PED-1407 Chocolate to Crystal Methamphetamine to the Cinnamon Challenge - Emergency Approach to the Intoxicated Child

BLS 08 / ALS 75 / 1.5 CEU

*Target Audience: All*

Pediatric and adolescent ingestions are common reasons for 911 dispatches and emergency department visits. With greater availability of medications and drugs, healthcare professionals need to stay sharp on current trends in medical toxicology. This lecture examines mind altering substances, initial prehospital approach to toxicology and stabilization for transport, poison control center resources, and ultimate emergency department and intensive care management.
Objectives

• Epidemiology

• History of Poisoning

• Review initial assessment of the child with a possible ingestion

• General management principles for toxic exposures
  • Case Based (12 common pediatric cases)

• Emerging drugs of abuse
  • Cathinones, Synthetics, Salvia, Maxy/MCAT, 25I, Kratom
Epidemiology

- 55 Poison Centers serving 295 million people
- 2.3 million exposures in 2011
  - 39% are children younger than 3 years
  - 52% in children younger than 6 years
- 1-800-222-1222
Introduction

• 95% decline in the number of pediatric poisoning deaths since 1960
  – child resistant packaging
  – heightened parental awareness
  – more sophisticated interventions
  – poison control centers
Figure 1. Motor vehicle traffic, poisoning, and drug poisoning death rates; United States, 1980-2008

NOTE: In 1999, the International Classification of Diseases, Tenth Revision (ICD–10) replaced the previous revision of the ICD (ICD–9). This resulted in approximately 5% fewer deaths being classified as motor-vehicle traffic–related deaths and 2% more deaths being classified as poisoning-related deaths. Therefore, death rates for 1998 and earlier are not directly comparable with those computed after 1998. Access data table for Figure 1 Adobe PDF file.

Epidemiology

• Unintentional (1-2 years)
  – Exploratory
  – Boys > girls
  – Unable to discriminate safe from unsafe liquid

• Intentional (adolescent)
  – Purposeful
  – Girls > boys
Introduction

• 60% of poison control center calls are for patients under the age of 17
• Most pediatric ingestions are accidental and minimally toxic
• Higher morbidity in adolescent ingestions
• Many pediatric patients present with unexplained signs and symptoms
Epidemiology

• Most common Pediatric Exposure
  – Cosmetics and personal care products (13%)
  – Cleaning substances (10%)
  – Analgesics (7.8%)
  – Foreign Bodies (7.4%)
  – Topicals (7.4%)
  – Cold and Cough Preparations (5.5%)
  – Plants (4.6%)
  – Pesticides (4.1%)
Epidemiology

- Around meal time
- Grandparents home
- Kerosene or gasoline in a soda bottle
- Older sibling can pharmaceutically treat younger sibling
History of Poison
Poison

- English literature 1230 A.D.
- A potion or draught prepared with deadly ingredients
- History of poisoning dates back thousands of years
Poisons, Poisoners, and Antidotes of Antiquity

• Earliest poisons
  – Plant extracts
  – Animal venoms
  – Minerals

• Uses
  – Hunting
  – Waging war
  – executions
Ebers Papyrus

• Ancient Egyptian text 1500 B.C.
  – Considered among earliest medical text
• Describes ancient poisons
  – Aconite, antimony, arsenic, cyanogenic glycosides, hemlock, lead, mandrake, opium, and wormwood
  – Mystical properties
  – Superstition and intrigue
  – Ingestion thought to be harmless to innocent and lethal to guilty
Origins of Toxicology

• Earliest humans used animal venoms and plant extracts for hunting, warfare and assassination.

• 400 BC: Hippocrates compiled a listing of a number of poisons and outlined some clinical toxicology principles.

• 1493-1541: Paracelsus—physician and philosopher
  • All substances are poisons; the right dose differentiates a poisons from a remedy.
  • “Dose determines toxicity.”

• 1775: Percival Pott found that soot caused scrotal cancer in chimney sweeps. Much later the carcinogens in soot found to be polycyclic aromatic hydrocarbons.

• 1972: Rachel Carson/EPA led to ban of insecticide DDT for environmental and health concerns
Examples of Toxicological Cases

- **399 B.C.** Socrates, Greek Philosopher, died of Hemlock poisoning (according to Plato)
  - Coniine is the active toxic ingredient
  - Antagonist for the nicotinic acetylcholine receptor, leading to cessation of neurotransmission, muscular and respiratory collapse and death

- **October 20th, 1740** Charles VI, Holy Roman Emperor, King of Bohemia, Hungary, and Croatia died from eating death cap mushrooms
  - Active ingredient is alpha-amanitin that inhibits RNA polymerase inhibiting protein synthesis leading to hepatocellular lysis, liver failure, kidney failure, coma, respiratory failure, and death
Examples of Toxicological Cases

- April 30th, 1945, Eva Braun, long-time companion of Hitler committed suicide with a cyanide capsule
  - Inhibitor of cytochrome c oxidase, part of complex IV of the electron transport chain and inhibits ATP production leading to brain death and heart cessation, hypoxia, and death

