Toxicology ITE Review

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Toxicology Review

- Acetaminophen
- Salicylate
- Alcohols
- Anticoagulants
- Anticonvulsants
- Hydrocarbons
- Antidepressants
- Antipsychotics
- Antihistamines
- Carbon Monoxide
- Caustics
- Iron

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Which lab abnormality following an acetaminophen overdose is most concerning and associated with a POOR prognosis where liver transplant may be needed?

A. Bilirubin > 3

B. AST > 10,000

C. Alkaline phosphatase >300

D. INR > 6.5

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AST/ALT elevations following acetaminophen overdose typically begins to occur by what time period following an acute ingestion?

A. 4-8 hours

B. 8-16 hours

C. 16- 24 hours

D. 28-32 hours

E. 48-72 hours

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Which of the following regarding acetaminophen overdose is most accurate?

A. Children are more susceptible to hepatotoxicity following acute ingestions compared to adults

B. The enzyme responsible for NAPQI production is alkaline phosphatase

C. A typical threshold for a toxic acetaminophen ingestion is > 50 mg/kg

D. N-acetylcysteine is indicated >24 hours after ingestion in the presence of ongoing liver toxicity

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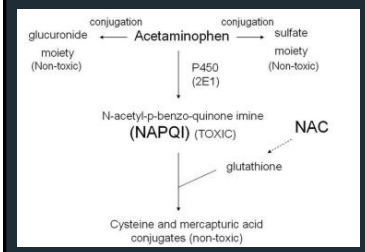
Acetaminophen Toxicity



1. Therapeutic ingestions
   - Glucuronidation
   - Sulfation
2. Toxic ingestion
   - ↑ NAPQI produced
3. Enzyme responsible for NAPQI?
   - p450 (2E1)
4. How the body clears NAPQI?
   - Glutathione
5. How to replenish glutathione?
   - N-Acetylcysteine

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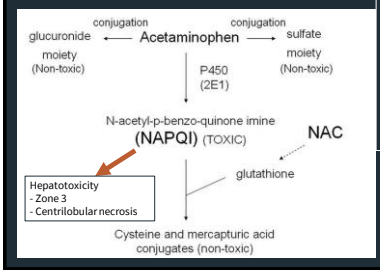
Acetaminophen Toxicity



1. Therapeutic ingestions
   - Glucuronidation
   - Sulfation
2. Toxic Ingestion:
   - NAPQI produced
3. Enzyme responsible?
   - p450 2E1
4. How to rid the body of NAPQI?
   - Glutathione
5. How to replenish Glutathione?
   - N-Acetylcysteine

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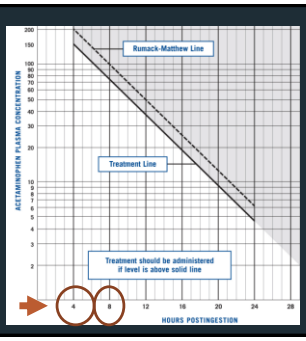
Acetaminophen Toxicity

- Potentially Toxic ingestion: > 150 mg/kg
- Rumack-Matthew nomogram for ACUTE ingestions only
- Level at 4 hours or later guides treatment
- Ideally treat by < 8 hours to prevent hepatotoxicity



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Acetaminophen Toxicity

N-Acetylcysteine:

→ NAC replenishes endogenous glutathione + other mechanisms

→ IV/Oral formulations, Safe in pregnancy

→ For Acute ingestions above the RM treatment line

→ For Chronic and Unknown Time of Ingestions with Lab abnormalities

→ Can be given after 24 hours

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Acetaminophen Toxicity

4 Stages of Toxicity

Stage 1 (0-24 hr): No symptoms aside for N/V → Labs NORMAL until 20-24 hrs

Stage 2 (24-72 hr): Right upper quadrant pain → AST/ALT/INR climb

Stage 3 (72-96+ hr): Encephalopathy, N/V→ AST/ALT/INR peak, hypoglycemia

Stage 4 (96 + hr): Death OR Full recovery

** Children tend to tolerate acute overdoses better than adults

Stage 2/3/4 Predictors of Impending Liver Failure:
- ↑INR
- ↑Creatinine
- Acidosis
- Encephalopathy

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24 hours after a toxic ingestion which of the following is least likely to have an anion gap?

