Learning Objectives

• Define poisoning.
• Describe general principles for assessment and management of the patient who has ingested poison.
• Describe the causative agents and pathophysiology of selected ingested poisons and management of patients who have taken them.

Learning Objectives

• Describe how physical and chemical properties influence the effects of inhaled toxins.
• Distinguish among the three categories of inhaled toxins: simple asphyxiants, chemical asphyxiants and systemic poisons, and irritants or corrosives.
• Describe general principles of managing the patient who has inhaled poison.

Poisonings

• Any substance that produces harmful physiological or psychological effects
• Emergencies involving poison are major cause of morbidity and mortality in U.S.
  - In 2006, over 2 million unintentional poisonings reported by poison control centers
    • Resulted in over 700,000 emergency department visits
  - Additional 200,000 emergency department visits caused by intentional poisoning
    • 75 percent of these had to be hospitalized or transferred to another medical facility
Statistics

- Poisoning by solids and liquids second leading cause of unintentional death in U.S.
  - Second leading cause of unintentional death for all persons aged 17 to 39.2 (2005, National Safety Council)

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How many substances that fit the definition of a poison are there in or around your home?

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Poison Control Centers

- Over 60 poison control centers across U.S.
  - Most based in major medical centers or teaching hospitals
  - Belong to regional centers designated by American Association of Poison Control Centers
Poison Control Centers

• 2007 statistics
  – Estimated 4.2 million poisonings reported to poison control centers throughout U.S.
  – Over 90 percent of poisonings happen in home
  – 51.2 percent of poisoning victims are children under age 6
  – Centers prevent about 1.6 million hospitalizations and doctors' office visits per year by helping people manage emergencies at home

Poison Control Centers

• Staffed by medical professionals
  – 24-hour telephone access (1-800-222-1222) to population bases of at least 1 million
  – Give immediate information and treatment advice

Poison Control Centers

• Large database of 350,000 toxic substances
  – Drugs
  – Chemicals
  – Plants
  – Animals
  – Insects
  – Fish
  – Snakes
  – Cosmetics
  – Hazardous materials
  – Each request followed up to determine treatment's effectiveness and outcome
Poison Control Centers

- Elements of organized poison system
  - Treatment information and toxicological consultation with health care providers and public
    • Toll-free number with linkage into various 911 systems
  - Professional education to train those involved in care of poisoned patients
  - Data collection on all poisonings in region for epidemiological and evaluation purposes

Poison Control Centers

- Elements of organized poison system
  - Public education and prevention
  - Research
  - Regional EMS poison system development
    • Patient classification criteria
    • Triage and management protocols
    • Regional transfer agreements

Use by EMS Agencies

- Regional poison control centers are ready resource in any toxicological emergency
- Method of contacting poison control centers depends on local communications protocol
  - Directly by EMS and other public service agencies
    • Telephone, cellular phone
    • Dispatching center
    • Medical direction
Use by EMS Agencies

- Immediate determination of potential toxicity
  - Based on specific agent or agents
  - Amount ingested
  - Time of exposure
  - Patient's weight and medical condition
  - Any treatment given before EMS arrival
  - Poison center can coordinate by notifying receiving hospital while patient is en route to emergency department

General Guidelines for Managing Poisoned Patient

- Poison may enter body through
  - Ingestion
  - Inhalation
  - Injection
  - Absorption
- Most patients require only supportive therapy to recover
  - Some specific poisons require lifesaving antidotes

General Guidelines for Managing Poisoned Patient

- Guidelines
  - Ensure adequate airway, ventilation, and circulation
    - Prevent or reduce risk of aspiration by carefully monitoring airway
  - Obtain thorough history, perform focused physical examination
  - Consider hypoglycemia in patient with altered level of consciousness or convulsions
    - Confirm through serum glucose testing
  - Administer naloxone or nalmefene to patient with respiratory depression
General Guidelines for Managing Poisoned Patient

• Guidelines
  – If overdose suspected, obtain overdose history from patient, family, or friends
  – Consult with medical direction or poison control center for specific management to prevent further absorption of
    • Toxin
    • Antidote therapy
  – Frequently reassess
    • Monitor patient’s vital signs and ECG

General Guidelines for Managing Poisoned Patient

• Guidelines
  – Safely obtain any substance or substance container of a suspected poison
    • Transport along with patient
    • Collect sample of patient’s vomitus (if present) for lab analysis
  – Employ universal precautions for personal protection, especially if substance can be absorbed through skin
  – Transport patient for physician examination