- Jan 16th, 1975 Bando Mitsugoro VIII, a famous Japanese Kabuki actor died from eating 4 livers of pufferfish
  - Active toxic ingredient is tetrodotoxin
  - Tetrodotoxin blocks voltage-gated sodium channels leading to suppression of neurotransmission, numbness, bronchospasms, coma, respiratory failure, death
Examples of Toxicological Cases

- 1932-1968: Minamata disaster—caused by methylmercury toxicity from industrial wastewater from Chisso Corporation in Minamata City in Japan
  - 2265 victims
  - Caused neurological syndrome associated with methyl mercury poisoning including ataxia, numbness, insanity, muscle weakness, hearing and speech loss, birth defects, paralysis, coma, death
  - Alters neurochemistry and neurotransmission through multiple mechanisms
- 1988, Saddam Hussein used sarin on Kurds, 1995, Japanese subway sarin attack by terrorist group; 2006 day 5 of “24”—Jack Bauer saves LA from VX attack in TV show; 2013 Assad uses sarin against rebels
  - Sarin and VX are an organophosphorus chemical warfare agents that inhibits acetylcholinesterase, leading to excess acetylcholine and hyperstimulation of neurons, resulting in seizures, tremoring, convulsions, excess salivation, excess tearing, urination, defecation, bronchoconstriction, respiratory failure, death
Evaluation

- History of poisoning
- Physical Examination
- Laboratory studies
- Gastrointestinal decontamination
Initial Assessment: Overview

• Treat the patient, not the poison
• Assessment triangle
  – General appearance
  – Work of breathing
  – Circulation
• ABCDs
• IV access and monitors
• High Suspicion
Evaluation of Suspected Poisoning

• ABC’s and routine ICU management
• Establishing the diagnosis
  – Must consider poisoning, especially in “at risk” age groups
    • Less than 6 year old with acute decompensation (AMS, arrhythmias, hypotension, metabolic acidosis, etc.)
Initial Assessment: Physical Examination

• Directed exam (after ABCs)
  – mental status
  – vital signs
  – pupillary size
  – skin signs
Initial Assessment: Diagnostics

- Cardiac monitoring or 12-lead EKG
- Chest and abdominal radiographs
- Electrolytes (anion and osmolar gaps)
- Toxin screening rarely helpful
- Specific drug levels
Secondary Assessment

• AMPLE

• A- Allergies

• M- Medications

• P- Past Medical History

• L- Last PO Intake

• E- Events Prior To Presentation
Secondary Assessment

- Obtain detailed history of the amount and time of ingestion
- Use family or friends as historians
- May need to search the home
History

• What?
• When?
• How much?
• Reliability...
What?

• Medication
• Illicit drug
• Hazardous chemical
What forms?

- Pill
- Solid
- Liquid
- Gaseous
What route?

- Ingestion
- Inhalation
- Topical
- Intravenous
When?

- Elapsed time
How much?

- Estimate amount
- Concentration
Prevention or Minimization of Absorption

- Ipecac
  - No longer recommended

- Gastric lavage (also almost never used)
  - massive ingestions
  - arrival within one hour of ingestion
Activated Charcoal

- Ineffective in some ingestions (garage)
  - pesticides
  - hydrocarbons
  - acids, alkalis, and alcohols
  - iron
  - lithium
Activated Charcoal

• Recommended dose
  – child under 6 years: 1 - 2 grams/kg
  – 6 years and older: 50 - 100 grams

– Sorbitol?
  • Hypernatremia
  • Dehydration
Cathartics

• Studies of the effectiveness of cathartics are inconclusive

• Complications related to systemic absorption
  – electrolyte disturbance and severe dehydration
  – neuromuscular impairment and coma
Whole Bowel Irrigation

- **Golytely® (PEG-ELS)**
  - combination of electrolytes and polyethylene glycol (PEG)
  - 0.5 L/hr for small children and 2 L/hr for adolescents and adults
  - administer for 4 - 6 hours or until effluent is clear
  - useful for ingestions of iron, lithium, and sustained release preparations
Enhancement of Excretion

• Ion trapping
  
  – Traps weak acids in renal tubular fluid
  
  – Dose 1-2 mEq/kg every 3-4 hours
  
  – alkalinization of the urine (goal pH 7-8)
    • salicylates, phenobarbital, TCA
Enhancement of Excretion

• Multiple dose charcoal
  – May cause bowel obstruction
  – phenobarbital, theophylline

• Hemodialysis
  – Alcohols
  – Salicylates
  – Lithium
WHO INGESTS?
What is ingested?