A. Methanol
B. Ethylene glycol
C. Isopropanol
D. Propylene glycol

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Which of the following associations is correct?

A. Ethylene glycol metabolite → Formic acid
B. Methanol toxicity→ Renal Failure
C. Inhibition of alcohol dehydrogenase → Fomepizole
D. Methanol poisoning → Role for treatment with thiamine and Vitamin B6

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Most likely outcome after a significant isopropanol ingestion?

A. Renal failure
B. Blindness
C. Gastritis
D. Hepatic failure

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Toxic Alcohols

- **Methanol:** Window washer fluid, wood alcohol
- **Ethylene Glycol:** Antifreeze (Sweet taste!!), paints, solvents
- **Isopropyl alcohol** rubbing alcohol
  - Most common of alcohol ingestions

1. Toxic metabolites?
2. End organ toxicity if not treated?
3. Which create an anion gap?
4. Which create an osmolar gap?
5. Appropriate treatments?

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Osmol and Anion Gap

Osmol Gap:

Measured osmolality - Calculated osmolarity

Calculated Osmolarity:

2 Na + BUN/2.8 + Glucose/18 + EtOH/4.6

An Osmol Gap > 30 is highly suggestive of a toxic alcohol ingestion

Anion Gap: Na – (HCO_3 + Cl)

M: Methanol
U:
D:
P: Phenformin → Metformin
I: INH
L: Lactic acidosis
E: Ethylene Glycol
S: Salicylates

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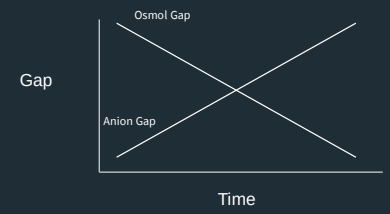
Toxic Alcohols- Timing of anion and osmol gap



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Toxic Alcohols

Methanol
Alcohol Dehydrogenase
Formaldehyde
Formic Acid
Folic Acid
CO2 + H2O

Ethylene Glycol
Alcohol Dehydrogenase
Glycoaledehyde
Glycolic acid
Thiamine, B6, Mg
Oxalic Acid
Glycine + others

Isopropanol
Alcohol Dehydrogenase
Acetone

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Methanol

Methanol produces inebriation

Toxic Metabolite → Formic Acid (delayed)

Formic acid accumulation→ Optic nerve damage (irreversible blindness)

Treatment:
- Prevent Methanol to formic acid conversion- fomepizole/ Ethanol
- Remove methanol → Hemodialysis
- Correct acidosis
- Speed up formic acid breakdown

Methanol
Alcohol Dehydrogenase
Formaldehyde
Formic Acid
Folic Acid
CO2 + H2O

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Ethylene glycol

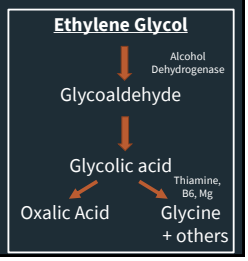
Ethylene Glycol produces inebriation

Toxic Metabolite → Oxalic Acid (delayed)

Oxalic acid accumulation→ Renal Failure, Hypocalcemia, acidosis

- Calcium oxalate crystals & Fluorescein in urine

Treatment:
- Prevent Ethylene glycol to oxalic acid conversion- fomepizole/ Ethanol
- Remove Ethylene Glycol→ Hemodialysis
- Correct acidosis. - Shift metabolism away from oxalic acid



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Isopropanol

Isopropyl Alcohol produces inebriation

Metabolite → Acetone (delayed)

Mild to Moderate GI Irritation from Isopropyl Alcohol

Inebriation from acetone

No need for treatment

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What is the half life of carbon monoxide on room air?

A.12 hours

B.6 hour

C.3 hours

D.90 minutes

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What is true regarding the effects of carbon monoxide?