General Guidelines for Managing Poisoned Patient

• Personal safety is top priority
  – Toxicological emergency response
    • May involve hazardous materials
    • Unpredictable or violent patient behavior
  – If scene is not safe
    • Retreat to safe staging area
    • Wait there until scene is secured by proper personnel
Poisoning by Ingestion

- 80 percent of all accidental ingestion occurs among children 1 to 3 years old
  - Most common poison are household products
    - Petroleum-based agents
    - Cleaning agents
    - Cosmetics
    - Medications
    - Toxic plants
    - Contaminated foods

Poisoning by Ingestion

- Poisoning in adults usually intentional
  - Suicide attempts
  - Recreational or experimental drug use
  - Chemical warfare or acts of terrorism
  - Assault and homicide

Poisoning by Ingestion

- Toxic effects of ingested poisons
- Immediate
  - Corrosive substances may produce immediate tissue damage
    - Strong acids
    - Alkalis
  - Evidenced by burns to lips, tongue, throat, upper gastrointestinal tract
Poisoning by Ingestion

- Delayed
  - Usually require absorption and distribution through bloodstream
    - Medications
    - Toxic plants
  - May require alterations by different organs to produce toxic effects
  - Minimal absorption in stomach
    - Poisons may take several hours to enter bloodstream through small intestine

Assessment and Management

- Begin with ensuring scene safety
  - Then manage immediate threats to patient’s life
- Scene "size up"
  - Be alert for clues/details suggesting toxicological emergency
    - Open medication bottles
    - Scattered pills
    - Vomitus
    - Open containers of household products

Assessment and Management

- Patient findings
  - Decreased level of consciousness
  - Airway compromise/injury
    - Vomitus or pills in the mouth
    - Burns in oral cavity
  - Abnormal respiratory patterns
  - Dysrhythmias
    - Tachycardia
    - Bradycardia
Assessment and Management

• Primary goal
  – Identify effects on three vital organ systems most likely to produce morbidity or death
    • Respiratory
    • Cardiovascular
    • Central nervous

Assessment and Management

• Detailed history
  – Helps direct treatment in field or emergency department
  – Toxic ingestion may worsen these
    • Preexisting cardiac, liver, or renal disease
    • Some psychiatric illnesses
  – These may require care in addition to managing toxic ingestion

Respiratory Complications

• First priority after scene safety
  – Secure patient airway
  – Provide adequate ventilatory support as needed
    • High-concentration O₂
    • Monitoring pulse oximetry
    • Possibly advanced airway management to protect airway and prevent aspiration
Respiratory Complications

- Other respiratory complications
  - Early development of noncardiogenic pulmonary edema
  - Later development of adult respiratory distress syndrome
  - Bronchospasm from direct or indirect toxic effects

Cardiovascular Complications

- Most common
  - Cardiac dysrhythmias
    - Assess patient’s circulatory status
    - Continuously monitor by ECG and frequent BP measurements
  - Tachydysrhythmias or bradydysrhythmias may indicate serious metabolic disorders: hypoxia, acidosis
  - Hypotension associated with decreased vascular tone

Neurological Complications

- Perform and document baseline neurological examination
  - Range of deviations from normal level of consciousness
    - Mild drowsiness and agitation
    - Hallucinations, seizures, coma, death
  - Complications may result from toxin itself
    - Example: lead poisoning in children who have ingested paint chips
  - Or may result from metabolic or perfusion disorder
    - Example: poor cardiac output from dysrhythmias
**History**

- Obtain thorough history of exposure, significant medical history from patient, family members, bystanders
  - What was ingested?
    - Obtain poison container, remaining contents unless this poses threat to rescuer safety
  - When was/were substance(s) ingested?
    - May affect decision to use activated charcoal or gastric lavage, or administer antidote

**History**

- Obtain thorough history of exposure, significant medical history from patient, family members, bystanders
  - How much was ingested?
  - Was attempt made to induce vomiting?
    - Did patient vomit?
  - Has antidote or activated charcoal been administered?
  - Does patient have psychiatric history pertinent to suicide attempts?
    - Episodes of recent depression?

**Gastrointestinal Decontamination**

- Medical methods to empty stomach of ingested toxins to prevent absorption
  - Activated charcoal
  - Gastric lavage
  - Whole-bowel irrigation
- Consult with medical direction or poison control center before attempting to remove poison from GI tract
Activated Charcoal

- Inert, nontoxic product of wood material
  - Heated to extremely high temperature
  - Surface characteristics enable to adsorb molecules of chemical toxins in intestinal tract
  - Indicated for some toxic ingestions or for drugs that have delayed emptying

Activated Charcoal

- Should not be given when
  - Toxicant is strong acid, strong alkali, or ethanol
  - Specific oral antidotes available
- Comes mixed in aqueous solution with or without cathartic (most commonly sorbitol)
  - Agent that causes bowel evacuation
  - Decreases transit time and expels charcoal within short period

Activated Charcoal

- Complications
  - Poor patient acceptance in consuming it
  - Vomiting
- Protect yourself, patient, and immediate area from charcoal's staining properties
  - Take personal protective measures when administering this agent
Why might a patient be reluctant to take activated charcoal?