• Toddler/Preschoolers
  – Most common ingestion: *Acetaminophen*
  – Most common **fatal** ingestion: *Iron*

• Adolescents
  – Most common ingestion: *Acetaminophen*
  – Most common **fatal** ingestion: *Cyclic antidepressants*
NEW! Extra Strength PAIN RELIEF CAPLETS

ACETAMINOPHEN
CONTAINS NO ASPIRIN

Pills
Case #1

- You are called to transport a 16 year old girl after she tells her boyfriend “I took as much Tylenol® as I could”

- Denies other ingestions or medication use

- Ingestion occurred three hours prior
Case Progression

• Patient is anxious, diaphoretic nauseated

• PE reveals a mildly tender abdomen

• HR- 120 RR-20 BP 100/70
Do You Transport?

YES
Case Discussion: Acetaminophen

- Most widely used pediatric analgesic on the market
- Most common ingestion in toddlers, preschoolers and adolescents
- Normal cytochrome P-450 metabolism yields small amounts of free oxidants that are hepatotoxic
  - Glutathione depletion
Case Discussion: Stages

- **stage 1 (4 - 12 hours)**
  - malaise, nausea, vomiting
- **stage 2 (24 - 72 hours)**
  - asymptomatic, increasing LFTs
- **stage 3 (48 - 96 hours)**
  - liver failure, elevated prothrombin time
- **stage 4 (7 - 8 days)**
  - resolution of liver injury
Case Discussion: Diagnosis

- Kinetics dictate that a serum level be checked 4 hours after ingestion
- Toxic dose: 150 mg/kg
- 4 hour toxic blood level 150mg/dl
- Apply the level to the management nomogram
Rumack-Matthew nomogram for predicting prognosis of hepatotoxicity in acetaminophen overdoses.
Our Patient

• Charcoal 50mg

• 4 hour level is 215 µg/ml

• Now What?
Case Discussion:

*N*-acetylcysteine (NAC) Therapy

- Proven to be 100% effective when given within 8 - 16 hours of ingestion
- Load with 140 mg/kg orally
- Complete regimen with 17 subsequent doses of 70 mg/kg every four hours
Case Discussion:
\textit{N}-acetylcysteine (NAC) Therapy

- IV NAC (Acetadote)
- Load with 50 mg/kg over 4 hours
- Maintenance 100mg/kg over 16 hours
Case #2
Case #2

• 12 year old boy was dared by his friends to drink from a bottle filled with antifreeze

• Swallowed a few gulps, and then yelled and dropped the bottle

• His father, utters a few choice words and calls an ambulance
Case Progression

• Upon arrival, the child has clumsy movements with a decreased level of consciousness

• Vital signs: HR 120, RR 20, BP 80/50, T 37.4° C, weight 12 kg

• What class of toxin has this child ingested?
Alcohol

• Why can’t we let him ‘sleep it off’?
Case Discussion: Alcohols

- **Ethanol**
  - hypoglycemia, osmolar gap, ketoacidosis

- **Methanol**
  - blindness, large osmolar gap, metabolic acidosis

- **Ethylene glycol**
  - renal failure (calcium oxalate crystals), osmolar gap, metabolic acidosis
Alcohol metabolism

• Ethylene glycol
  – Broken down by ADH to oxalic acid
  – Results in renal failure

• Methanol
  – Broken down by ADH to formic acid
  – Results in blindness
Alcohol metabolism

• Ethanol
  – Broken down by ADH to CO2 and H2O
  – Results in DRUNK

• Isopropanol
  – Broken down by ADH to CO2 and H2O
  – Results in REALLY DRUNK
Osmolar Gap

• osmolar gap = measured – calculated

• calculated = (2 x Na) + (glucose/18) +(BUN/2.8)

• normal = 10 – 15 mOsm/kg H$_2$O

• all alcohols cause an elevated osmolar gap
Anion Gap

• \([\text{Na} + \text{K}] - [\text{HCO}_3^- + \text{Cl}] > 12\)
  • M- Methanol
  • U- uremia
  • D- DKA
  • P- Paraldehyde
  • I- Iron
  • L- Lactic Acidosis
  • E- Ethylene Glycol
  • S- Salicylates
Case Progression

• Patient has an osmolar gap and metabolic acidosis consistent with ingestion of ethylene glycol

• Now what?
Therapeutic Intervention

• IV ethanol (old)
  – competes for alcohol dehydrogenase (ADH) to prevent build up of toxic metabolites

• Fomepizole (4-methyl pyrazole)
  – Blocks alcohol dehydrogenase (ADH)

• Requires ICU admission
Case #3
Case #3

• You arrive at a home where a parent has called 911. You find a 5 year old who is crying and rubbing at his arms yelling “get the bugs off me.”
• T-102, HR- 150, RR-23, BP- 100/60
• Skin is flushed, pupils are dilated and extremities are warm and dry.
• His neuro exam is nonfocal
• What toxidrome?
ANTI-CHOLINERGIC
Toxidrome: Anticholinergics/antihistamines

• Mad as a hatter
• Red as a beet
• Dry as a bone
• Hot as a hare
• Blind as a bat
Anticholinergic Toxidrome

“Blind as a Bat”
I can’t see!

“Mad as a Hatter”
confused

dilated pupils
(mydriasis)

“Dry as a Bone”
dry mouth
urinary retention

shaking

“Red as a Beet”
flushed skin

tachycardia
absent bowel sounds

grabbing invisible objects

“HOT as a Desert”
hyperthermia

So Hot!

