

Stimulant Drugs of Abuse

I. Amphetamines

A. Effects

1. Euphoria
2. Agitation/psychosis
3. Chest pain
4. Seizures
5. Hyperthermia

B. Mechanism

1. NE, serotonin, dopamine reuptake inhibition

C. Antidote/Treatment

1. Supportive cares
2. Temperature management
3. Agitation/Psychosis
 - a) Benzodiazepines
 - b) Antipsychotics

II. Cocaine

A. Effects

1. Euphoria
2. Agitation/psychosis
3. Chest pain
4. Seizures
5. Hyperthermia

B. Mechanism

1. NE, serotonin, dopamine reuptake inhibition
2. Snorted in cocaine form
3. "Crack" form can be smoked

C. Antidote/Treatment

1. Supportive cares
2. Temperature management
3. Agitation/Psychosis
 - a) Benzodiazepines
 - b) Antipsychotics

III. MDMA (methylenedioxymethamphetamine)
(AKA: Molly, Ecstasy)

A. Effects

1. Euphoria
2. Increased social interaction
3. Tactile stimulation enjoyed by users
4. Serotonin syndrome
5. Renal failure
6. Agitation
7. Seizures
8. Hyperthermia

B. Mechanism

1. Serotonin reuptake inhibition

C. Antidote/Treatment

1. Supportive cares
2. Temperature management
3. Agitation/Psychosis
 - a) Benzodiazepines
 - b) Antipsychotics

IV. Synthetic cathinones (AKA: Bath salts)

A. Effects

1. Euphoria
2. Agitation/psychosis
3. Seizures
4. Hyperthermia

B. Mechanism

1. NE, epinephrine, dopamine reuptake inhibition

C. Antidote/Treatment

1. Supportive cares
2. Temperature management
3. Agitation/Psychosis
 - a) Benzodiazepines
 - b) Antipsychotics

Selected Toxins

I. Acetaminophen

A. Mechanism

1. Depletes glutathione → increased toxic NAPQI levels

B. Symptoms/Findings

1. 0-24 hrs.: Loss of appetite, nausea
2. 24-72 hrs.: Abdominal pain, elevated liver enzymes
3. 72-96 hrs.: Liver failure, coagulopathy, jaundice, encephalopathy, +/-death
4. 96 hrs.: recovery

C. Antidote/Treatment

1. N-acetylcysteine
2. Rumack-Mathew nomogram guides treatment necessity

II. Methanol & Ethylene Glycol

A. Mechanism

1. Converted to toxic metabolites by alcohol dehydrogenase

B. Symptoms/Findings

1. Ethylene glycol
 - a) Renal failure
 - b) Heart failure
 - c) Metabolic acidosis
 - d) Hypocalcemia
2. Methanol
 - a) Blindness
 - b) Seizures
 - c) Coma
 - d) Metabolic acidosis

C. Antidote/Treatment

1. Fomepizole
 - a) Inhibits alcohol dehydrogenase
 - b) Alcohols excreted by kidneys prior to conversion to toxic form
2. Ethanol
 - a) Competes for alcohol dehydrogenase
 - b) Alcohols excreted by kidneys prior to conversion to toxic form

III. Carbon Monoxide

A. Mechanism

1. Binds to hemoglobin and converted to carboxyhemoglobin
 - a) Limits oxygen transport
 - b) Oxygen carrying sites bind oxygen more strongly, reducing release to tissues, causing tissue hypoxia

B. Symptoms/Findings

1. "Cherry red" skin appearance
2. Lethargy
3. Headaches
4. Confusion
5. Seizures
6. Coma
7. Death

C. Antidote/Treatment

1. Removal from environment
2. 100% oxygen
3. Hyperbaric oxygen

IV. Cyanide

A. Mechanism

1. Inactivates metalloenzymes after binding
2. Inactivation of cytochrome oxidase arrests cellular respiration (inadequate ATP production)

B. Symptoms/Findings

1. Cellular death
2. Highly metabolic organs effected first
3. Death

C. Antidote/Treatment

1. Hydroxocobalamin
2. Sodium nitrite

V. Iron

A. Mechanism

1. High levels toxic to GI tract, cardiovascular, & CNS systems

B. Symptoms/Findings

1. Toxicity varies upon levels
2. GI disturbances
3. Hypotension
4. Hypovolemia
5. Metabolic acidosis
6. Liver failure
7. Multisystem organ failure
8. Death

C. Antidote/Treatment

1. Deferoxamine
 - a) Chelates iron & facilitates renal excretion

VI. Lead

A. Mechanism

1. Exposure from environment
2. Absorbed readily by children
3. Long half-life, 20-30 yrs. in bone tissue

B. Symptoms/Findings

1. CNS
 - a) Depressed IQ
 - b) Headaches
 - c) Clumsiness
 - d) Confusion
 - e) Convulsions
 - f) Coma
2. GI
 - a) Intestinal spasms/constipation
3. Hematologic
 - a) Anemia (hypochromic, microcytic)

C. Antidote/Treatment

1. Succimer
2. Dual therapy in encephalopathy
 - a) Dimercaprol
 - b) Calcium disodium EDTA

VII. Organophosphates/Carbamates

A. Mechanism

1. Increased acetylcholine levels
2. Carbamates
 - a) Reversible inhibition of acetylcholinesterase
3. Organophosphates
 - a) Irreversible inhibition of acetylcholinesterase

B. Symptoms/Findings

1. Excess acetylcholine
 - a) Mydriasis
 - b) Diarrhea
 - c) Lacrimation
 - d) Salivation
 - e) Vomiting
 - f) Urination
 - g) Weakness
 - h) Hypertension

C. Antidote/Treatment

1. Atropine
2. Pralidoxime (2-PAM)

Hallucinogens

I. LSD (lysergic acid diethylamide)

A. Mechanism

1. Partial agonist at 5-HT_{2A} receptors

B. Symptoms/Findings

1. Colorful hallucinations
2. Sleep disturbance
3. Loss of judgement

II. Marijuana (THC)

A. Mechanism

1. Cannabinoid receptor agonist

B. Symptoms/Findings

1. Relaxation
2. Increased appetite
3. Improved pain control
4. Tachycardia
5. Hallucinations
6. Diminished short-term memory
7. Decreased motor activity

Ethanol

I. Mechanism

A. Enhances effects of GABA

1. Induces release of serotonin & dopamine

B. Symptoms/Findings

1. CNS depressant
 - a) Inhibition
 - b) Impaired cognition
 - c) Respiratory depression & coma at high levels
2. Wernicke encephalopathy
 - a) Confusion, ataxia, ophthalmoplegia
3. Alcohol withdrawal
 - a) Life-threatening complication of alcohol abstinence in history of alcohol dependence
 - b) Symptoms/signs
 - (1) Anxiety/agitation
 - (2) Psychosis
 - (3) Autonomic dysregulation (hypertension/tachycardia)
 - (4) Tremors/tongue fasciculations
 - (5) Seizures (peak @ 24 hours)

C. Antidote/Treatment

1. Wernicke encephalopathy
 - a) Glucose/thiamine
2. Withdrawal
 - a) Benzodiazepines
 - b) Thiamine/folate/glucose
 - c) Electrolyte management
 - d) Other agents: dexmedetomidine, barbiturates, antipsychotics
3. Alcohol use disorder
 - a) Behavioral counseling
 - b) Acamprosate
 - c) Naltrexone
 - d) Disulfiram

www.catapultreview.com