication :(Overdose) large amounts of ethanol can lead to Alcohol intoxication.

KEY CONCEPTS

- Small doses (single swallows) of methanol and ethylene glycol may cause toxicity.
- A latent period before the development of symptoms is characteristic for ethylene glycol and methanol toxicity, especially when ethanol has been co-ingested.
- A double-gap acidosis (anion gap and osmol gap) should suggest methanol or ethylene glycol toxicity.
- Toxic alcohol exposure cannot be ruled out by a "normal" osmol gap.
- Therapy should begin immediately based on clinical suspicion of exposure to ethylene glycol or methanol.

Acidosis should be corrected rapidly with bicarbonate, cofactors should be administered, and ADH should be blocked with ethanol or fomepizole.

- Because acidosis in the setting of exposure to either substance indicates toxic metabolite accumulation, immediate consultation for hemodialysis should be made, even before laboratory confirmation of toxic ethylene glycol or methanol levels.
- The presence of an osmol gap without acidosis is characteristic of isopropanol ingestion. Prolonged coma may be seen, and hypotension portends a poor prognosis.

MCQs

Q1: Hypothermia may be seen as a complication of which of the following:

A. Cocaine
B. Amphetamine
C. Ethanol
D. Iron

Q2: A 45 years old female, who is known to have ESRD on hemodialysis missed her last cession of dialysis two days ago. she presented to the emergency department with SOB. Her investigation areas follows: Serum Na=135, Serum Cl=90, Serum HCO3=17. Blood gas shows: Ph=7.27, CO2=32, which one of the following is the calculated anion gap in her blood result?

A. 10
B. 14
C. 20
D. 28

Q3: A 25-year-old man presents to the ED with nausea and abdominal pain after drinking some "bitter liquid" at his friend's house. His BP is 130/70 mmHg, HR is90b/m, RR is 18 breaths per minute, temperature 37°C, and oxygen saturation is 98% on room air. Physical examination is unremarkable, except for slurred speech and smell of acetone on the patient breath. Laboratory results reveal serum sodium 138 mmol/L, potassium 3.5mmol/L,chloride 105 mmol/L, bicarbonate 23 mmol/L, glucose 5 mmol/L, arterial blood pH 7.37, and lactate 1.5 mmol/L. Urinalysis shows moderate ketones. Which one of the following is the most likely diagnosis?

A. Ethanol poisoning
B. Methanol poisoning
C. Isopropyl alcohol poisoning
D. Ethylene glycol poisoning

Q4: A 40-year-old man is brought to the ED by the ambulance who state that the man is an engineer and was found lying on the floor of an aircraft hangar. He is drowsy, speaks with slurred speech, and is vomiting. His BP is 140/85 mmHg, HR is 94 bpm, and temperature is 36.80°C, RR is 18 per minute. Laboratory results reveal serum sodium 139 mmol/L, potassium 3.5 mmol/L, chloride 101 mmol/L, bicarbonate 14 mmol/L, glucose 5.5 mmol/L, arterial blood pH 7.27 and lactate 2 mmol/L. Urinalysis shows multiple calcium oxalate crystals. Which one of the following would best explain the metabolic state of this man?

A. Ethylene glycol poisoning
B. Diabetic ketoacidosis
C. Lactic acidosis
D. Isopropyl alcohol poisoning

Q5: The hemodialysis is useful for the removal of which of the following poisons?

A. Methanol
B. Caustics
C. Cyanide
D. Organophosphorus

Q6: A 29-year-old male is brought to the emergency department after methanol ingestion. Which of the following treatments are proven to be beneficial in the management?

A. Activated charcoal
B. Sodium bicarbonate
C. Flumazenil
D. Plasmapheresis

Q7: An ambulance brings a 37-year-old man to the emergency department with altered mental state. His BP is 130/80, HR 93 bpm, temperature is 36.8°C, RR is 18, and oxygen saturation is 99% on room air. Physical examination reveals a strong odor of alcohol on his breath. Laboratory result reveals high anion gap metabolic acidosis and high osmolar gap. Which one of the following is the antidote for this poisoning?

A. Flumazenil
B. Physostigmine
C. Desferrioxamine
D. Fomepizole

Q8: Which ONE of the following is the antidote for Methanol?

A. n-acetylcysteine
B. Ethanol/fomepizole
C. Oxygen/hyperbarics
D. Naloxone/nalmefene

Q9: Which one of the following is a consequence of vitamin and mineral deficiencies that may lead to dementia and memory disorder in alcohol abuse?

A. Seminoff's syndrome
B. Korsakoff's syndrome
C. Hemert syndrome
D. Huynh-feldt syndrome

Q10: which one of the following can cause high anion gap metabolic acidosis?

A. Methanol
B. Mannitol
C. Isopropanol

Q11: what is the treatment of choice for alcoholic toxicity?

A. Ethanol
B. Sodium bicarbonate
C. Aspirin
D. Hydroxocobalamin

Q12: a 30 years old male presents with methyl alcohol poisoning. There is CNS depression, cardiac depression and optic nerve atrophy. What is the cause?

A. Formaldehyde and formic acid.
B. Acetaldehyde
C. Pyridine
D. Acetic acid

Q13: Calcium oxalate is found in toxication of what type of alcohol intoxication?

A. Ethanol
B. Ethylene glycol
C. Methanol
D. Cyanide

Q14: 4 years old boy drink a windshield washer fluid and after few hours he came drowsy and comatose. What did he drink?

A. Ethanol
B. Iron
C. Methanol
D. TCA

Q15: Which of the following is considered a non-toxic alcohol?

A. Ethylene glycol
B. Ethanol
C. Methanol
D. Isopropyl alcohol

Q16: A33 years old man is brought to the ED with altered mental status ,flank pain, the man works in an airport and has an access to ethylene glycol, toxicity with ethylene glycol is suspected , how would you approach this patient "first step" ?

A. ECG
B. Urinalysis
C. ABC
D. Hemodialysis

Q17: The difference between (methanol/ethylene glycol) and (isopropanol) intoxication is the:

A. Presence of CNS symptoms.
B. Metabolic alkalosis.
C. Ketosis without acidosis
D. Increased osmolar gap.

Answers | 1:C - 2:D - 3:C - 4:A - 5:A - 6:B - 7:D - 8:B - 9:B - 10:A - 11:A - 12:A - 13:B - 14:C - 15:B - 16:C - 17:C