Kloss and Bruce.com
Anticholinergic Toxidrome

• CNS
  – agitation, hallucinations, coma
• Respiratory
• Circulation
  – tachycardia, arrhythmias, hypertension
• Skin
  – warm, flushed, dry
• Eyes
  – mydriasis
Case Progression

• gastric decontamination
  – charcoal, 50 grams
• supportive care
• antidote: physostigmine
  – indications: coma, unstable vital signs
  – 0.5 mg IV (child) or 1 - 2 mg IV (teen)
  – Contraindicated if wide QRS
Case #4

• You are dispatched to a home after a call by a parent whose 2 year old was found with a container of dishwasher detergent in his hands and some around the mouth

• patient is asymptomatic

• physical exam is normal, including oropharynx
Case #4

• What are you going to do?

• Reassure parents and leave them to follow-up with the pediatrician as needed?

• Offer transport to the local ED?
Case Discussion: Caustics

- drain cleaners, oven cleaners, automatic dishwasher detergents
- If pH <3 or >12 = BAD
- **DO NOT LAVAGE, GIVE ACTIVATED CHARCOAL, GIVE CATHARTICS OR GIVE IPECAC**
Caustics

• Acids
  – Coagulation necrosis
  – Stomach injury

• Alkali
  – Liquefaction necrosis
  – Oropharyngeal and esophageal injury
Caustics

• Dilution
  – Water
  – Milk
  – Saline
  – Give within 30 minutes
Caustics

- Can your physical exam predict injury?

NO!!!!!
Case #5

• Grandma says her 18 month old grandson “isn’t acting right”

• Grandmother is concerned that child may have ingested some of her medication
  – Digoxin
  – Furosemide
  – “some kind of” antihypertensive medication
Case Progression

• Examination reveals lethargic child with 1 - 2 mm pupils

• vital signs: HR 70, RR 12, BP 80/45, T 37º C, weight 13 kg
Case Progression

- 1 - 2 mm pupils - miosis
- HR- 70- bradycardia
- RR- 12- bradypnea
Which medication?

• Digoxin?

• Furosemide?

• Other Antihypertensive?

• Opiate?
Case Discussion: Clonidine

- central acting antihypertensive; also used to treat narcotic withdrawal
- comes in small tablets and in patch form
- low blood pressure (after transient hypertension), miosis, coma
- naloxone may work to reverse respiratory depression
Clonidine

• Always be ready to support breathing

• Rapid decline
Opiate/Clonidine Toxidrome

- CNS
  - lethargy, seizures, coma
- respiratory
  - slow respirations, pulmonary edema
- circulation
  - hypotension, bradycardia
- skin
- eyes
  - miosis
Case #6

- 3 year old boy who drank from a soda bottle containing gasoline
- Cried immediately, gagged and coughed, and then vomited
- Alert and crying. HR- 122, RR-24, BP-90/60
- You arrive on the scene...do you transport?
Case Discussion: Hydrocarbons

• Degreasers, solvents, fuels, pesticides, and additives in household cleaners and polishes

• Low surface tension allows for rapid movement through pulmonary system

• Toxic effects
  – pulmonary, cardiovascular, or systemic
Case Discussion: Management Issues

• Admit all symptomatic patients and obtain ABG, EKG, and CXR
• Absence of symptoms for 4-6 hours after ingestion makes chemical pneumonia unlikely
• Ipecac? **NO!!**
• Steroids? **NO!!**
• Prophylactic antibiotics? **NO!!**
Case #7

- A 5 year old girl was at school, when she developed
  - Nausea
  - Vomiting
  - bloody diarrhea
Case #7

- Patient reports that she ate some of her mother’s prenatal vitamins at breakfast
- The bottle had contained 30 pills of ferrous sulfate, and is now empty
Case Discussion: Iron

- Toxic exposure is based on elemental iron load

- Most children’s preparations contain less iron than adult preparations
  - children’s: 3 - 25 mg per pill
  - adult: 37 - 65 mg per pill
Case Discussion: Iron

- Toxic dose: 40-69 mg/kg elemental iron
- Lethal Dose: 180 mg/kg elemental iron
Case Discussion: Clinical Presentation

- Gastrointestinal stage (30min-6h)
  - nausea, vomiting, and bloody diarrhea
- Relative stability (6-24h)
  - apparent clinical improvement
- Shock stage (12-48h)
  - coma, shock, seizures, coagulopathy
- Hepatotoxicity stage (within 48 hours)
- GI scarring (4-6 weeks)
Case Discussion: Management

- AXR- iron tablets are radio-opaque
Case Discussion: Management

• Whole bowel irrigation
  – 500cc/hour (children) 1-2L/hr (adults)
  – Effluent=Influent

• Deferoxamine
  – Serum fe >500mcg/dl
  – Significant clinical toxicity
  – Persistent XR findings despite GI decontamination
DANGER PELIGRO
PESTICIDES PESTICIDAS
KEEP OUT
NO ENTRE
Case #8

