



Toxic Alcohols

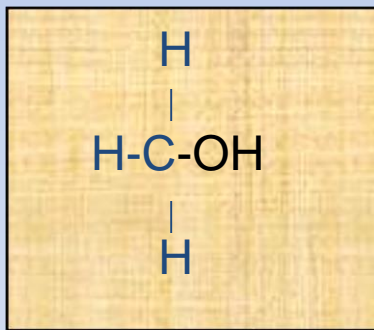
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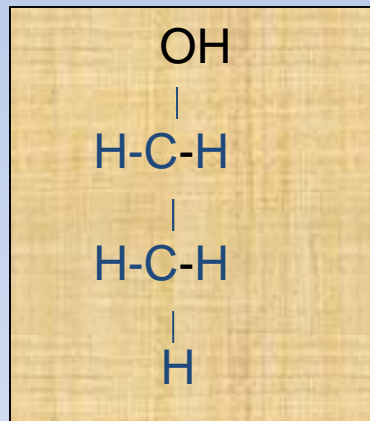
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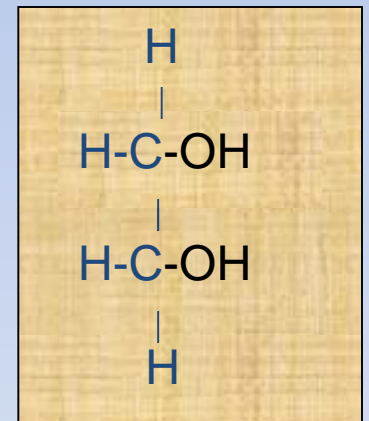
Alcohols: R-OH