A. Leads to decreased ability to release oxygen at cellular level
B. Produces pulse ox levels routinely measured at 85%
C. Can present with profound cyanosis at moderate levels (25-30%)
D. Leads to sudden unconsciousness with brief exposures at high levels

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Which patient most clearly would require hyperbaric oxygen with the following CO levels obtained within 1 hour from exposure?

A. Pregnant woman with a COHb level of 18% and minimal symptoms

B. Young adult with a COHB level of 24% and no symptoms

C. Middle aged woman with a COHb level of 18% and profound diarrhea

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Carbon Monoxide Poisoning

Common sources: Faulty furnaces, indoor grilling, portable Generators, house fires

One of most common toxicological cause of death in the US

Carbon monoxide:

- Odorless
- Colorless

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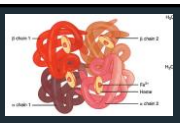
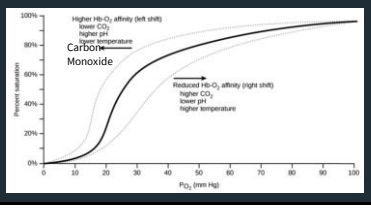
Carbon Monoxide Poisoning

Carbon Monoxide:

- CO binds to Hb with 250X the affinity of Oxygen
- Oxyhemoglobin dissociation curve is moved to the **left (**O_2 not released from Hb**)**





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Carbon Monoxide Poisoning

Presentation

- Headache (most common)
- Nausea/Vomiting (common in peds)
- Altered mental status
- Seizure
- Syncope/MI
- Coma

Laboratory Studies

- Normal bedside Pulse Ox
- ABG: High pO_2 High OxyHb UNLESS co-oximetry used to directly measure various forms of Hb
- Troponin
- Carboxyhemoglobin level

Always consider with more than one person sick in the same household

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Carbon Monoxide- Levels and Treatment

Carbon Monoxide levels drop over time

CO half-lives

- Room air → 4-6 hours
- 100% NRB → 90 minutes
- Hyperbaric O_2 → 20 minutes

Who requires Hyperbaric Treatment?

- Controversial
- Pregnant: COHb >15%
- Seizures, Coma, Syncope
- Other indications? (particular levels?)

HBO may reduce acute and Long term Neuro deficits

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Which is true of alkali ingestions?

A. Associated with coagulation necrosis
B. Increased long term risk of esophageal cancer
C. Appropriate to treat with neutralization with acidic juice (orange juice)
D. Mild/Moderate ingestions require early antibiotics/steroids to reduce mortality

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Common electrolyte abnormality seen following hydrofluoric acid exposures?

A. Hyperkalemia
B. Hypocalcemia
C. Hypermagnesemia
D. Hyponatremia

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Caustic Ingestions

Acids

- Coagulation necrosis
- Typical scar and eschar formation

Alkalis

- Liquefaction necrosis
- Full thickness injury may occur

Presence or absence of oropharyngeal injuries does adequately predict esophageal injuries

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Caustic Ingestions- Evaluation and Treatment

- Charcoal, neutralization, and Lavage NOT recommended
- Chest XRAY/CT scan if perforation suspected
- Endoscopy in first 24-48 hours may be considered
- Long term risk of Esophageal CA (alkali>acid)

Important questions?
- What substance
- What pH
- What Concentration
- What Quantity

Asymptomatic children with low volume household bleach ingestions are at LOW RISK

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Hydrofluoric Acid Exposure

Rust remover & Glass etchers

- Fluoride is a STRONG negative ion that binds Calcium & Magnesium
- Dermal, Inhalation, and Ingestion all produce tissue damage and possible systemic toxicity (hypocalcemia)
- Arrhythmia due to hypocalcemia → prolonged QT (most common cause of death)
- Pain on skin may be profound with little initial dermal findings

HF acid treatment:
- Calcium gluconate (local infiltration, topical gel, arterial)
- Calcium gluconate if swallowed
- Inhaled Calcium gluconate if inhaled

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Which is true regarding salicylate poisonings

A. 60 mg/dL is typically used in acute overdoses as a threshold to consider hemodialysis

B. A metabolic acidosis is typically seen in adults BEFORE the respiratory alkalosis

C. Hypokalemia must be corrected for urine alkalinization to be effective

D. Salicylate induced seizures should be treated with phenytoin/fosphenytoin or keppra

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Initial manifestations of mildly elevated salicylate poisoning most commonly include:

A. Myoclonus

B. Nystagmus

C. Tinnitus

D. Seizures

E. Hyperthermia

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Aspirin Poisoning

- Primary respiratory alkalosis (less common in children)
  - Medulla is directly effected → ↑Ventilation
- Uncouples oxidative phosphorylation
  - Leads to heat generation (↑ temperature)
  - Contributes to a primary metabolic acidosis
- Metabolic alkalosis from profound GI effects

Classic Labs:
- pH 7.45 (but may be low)
- Low pCO_2
- Low Bicarb
- Anion Gap ↑
- Hypoglycemia?

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Aspirin Poisoning

- Toxic acute ingestions typically > 150 mg/kg
  - No treatment nomogram exists
- Single salicylate levels are not adequate
  - Absorption rates are erratic
  - Bezoar and pylorospasm can occur
- Levels >30 mg/dL are abnormal
  - Acute OD >90 mg/dL potentially lethal
  - Chronic OD levels >60 mg/dL potentially lethal

5 ml= 5-7 grams of salicylic acid

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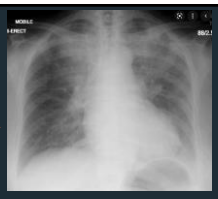
Aspirin Poisoning

- Tachypnea
  - Primary alkalosis
  - Non Cardiogenic Pulmonary Edema
- Hyperthermia and Tachycardia
- Nausea and Vomiting
  - Less common with chronic ingestions
- Tinnitus
- Altered Mental Status: with higher levels
- Coma & Seizures → Signs of impending death



Classically described with heroin & Aspirin

Consider in elderly with altered mental status + fever with no clear source

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Aspirin Poisoning

- Charcoal + Whole Bowel Irrigation (EC products)
- Alkalinize urine
  - $NaHCO_3$ for goal pH of 7.45-7.55
- Correct hypokalemia
  - Without Potassium normalization urine alkalinization doesn't work
- Intubate carefully: Must maintain respiratory alkalosis

Hemodialysis indications:
- Seizures
- Significant AMS
- Renal Failure
- Volume overload
- Refractory acidosis
- >90-100 acute
- >60 chronic

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Which would be the most important intervention following an ingestion of cup of gasoline?

A. Charcoal

B. NG tube placement and lavage

C. No intervention and NPO status

D. Whole bowel irrigation

E. Syrup of ipecac

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Your 18 yo patient presents in pulsatile V-tach following inhalation of carburetor cleaner to get high. What is the best treatment?

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B. Procainamide

C. Epinephrine

D. Benzos

E. Esmolol

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Hydrocarbon Toxicity

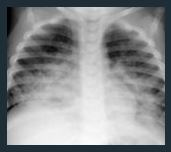
Toxicity is Pulmonary 2° aspiration → pneumonitis

High risk substance:

- Low viscosity
- High volatility
- Low surface tension

Treatment:

- Observation for sign/symptoms at least 6 hours
- Don't induce vomiting (typically **NO** GI decontamination)
- No Steroids or Antibiotics for pneumonitis



Hydrocarbon pneumonitis
XRAY findings delayed up to 6 hours

No cough/SOB/hypoxia in first 6 hours makes this unlikely

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Hydrocarbon Toxicity

Sudden Sniffing Death:

- Occurs in recreational huffers & baggers
- Leads to myocardium hypersensitivity → Ventricular arrhythmia

Prevention:

- Avoid producing a catecholamine surge
- Should arrhythmia develop → Beta-Blocker over Epi

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What will be seen in the early stages of all significant iron ingestions (within 6 hours)?