• 6 year old boy who was playing outside and returned to his house with respiratory distress

• You arrive on the seen and you note him to be lethargic, diaphoretic, and in moderate respiratory distress
Case Progression

• Physical exam reveals rales and wheezing in all lung fields with copious oral secretions

• Lethargic with 1 mm pupils

• Vital signs: HR 50, RR 70, BP 90/palp, T 37.8°C, weight 25 kg
Cholinergic (Organophosphate) Toxidrome

- clinical presentation
  - D diarrhea
  - U urination
  - M miosis
  - B bradycardia
  - B bronchosecrections
  - E emesis
  - L lacrimation
  - S salivation
Cholinergic toxidrome- organophosphate poisoning

• **ATIONS**
  – Salivation
  – Lacrimation
  – Urination
  – Fasciculation

• **HEAS**
  – Diarrhea
  – Bronchorrhea
  – Rhinorrhea
  – Bradycardia
Cholinergic agents

• Inhibit

ACETYLCOLINESTERASE
Case Discussion: Management

- **REMOVE CLOTHING**- Skin decontamination

- **Atropine** (vagal block)
  - Dries secretions, decreases bronchoconstriction and increases heart rate
  - Large doses (0.5 - 10 mg IV) may be needed

- **Pralidoxime** (Protopam, 2-PAM)
  - Regenerates acetylcholinesterase
  - 20 - 50 mg/kg/dose (IM or IV)
Case #9

• 3 year old has fever, progressive sleepiness, and respiratory distress 2 hours after drinking some oil of wintergreen from the kitchen cabinet

• Patient noted to be lethargic and tachypneic, with adequate circulation
Case Progression

• Patient responds to mother’s voice, and there are no focal findings on neurologic exam

• Vital signs: HR 140, RR 60 and deep, BP 90/70, T 40º C, weight 12 kg

• I stat shows 7.25/25 HCO3-10
What did this patient ingest????

- Hint: Remember your blood gas

- PH: 7.25
- CO2: 25
- HCO3: 10
Salicylates

- Metabolic acidosis with respiratory alkalosis=

- SALICYLATE toxicity until proven otherwise
Case Discussion: Salicylates

- Respiratory alkalosis
- Increased Temp, HR, RR
- Alters platelet function and bleeding time
- May develop cerebral edema secondary to vasoactive effects
- Tinnitus
Case Discussion: Clinical Manifestations

• Vomiting, hyperpnea, tinnitus, and lethargy

• Severe intoxication: coma, seizures, hypoglycemia, hyperthermia, and pulmonary edema

• Death from cardiovascular collapse
Case Discussion: Toxic Dose

- Therapeutic dose is 10 - 15 mg/kg
- Toxic dose is over 150 mg/kg
- Done nomogram ONLY useful in acute toxicity
Salicylate toxicity management

- Urinary alkalinization with sodium bicarbonate to maintain urine pH > 7
  - Keeps ASA in renal tubules
Salicylate toxicity management

• Hemodialysis is very effective for drug removal and to control acid-base imbalance
  – Acute ingestions > 100mg/dl
  – Chronic ingestions > 60 mg/dl
  – Persistent rise in ASA
  – Renal insufficiency
  – Refractory metabolic acidosis
  – Altered mental status
Case #10

• Called to transport a 13 year old after her parents arrived home from work to find the patient unresponsive

• Long history of psychiatric problems in the family, including the patient
Case Progression

• VS: T 38°C, HR 120s with widened QRS on the monitor, RR 24, BP 90/50

• Pupils are dilated and reactive, skin is dry and flushed, and patient is responding to deep pain only
Case Discussion: Tricyclic Antidepressants

• Clinical picture is... anticholinergic intoxication, CNS depression, and cardiovascular instability

• Mainstay of therapy is sodium bicarbonate in addition to supportive measures
Case Progression: Management

- Charcoal, 50 grams after airway secured
- Fluid bolus
- Alkalinization
  - 100 meq/L of NaHCO₃
- EKG
  - QRS duration, PR interval, QTc
    - R wave height of > 3 mm in aVR
    - QRS duration of > 120 ms
QRS duration

- QRS > 100ms associated with seizures
- QRS > 160ms associated with cardiac arrhythmia
Case #11

• 2 year old who was found unconscious with empty bottle of grandma’s calcium channel blockers at his side

• multiple episodes of vomiting on transport to the hospital, producing pill fragments
Case Progression

- VS: T 37.5°C, HR 45 with third degree heart block, RR10, BP 70/25
- Patient responsive to deep pain only, extremities cool with decreased pulses
Case Discussion: Calcium Channel Blockers

• Morbidity and mortality after toxic exposures result from cardiovascular collapse

• Therapy
  – gastric decontamination (charcoal, WBI)
  – blood pressure support
  – calcium
  – glucagon
Case Progression

- On arrival in the ER, the boys are afebrile with normal vital signs
- $O_2$ sats of 98%
- CBC, EKG, and CXR are normal
Case # 12

• 15 yo twins are brought to the ED by mom.
• She found them both unconscious in the hallway at home and dragged them out of the house where they both woke up.
• She is now in the ED and they both are alert and appropriate.
You are bothered by the fact that both boys had LOC. And, you cannot chalk it up to teenage pregnancy.