Methanol
1C

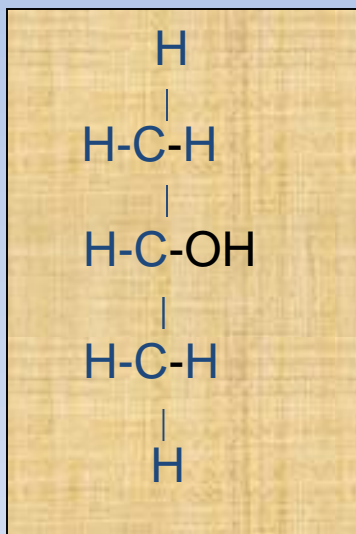


Ethanol
2C

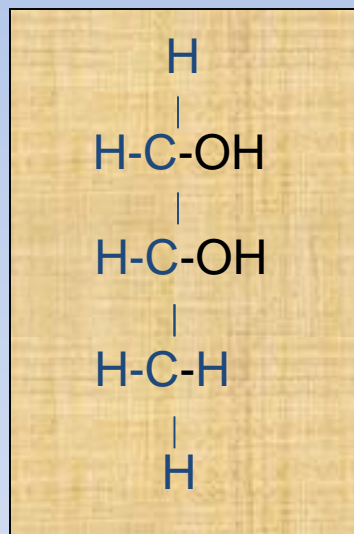


Ethylene Glycol
2C

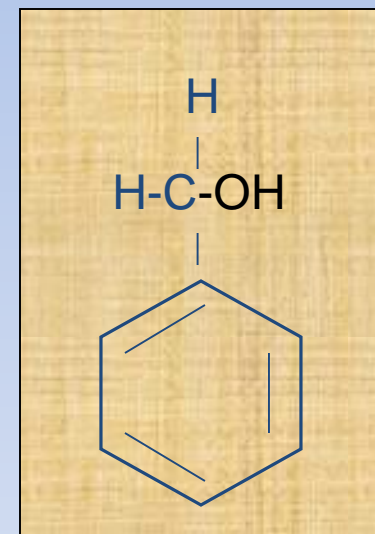
Alcohols: R-OH



Isopropanol
3C

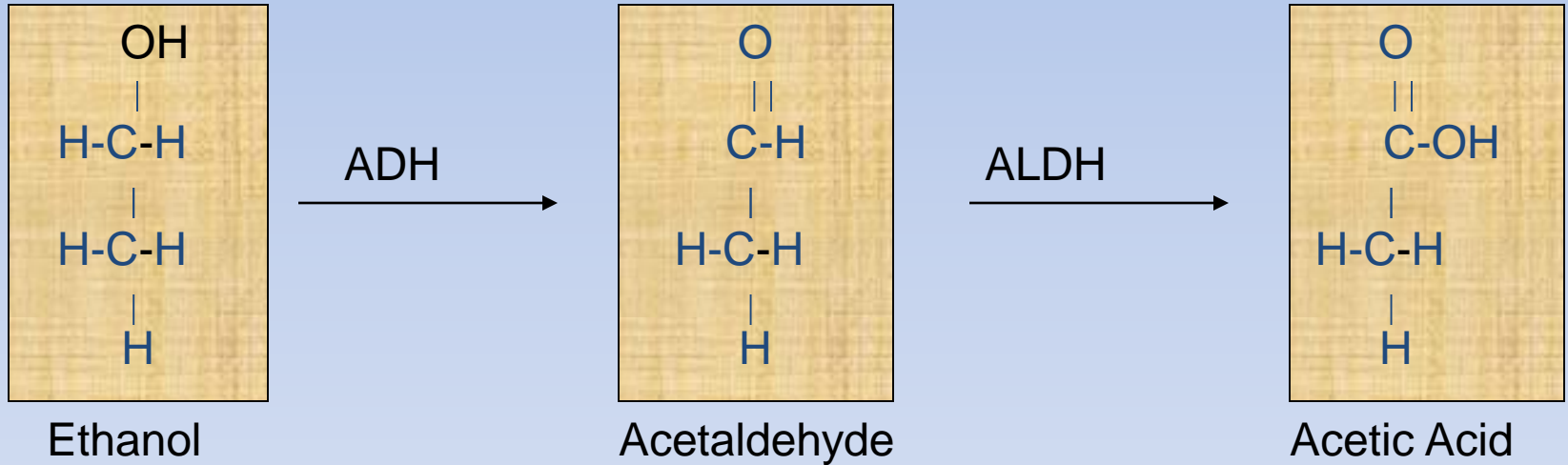


Propylene Glycol
3C



Benzyl Alcohol

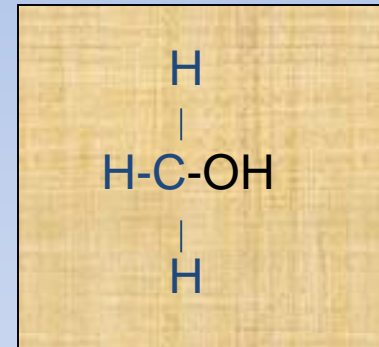
Ethanol



ADH = Alcohol Dehydrogenase
ALDH = Aldehyde Dehydrogenase