A. Nausea and vomiting
B. Fever
C. Lactic acidosis
D. Hypotension

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Iron Poisoning- 5 stages to remember

Typically need 30-40 mg/kg of elemental iron

Different Iron containing products contain varying elemental iron

1. GI irritation (0-6 hr): Vomiting, diarrhea, Abdominal pain (MUST HAVE THIS PHASE TO HAVE TOXICITY)
2. Quiescent phase (6-24 hr): As you slowly go into shock
3. Shock and Metabolic acidosis (6-72 hr): Lactic acidosis, hypotension
4. Hepatotoxicity (12-96 hr): Fulminant Liver failure
5. Bowel obstruction (2-8 wk): Gastric outlet obstruction

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Iron Poisoning

Assessment

→ 4 hour Iron level can be helpful
→ Poor predictive labs: ↑ WBC, ↑Glucose, ↑Anion Gap
→ XRAYS may identify radio-opaque pills
→ Monitor Liver labs

Treatment:

→ No role for charcoal
→ Whole bowel Irrigation
→ Deferoxamine as a chelating agent

Deferoxamine Indications
Severe GI symptoms, acidosis, shock

Serum iron level >500

Vin Rosa Urine

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Your patient presents 2 hours after an ingestion of D-Con superwarfarin. What are appropriate steps at this stage?

A. Obtain PT,PTT and administer Vitamin K 10 mg PO
B. Obtain PT,PTT and administer Vitamin K 10 mg IV
C. Obtain PT,PTT and administer FFP/PCC
D. Obtain PT,PTT and no medications should be administered.

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Anticoagulants

Warfarin vs Superwarfarin

- PT begins to rises around 12 hours
- Peak PT elevation at 36-72 hours, Effect up to 8 days
- Duration of Superwarfarin → 6-8 weeks or longer

Mechanism: Inhibits Vit K clotting factor production (II, IV, IX, X)

Treatment:

- Vitamin K (delayed effects), FFP, PCC
- Treat only if bleeding or High INR (>10?)
- INR checks every 12- 24 hours for 2-3 days

Drugs Producing ↑ INR
Bactrim
Quinolones
Doxy/Tetracycline
Metronidazole
Erythromycin
Azithromycin

Drugs producing ↓ INR
Rifampin

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Antidepressants- TCAs

Clinical Effects Occur **within 6 hours (**no symptoms in 6 hours can be cleared)

1. Sodium Channel Blockade (QRS Prolongation) → Sodium Bicarbonate
2. Alpha antagonism (Hypotension) → Norepinephrine
3. GABA antagonist (Seizures) → Benzos
4. Antimuscarinic (Anticholinergic toxidrome) → Supportive care
   - Dry mouth and skin, Dilated pupils, Urinary retention, Flushed appearance
5. Potassium Channel Blockade (QT prolongation)

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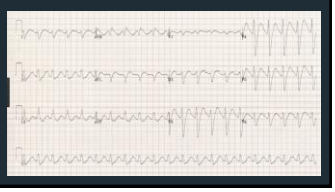
Antidepressants- TCAs

EKG findings:

- Tachycardia
- QRS prolongation (treat if >100 msec)
- Right axis deviation
- Large prolonged R wave in aVR
- QT prolongation

QRS prolongation Treatment
- $NaHCO_3$ boluses to drive QRS <100-120 msec
- 3 amps $NaHCO_3$ in 1 liter D5W at 200 mL/hr
- Lidocaine as second line treatment
- Lipid emulsion can be considered



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Antidepressants- Others

SSRIs: overall safe

- Serotonin syndrome uncommon with single serotonergic med ingestion
- Citalopram: Delayed effects up to 12 hours, Seizures, QT prolongation

Bupropion:

- Effects onset occurring up to 12-24 hours after ingestion
- Seizures occurring in overdose are common
- QRS and QT prolongation are reported

Trazodone:

- CNS depression, Hypotension, priapism

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Serotonin Syndrome

Most cases come from non-overdose

More than one medication with serotonergic effects

Classic features: (onset over 6 hours)

HOT: Elevated temps (mild to >106)

M: Mental Status Change

A: Autonomic dysfx: Tachy, Diaphoresis

N: Neuromuscular: myclonus (lower>upper)

Serotonin Related Meds
SSRI
SNRIs
Lithium
TCA
Phenothiazines
DXM
Cocaine/Amphetamines
Meperidine
MAO inhibitors
St. John's wort

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Serotonin Syndrome

Treatment:

- Cooling measures (cool mist, intubation)
- Supportive care
- Benzodiazepines (1st line)
- Cyproheptadine may be considered

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Antipsychotics

Typical Antipsychotics: Haldol, Thorazine

Migraine meds → metoclopramide, promethazine, prochlorperazine

Effects with therapeutic and supratherapeutic ingestions:

1. Sedation
2. Hypotension
3. Anti-cholinergic syndrome
4. Extrapyramidal Symptoms- Much less common with new 'atypical' agents

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Antipsychotics- Extrapyramidal Symptoms

1. Pseudoparkinsonism: tremor, rigidity, gait disturbance, bradykinesia
2. Akathisia: intolerable restlessness
3. Dystonia: oculogyric crisis, torticollis, trismus, facial grimacing
4. Tardive Dyskinesia: with prolonged usage
   - Involuntary lip and tongue movements
   - Typically not reversible

EPS treatment?
- Diphenhydramine
- Benzotropine
- Benzodiazepines

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Neuroleptic Malignant Syndrome → Almost always with 'typical' antipsychotics

Most are idiosyncratic non-overdose effects

Classic features: (Delayed over hours to days)

HOT: Elevated temps (mild to >106)

M: Mental Status Change

A: Autonomic dysfx: Tachy, Diaphoresis

N: Neuromuscular: Lead pipe rigidity Upper>Lower

NMS Treatment
Cooling
Benzos
Bromocriptine
Dantrolene?
Paralysis if severe

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Good Luck!!

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Anticonvulsants

Phenytoin: "patients feel drunk, but not drunk"

- Nystagmus: initial finding
- Ataxia→ Can't walk
- Sedation and paradoxical seizure: w/ higher levels

Carbamazepine:

- Disorientation and ataxia
- Anticholinergic appearance, QRS prolongation
- Paradoxical seizures can occur

-Check Free phenytoin and Free carbamazepine levels
- Low albumin state can produce symptoms at lower total levels

Treatment is supportive for phenytoin and carbamazepine

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Anticonvulsants

Valproic Acid:

- Sedation at high levels
- Hyperammonemia is common (carnitine to treat)
- Supportive care

Phenobarbital

- Very prolonged half lives (60+ hours)
- Produces profound sedation, appear 'brain dead' at high levels
- Treatment → Urine alkalinization, Multi-dose charcoal, Hemodialysis

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Antihistamines

Presentation

→ Hot as a hare (elevated temp)
→ Dry as a bone (absence of sweating)
→ Full as a tea cup (urinary retention)
→ Blind as a bat (mydriasis)
→ Mad as a hatter (confusion)

Treatment

→ Cooling measure: cool mist, ice packs
→ No role for antipyretics
→ Urinary Catheter
→ Physostigmine may be considered but NOT TYPICALLY needed
  - Contraindicated with TCAs As
  - QRS prolongation, Seizures

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Antihistamines

Benadryl is a common ingestion

- Associated with seizures in overdose
- QRS prolongation is reported

Large number of medications with anti-histamine/anti-muscarinic features:

- Phenothiazines (Phenergan, Compazine, Reglan)
- Doxylamine, hydroxyzine
- TCA, Typical Antipsychotics

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Good Luck

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Ethanol Withdrawal

Early Findings:

→ Early Tremors, agitation, and GI → 6-8 hours

→ Withdrawal Seizures (6-48 hrs): short lived, no AMS (no traditional anti-epileptics needed)

→ Alcoholic Hallucinosis (12-24 hrs): visual or auditory, respond to benzos/ethanol

Late Findings:

→ Delirium tremens (2-4 days): Confusion, Hallucinations, Fever, Tachycardia/HTN

→ Treatment: Benzos/Barbs + cooling measures
- A true medical emergency

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Important Ethanol Related Diagnosis

Wernicke's encephalopathy: Classic triad

1. Ataxia
2. Ophthalmoplegia(6th nerve palsy) and/or Nystagmus
3. Altered mental status

- Responsive to high dose thiamine supplementation

Korsakoff's pychosis: due to chronic thiamine depletion

- Irreversible
- Retrograde amnesia and confabulation

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