Carboxy hemoglobin level
Case Discussion: Carbon Monoxide Poisoning

• CO-hgb affinity is 250 \textit{times} O_2-hgb affinity; results in decreased oxygen delivery to the tissues

• Non-irritating, tasteless, odorless, and colorless gas

• Sources: smoke inhalation, auto exhaust, poorly ventilated charcoal, kerosene or gas heaters, and cigarette smoke
Case Discussion: Carbon Monoxide

• Toxic effects are the result of cellular hypoxia
• Concentrations of 20% produce neurologic symptoms, and death can occur with concentrations over 60%
• Pulse oximetry may be normal
• Peak level may occur in the field prior to O₂ delivery
Case Discussion: Therapy

- Administering oxygen at high concentrations reduces half life of CO from 6 hours to 1 hour
- Hyperbaric therapy
  - neurologic dysfunction
  - pregnant women
  - Unstable
  - children with levels over 25%
Summary

• Most pediatric ingestions are non-life threatening

• Recognition of toxidromes and knowledge of available antidotes MAY assist in the initial management of the poisoned patient, but supportive measures are more likely to be life saving
Emerging Drugs of Abuse

Monitoring the Future is an annual survey of 8th, 10th, and 12th-graders conducted by researchers at the University of Michigan, Ann Arbor, under a grant from the National Institute on Drug Abuse, part of the National Institutes of Health. Since 1975, the survey has measured drug, alcohol, and cigarette use and related attitudes in 12th-graders nationwide. Eighth and 10th graders were added to the survey in 1991.

Overall, 41,675 students from 384 public and private schools participated in the 2013 survey.
Emerging Drugs of Abuse

• 25I
• Maxy/MCAT/MCE
• Kratom
• Synthetic cathinones
• Synthetic cannabinoids
• Salvia divinorum
MCAT – Maxy -- Ket

What's getting up your nose?
Legal doesn't mean safe

KET

MCAT

Meow

'Bombing' or 'snorting' powders - even so called 'legal highs' - could cause you serious harm. They can even kill.

Mixing MCAT or ketamine with other drugs, including alcohol, increases the risk.

Some people are having serious problems with MCAT or ketamine.

LifeLine: Ktikses can talk to you confidentially if you are worried about your drug or alcohol use.
Kratom

stimulant
Synthetic Cathinones
Synthetic cathinones

- 2009 – Western Europe
- 2010 – First appeared in U.S.
  - December Poison Center Calls
- 2011 – Drastic increase due to media
- 2014 – Peak incidence likely passed
Synthetic cathinones

• Sold over Internet
• Gas stations
• Convenience stores
• Head shops
• Smoke shops

• Labeled “not for human consumption”
  – Circumvent drug abuse legislation
Synthetic cathinones aka

Bath salts, bloom, blue silk, chare+, hurricane Charlie, ivory snow, ivory wave, lunar wave, ocean burst, ocean snow, pure ivory, purple wave, red dove, scarface, sextacy, snow leopard, stardust, vanilla sky, white dove, white knight, white lightning, white rush, zoom.
Cathinones

- Leaves and stems of khat plant
- Slow growing shrub native to Ethiopia, East Africa, SW Arabian Peninsula
- Practice of chewing the leaves
- Psychostimulant and euphoric effect
- Used since 11th century
- 10 million daily khat users worldwide
Cathinone

- Stimulant
  - Central nervous system
- Increased heart rate
- Increased blood pressure
- Euphoria
- Alertness
- Psychomotor hyperactivity
Cathinone

- Hypertension
- Myocardial infarction
- Acute coronary vasospasm
- Esophagitis
- Gastritis
- Liver toxicity
- Insomnia
- Depression
- Anorexia
- Psychosis
- Impaired memory
- Withdrawal
- Cerebral edema
- Brainstem herniation
Synthesis of Cathinone Derivatives

- 1920s
  - Methcathinone
  - Mephedrone
- Bupropion
  - Only cathinone derivative that carries medical indication
- First mention internet forms 2007
- # of different cathinone derivatives is emence and ever growing
Schedule I Controlled Substance

• 3 most popular cathinones
  – MDPV
    • U.S.
  – Methylone
  – Mephedrone
    • Europe

• Synthetic Drug Abuse Prevention Act 2012
  – “any material, compound, mixture, or preparation which contains specified cannabimimetic agents (salts, isomer, or salts of isomers)”
  – “specified additional hallucinogenic substances”
  – Verbiage broadens spectrums of substances classified under Schedule I
Patterns & Method of Abuse -- Cathinones