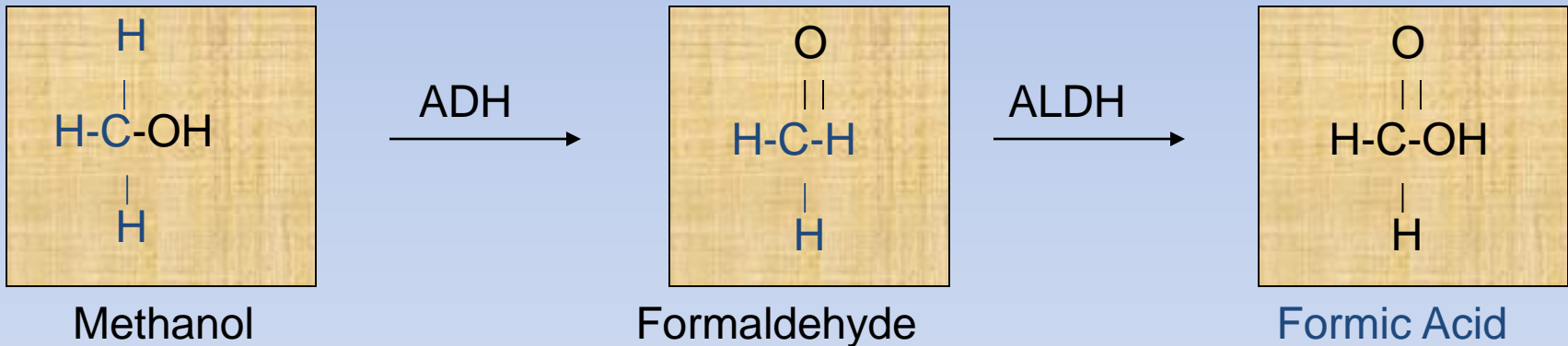
Methanol

- Molecular weight 32
- Low freezing point
- Highly volatility



Methanol

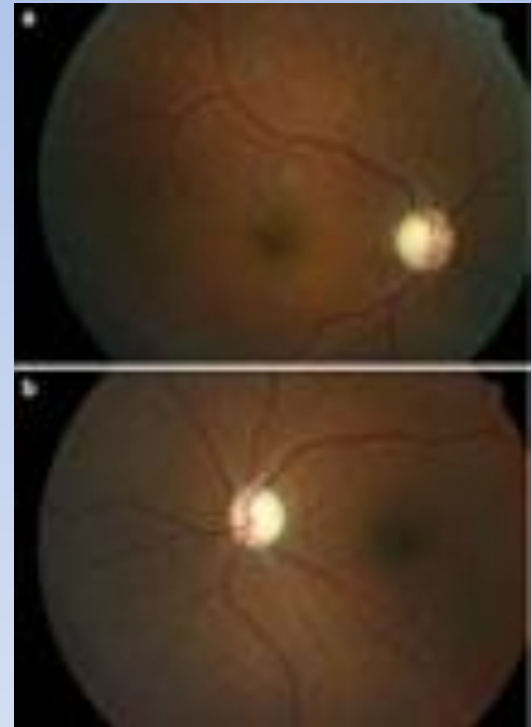
Methanol Metabolism



ADH: Alcohol Dehydrogenase
ALDH: Aldehyde Dehydrogenase

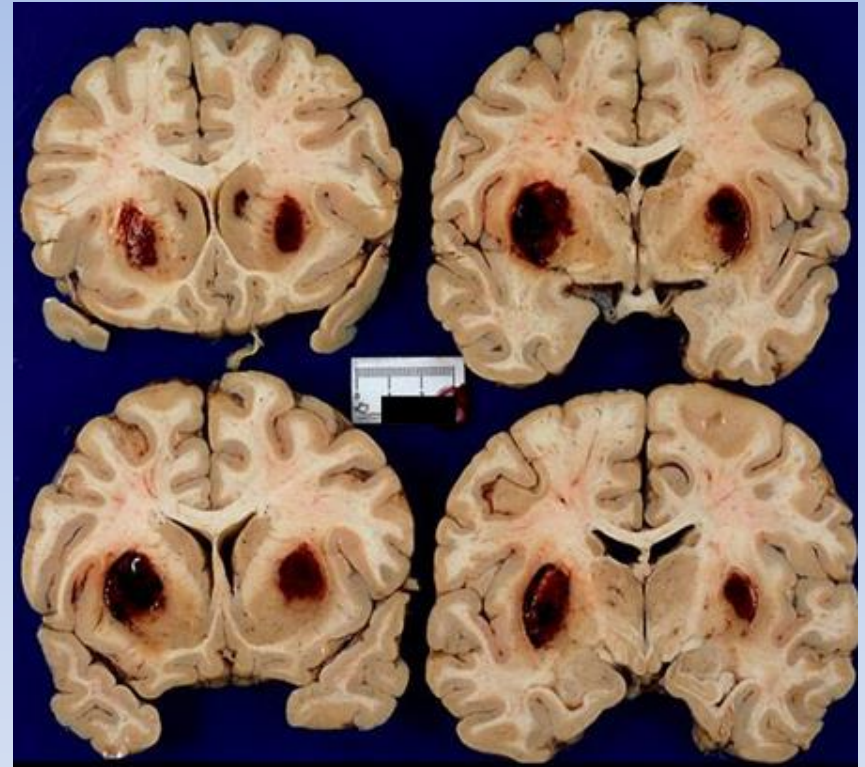
Methanol Toxicity

- Delayed onset (8-12hrs)
- Anion gap acidosis
 - Tachypnea
 - Visual complaints
 - Retinal metabolism
 - “Snow storm”



Methanol Toxicity

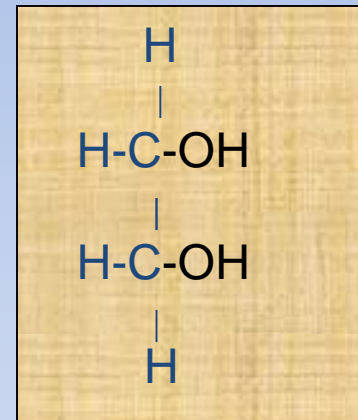
- CNS depression
 - Bilateral hemorrhage putamen
- Abdominal pain
- Multisystem organ failure



University of Western Ontario:
Neurology Collection

Ethylene Glycol

- Molecular Weight 62
- Low Volatility
- High boiling point



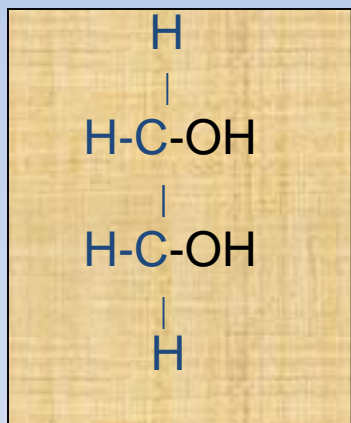
Ethylene Glycol

Ethylene Glycol

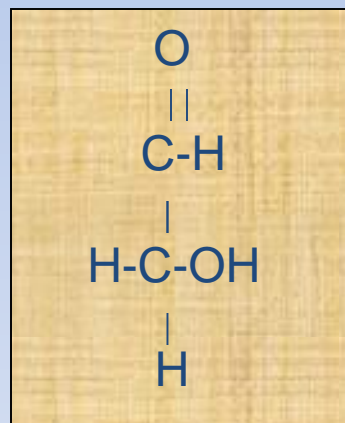
- Coolant/Antifreeze



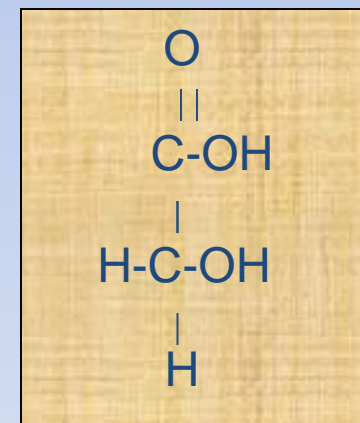
Ethylene Glycol Metabolism



Ethylene Glycol



Glycoaldehyde

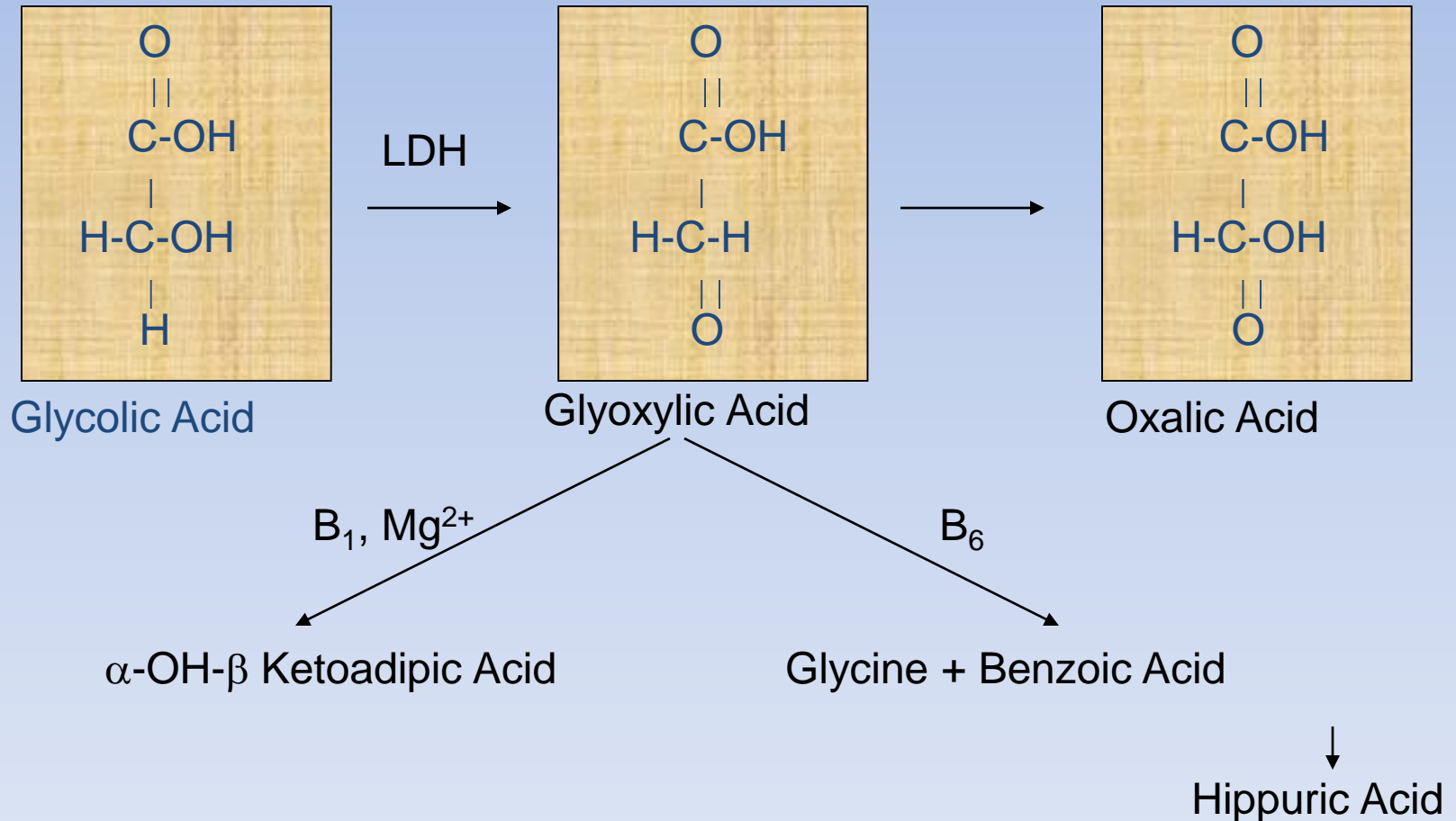


Glycolic Acid

ADH = Alcohol dehydrogenase

ALDH = Aldehyde dehydrogenase

Ethylene Glycol Metabolism



LDH = Lactate dehydrogenase

Ethylene Glycol Toxicity

- Onset 4-6 hours
- Anion gap acidosis
- Tachypnea

Ethylene Glycol Toxicity

- Abdominal pain
- Hypocalcemia
- Calcium oxalate crystals in urine
- Renal failure



Clinical Manifestations and Pathophysiology

Central Nervous System Effects

All alcohols may cause inebriation,
depending on the dose
isopropanol > ethylene glycol >
ethanol > methanol

Metabolic Acidosis

Metabolic acidosis with an elevated anion gap is a hallmark of toxic alcohol poisoning

Specific End-Organ Effects

Methanol causes visual impairment ranging from blurry or hazy vision or defects in color vision, to snowfield vision or total blindness in severe poisoning

The formate metabolite of methanol is a mitochondrial toxin, inhibiting cytochrome oxidase (much like cyanide) and thereby interfering with oxidative phosphorylation

The most prominent end-organ effect of ethylene glycol is nephrotoxicity hypocalcemia by precipitation with calcium. This can result in prolongation of the QTc interval