- 2010 – 310 calls
- 2011 6138 calls
- 2012 2363 call
- Exposures reported in children to 6 years-old
- United Kingdom 20% of high school and college students report using mephedrone at least once
- Commonly nasally insufflated or ingested
  - Keying – snort off key
  - Bombing – wrapped in cigarette paper and orally ingested
  - Less common – gingival, rectal, IM, IV
Synthetic Cathinone Treatment

- Supportive care
- Sympton control
- IVF
- Benzodiazepines
- Oxygen
- Sedatives
- Correct hyponatremia slowly with hypertonic saline to avoid central pontine myelinlysis
- Surgical debridement if necrotizing fasciitis at IM site
Synthetic Cannabinoids
Synthetic Cannabinoids

- Herbal marijuana alternative
- Labeled Spice or K2
- U.S. 2008
- Marketed as incense or potpourri
- Labeled “not for human consumption” to bypass legislation
- No ingredients listed on packaging
- Bay bean, blue lotus, dwarf scullcap, honey, Indian warrior, lion’s-tail, lousewort, maconha brava, marshmallow, pink lotus, red clover, rose, Siberian motherwort, and vanilla
Synthetic Cannabinoid Consumption

• Smoked
  – Similar to marijuana
    • Euphoria
    • Relaxation

• Sprayed no plant
Synthetic Cannabinoid Street Names

Spice, K2, albino rhino buds, aroma, barely legal, black mamba, bliss, Damiana, drolle, exclusive cherry, exclusive mint, galaxy, genie, gorilla, halo, k2 summit, krypto buds, red magic, sence, skunk, solar flare, space, spice diamond, spice gold, spice silver, star fire, tai fun, Yucatan fire, zohai, and many more

$40 per 3-g packet
Adverse effects of synthetic cannabinoids

- Tachycardia
- Agitation
- Irritability
- Anxiety
- Hallucinations
- Nausea
- Vomiting
- Hypertension
- Confusion
- Conjunctival injection
- xerostomia
- Seizure
- Psychosis
- Supraventricular tachycardia
  - cardioversion
Poison center calls synthetic cannabinoids

- 2010 – 2906 calls
- 2011 – 6959 calls
- 2012 – 4460 calls

- Texas: 40% of users <20 years-old
- 35% of samples from juvenile probation centers in 2010 tested positive for synthetic cannabinoids
- 2010 classified as Schedule I substance
  - JWH-018, JWH-073, JWH-200, CP-47,497, cannabicyclohexanol
Synthetic Cannabinoid Tx

- Supportive care
- Monitor Vitals
- Benzos
  - Seizure
  - Agitation
  - Anxiety
- Antipsychotic medications
  - Psychosis
  - Behavioral symptoms
Not all emerging toxins are novel and synthetic.
Salvia divinorum
Salvia divinorum

- Herb native to Mexico
- Used for centuries
- Mind-altering
- Potent visionary effects
- Active component is salvinorin A
  - Hallucinogenic properties
Salvia divinorum street names

Sally D, diviner’s sage, Maria Pastora, SkaPastora, magic mint, mystic sage, purple sticky, and sage of the seers

• Sold at head shops, gas stations, Internet
SÁLVIA

ORIGEM
É oriunda de uma pequena região em Oaxaca, no México, onde cresce na área montanhosa dos índios Mazatecas.

"ERVA DIVINA"
Além desse nome, a planta também é conhecida por termos mais populares, tais como: Ska (María) Pastora, folha da pastora, menta mágica e sava.

FORMA VENDIDA
A Sálvia divinorum é vendida normalmente em folhas secas ou em forma de extrato.

EFEITOS
Dependendo de peso do corpo, sensibilidade, dose tomada, método de ingestão e potência da sálvia usada, os efeitos variam desde sutis a extremamente fortes.

USO
A maioria das pessoas fuma a sálvia num bongo ou cachimbo, mas os índios mexicanos Mazatecas usavam dois métodos tradicionais: infusão ou mastigar e engolir.

INTERAÇÃO
A sálvia não pode ser considerada uma droga para festas, pois as pessoas geralmente não se interagem quando se encontram sob o efeito da sálvia, mas têm uma experiência alucinogênea mais pessoal.
Salvia divinorum

• Increase in recreational use by adolescents and young adults
  – U.S. Canada, Europe, Japan
• “unique and intense” high, hallucinations, perceptions of bright lights and vivid colors and shapes, “out-of-body experience”
Cinnamon Challenge

- Swallowing a tablespoon of ground cinnamon
- 60 seconds
- Without drinking fluids

- Pneumonitis
- Aspiration
- Ventilation support
YouTube

• 51,100 clips depicting Cinnamon Challenge
• One video viewed >19 million times
• Viewed by 13- to 24- year-olds
  – People taking challenge
  – Associated with greatest need for conformity
Thank You
Initial Assessment: Pupillary Size

• Miosis
  – C cholinergics, clonidine
  – O opiates, organophosphates
  – P phenothiazines, phenobarbital, pilocarpine
  – S sedative-hypnotics
Initial Assessment: Pupillary Size