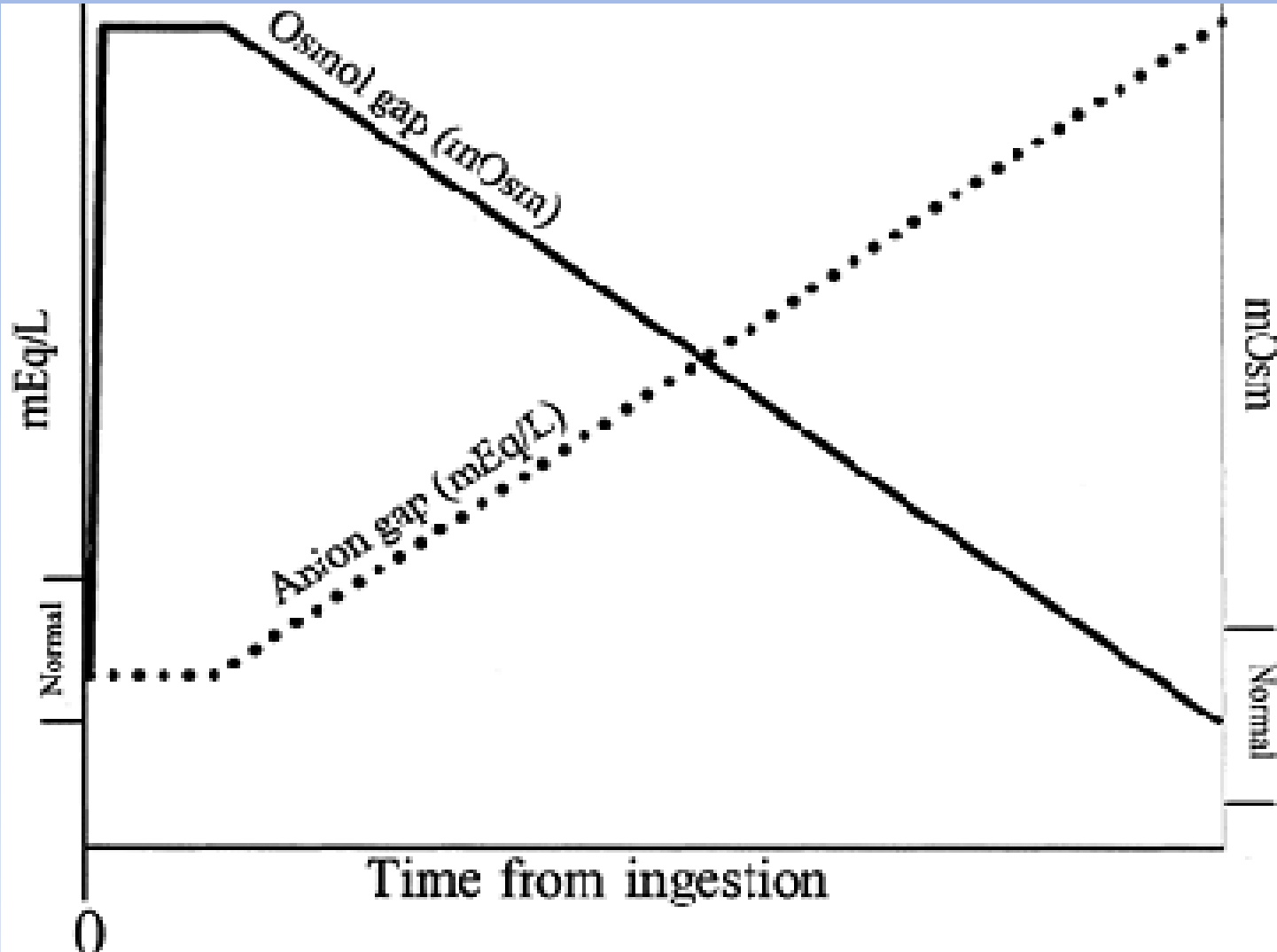
Diagnostic Testing

Toxic Alcohol Concentrations

methanol or ethylene glycol level
greater than 25 mg/dL has
been considered toxic

Anion Gap and Osmol Gap

hallmark of toxic alcohol poisoning
osmol gap -14 to +10,
patients who present early after
ingestion may have a high osmol gap
and normal
anion gap, whereas those who present
later may have the reverse



Other Diagnostics

Calcium oxalate monohydrate (spindle-shaped) and dihydrate (envelope-shaped) crystals



Treatment



Management

Alcohol Dehydrogenase Inhibition

affinity for ethanol that is 15 times greater in vitro than its affinity for methanol, and 67 times greater than its affinity for ethylene glycol on a molar basis

Ethanol Infusion: Management

- Serial ethanol levels
- Watch glucose and sodium
- Observe for respiratory status



Ethanol

0.8 g/kg of ethanol is given orally

over 20 minutes

100-154 mg/kg/h

hemodialysis (250-350 mg/kg/h)

Fomepizole

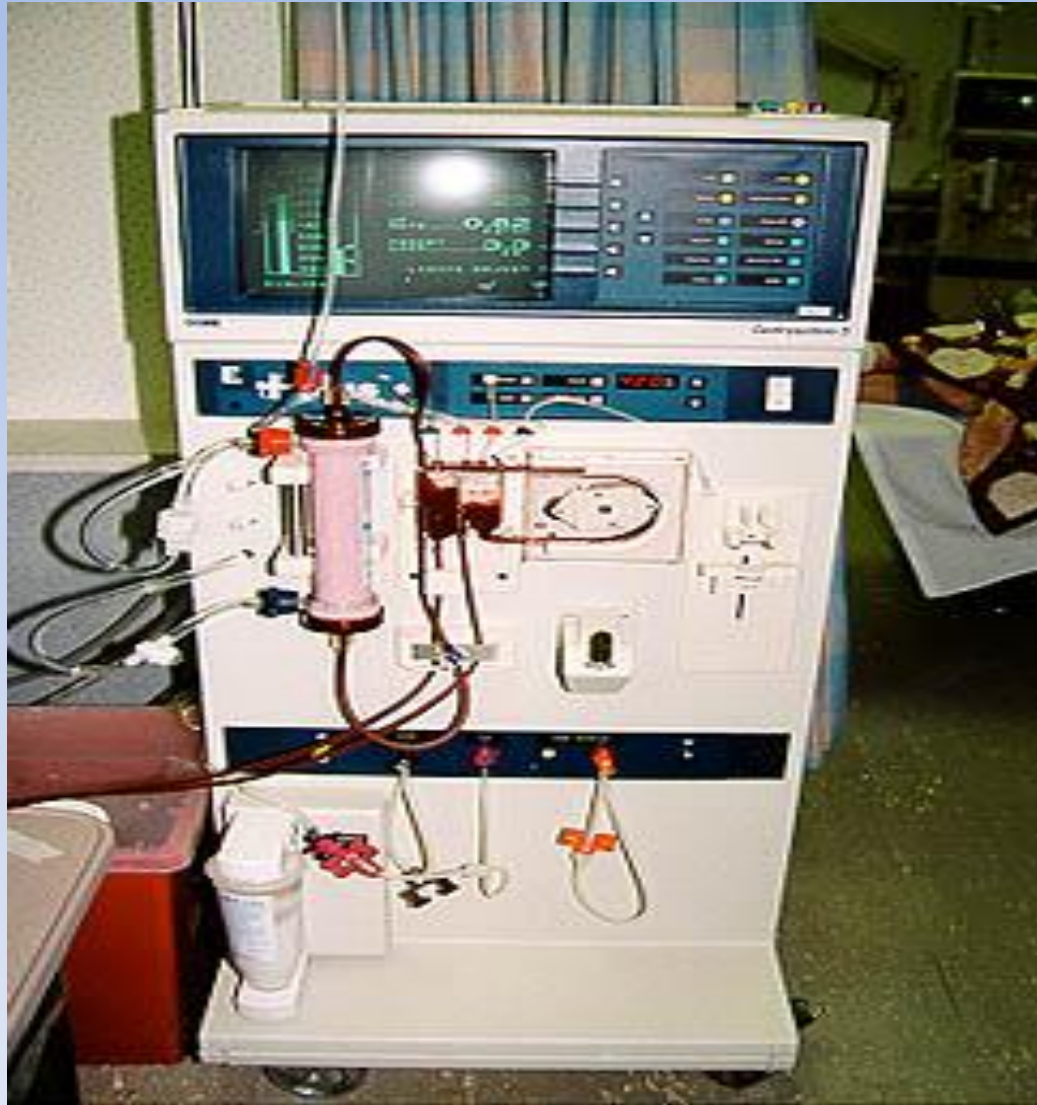
- A blocker of alcohol dehydrogenase
- Has replaced ethanol as the agent of choice in known or suspected exposures
- Minimal adverse effects

Fomepizole

The dose of fomepizole is 15 mg/kg intravenously as an initial loading dose followed by 10 mg/kg every 12 hours.

After 48 hours of therapy, fomepizole induces its own metabolism, so the dose must be increased to 15 mg/kg every 12 hours

Hemodialysis



Adjunctive Therapy

Folate and

leucovorin enhance the clearance of
formate

Thiamine enhances the
metabolism of ethylene glycol to
ketoadipate, and pyridoxine enhances
its metabolism to
glycine

Leucovorin (Folinic Acid) and Folic Acid

1-2 mg/kg every 4-6 hours

100 mg/d of pyridoxine IV

200 mg of daily intravenous
thiamine

Thanks for attention