• Mydriasis
  – A antihistamines
  – A antidepressants
  – A anticholinergics, atropine
  – S sympathomimetics
Initial Assessment: Skin Signs

• Diaphoresis
  – S sympathomimetics
  – O organophosphates
  – A ASA (salicylates)
  – P PCP (phencyclidine)
Antidotes

- opiates \( \Leftrightarrow \) naloxone
- acetaminophen \( \Leftrightarrow \) NAC
- iron \( \Leftrightarrow \) deferoxoxamine
- digoxin \( \Leftrightarrow \) Fab fragments (Digibind®)
- phenothiazines \( \Leftrightarrow \) diphenhydramine cogentin
- organophosphates \( \Leftrightarrow \) atropine pralidoxime
Antidotes

- ethylene glycol, methanol $\Rightarrow$ ethanol
  fomepizole
- nitrates, dapsone $\Rightarrow$ methylene blue
- $\beta$ and Ca$^+$ channel blockers $\Rightarrow$ glucagon
- carbon monoxide $\Rightarrow$ oxygen
- isoniazid $\Rightarrow$ pyridoxine
- cyanide $\Rightarrow$ amyl or sodium nitrite
  sodium thiosulfate
Antidotes

• sulfonylureas $\rightarrow$ glucose
  octreotide
• tricyclic antidepressants $\rightarrow$ $\text{Na}^+ \text{HCO}_3^-$
• crotalid snakebite $\rightarrow$ antivenom
• midazolam $\rightarrow$ flumazenil (WITH CAUTION)
• methemoglobinemia $\rightarrow$ methylene blue
Clinical Clues: Odor

- Bitter almond
  - cyanide
- Acetone
  - isopropyl alcohol, methanol, ASA
- Oil of wintergreen
  - salicylate
- Garlic
  - arsenic, phosphorus, thallium, organophosphates
Clinical Clues: Skin

• Cyanosis
  – methemoglobinemia secondary to nitrites, nitrates, phenacetin, benzocaine

• Red flush
  – carbon monoxide, cyanide, boric acid, anticholinergics
Clinical Clues: Skin

• Sweating
  – amphetamines, LSD, organophosphates, cocaine, barbiturates

• Dry
  – anticholinergics
Clinical Clues: Mucous Membranes

- **Dry**
  - anticholinergics
- **Salivation**
  - organophosphates, carbamates
- **Oral lesions**
  - corrosives, paraquat
- **Lacrimation**
  - caustics, organophosphates, irritant gases
Clinical Clues: Temperature

• Hypothermia
  – sedative hypnotics, ethanol, carbon monoxide, clonidine, phenothiazines, TCAs

• Hyperthermia
  – anticholinergics, salicylates, phenothiazines, cocaine, TCAs, amphetamines, theophylline
Clinical Clues: Blood Pressure

• Hypertension
  – sympathomimetics (including phenylpropanolamine in OTC cold meds), organophosphates, amphetamines, phencyclidine, cocaine

• Hypotension
  – antihypertensives (including beta and Ca channel blockers, clonidine), barbiturates, benzodiazepines, TCAs
Clinical Clues: Heart Rate

• Bradycardia
  – digitalis, sedative hypnotics, beta blockers, opioids

• Tachycardia
  – anticholinergics, sympathomimetics, amphetamines, alcohol, aspirin, theophylline, cocaine, TCAs

• Arrhythmias
  – anticholinergics, TCAs, organophosphates, digoxin, phenothiazines, beta blockers, carbon monoxide, cyanide
Cinical Clues: Respirations

• Depressed
  – alcohol, opioids, barbiturates, sedative-hypnotics, TCAs, paralytic shellfish poison

• Tachypnea
  – salicylates, amphetamines, carbon monoxide

• Kussmauls
  – methanol, ethylene glycol, salicylates
Clinical Clues: CNS

• Seizures
  – carbon monoxide, cocaine, amphetamines and sympathomimetics, anticholinergics, aspirin, pesticides, organophosphates, lead, PCP, phenothiazines, INH, lithium, theophylline, TCAs

• Miosis
  – opioids, phenothiazines, organophosphates, benzodiazepines, barbiturates, mushrooms, PCP
Clinical Clues: CNS

• Mydriasis
  – anticholinergics, sympathomimetics, TCAs, methanol

• Blindness
  – methanol

• Fasciculations
  – organophosphates
Clinical Clues: CNS

• Nystagmus
  – barbiturates, carbamazepine, PCP, carbon monoxide, ethanol

• Hypertonia
  – anticholinergics, phenothiazines

• Myoclonus/rigidity
  – anticholinergics, phenothiazines, haloperidol
Clinical Clues: CNS

• Delirium/psychosis
  – anticholinergics, sympathomimetics, alcohol, phenothiazines, PCP, LSD, marijuana, cocaine, heroin, heavy metals

• Coma
  – alcohols, anticholinergics, sedative hypnotics, opioids, carbon monoxide, TCAs, salicylates, organophosphates

• Weakness/paralysis
  – organophosphates, carbamates, heavy metals
Thank You