Gastric Lavage

• Can immediately recover portion of gastric contents
  – Rarely performed in prehospital setting
  – Sometimes used in emergency department when highly toxic substance ingested within past hour
• Procedure
  – Insertion of large-bore orogastric tube through patient’s mouth into stomach
  – Instilling normal saline through tube, lavaging stomach of gastric contents until fluid returned is clear

Gastric Lavage

• Patients cannot protect their airway and have altered level of consciousness
• Patients have ingested low-viscosity hydrocarbons where risk of aspiration increases
  – Gasoline
  – Kerosene
  – Furniture polish
  – Mineral spirits
Whole-Bowel Irrigation

- In-hospital method of GI decontamination
  - Rapid administration of large amounts of specially balanced fluid to flush GI tract
  - Administered orally or via nasogastric tube
  - Useful in cases of
    - Severe, recent ingestion of lithium or metals, such as iron or lead
    - Ingestion of large amounts of sustained-release formulations of highly toxic drugs
    - Evacuating drug packets from “body packers”

Antidotes

- Agent used to neutralize or counteract effects of specific poisons
  - Most given under physician supervision in hospital setting
  - Work by
    - Increasing elimination of toxin
    - Reactivating enzymes altered by poison
  - Few effective ones available for ingested poisons
    - Makes managing symptoms main goal in caring for poisoned patient

Strong Acids and Alkalis

- Found in toilet bowl cleaners, rust remover, ammonia, most liquid drain cleaners
- Frequency of caustic ingestions highest in small children
  - 5,000 to 8,000 accidental exposures each year
  - Lye most commonly ingested substance
Strong Acids and Alkalis

• May cause burns to
  – Mouth
  – Pharynx
  – Esophagus
  – Upper respiratory and GI tracts (sometimes)

• Perforation of esophagus or stomach may result in
  – Vascular collapse
  – Mediastinitis (inflammation of mediastinum)
  – Pneumoperitoneum (gas in peritoneal cavity of abdomen)

• Generally produces immediate damage to mucous membrane and intestinal tract
• Acid damage generally completed within 1 to 2 minutes

• Alkalis damage may continue for hours
  – Cause liquefaction of tissue
    • Liquefaction: conversion of solid tissue to fluid or semifluid state
  – Prehospital care for alkali ingestion
    • Usually limited to airway and ventilatory support
    • IV fluid replacement
    • Rapid transport
Strong Acids and Alkalis

• Medical direction or poison control may recommend diluting acid or alkali if patient is conscious
  – Via oral administration of milk or water
    • 200 to 300 mL for an adult
    • 15 mL/kg maximum for child

• Contraindications: Do not try to neutralize ingested agent with other fluids
  – Fruit juice, lemon juice, vinegar are contraindicated
  – These fluids have potential to cause severe thermal burns

What is a risk of administering milk or water to a patient with acid or alkali ingestion?
Hydrocarbons

- Group of compounds derived mainly from crude oil, coal, or plant sources
- Vary in their viscosity, surface tension, and volatility and determine toxic effects of agents
  - Viscosity: resistance of liquid to flow
  - Surface tension: ability of liquid to be attracted to another surface
  - Volatility: ability of liquid to vaporize

Hydrocarbons

- Other contributing factors
  - Presence of other chemicals in product
  - Total amount
  - Route of exposure

Hydrocarbons

- Found in many household products
  - Cleaning and polishing agents
  - Spot removers
  - Paints
  - Cosmetics
  - Pesticides
  - Hobby and craft materials
  - Baby oil
    - Particularly dangerous if ingested
Hydrocarbons

- Also found in
  - Petroleum distillates
    - Turpentine
    - Kerosene
    - Gasoline
    - Lighter fluids
    - Pine oil products
  - Large group of halogenated hydrocarbons and aromatic hydrocarbons exist

Hydrocarbons

- Halogenated hydrocarbons
  - Carbon tetrachloride
  - Trichloromethane
  - Trichloroethylene
  - Methyl chloride
- Aromatic hydrocarbons
  - Toluene
  - Xylene
  - Benzene

Hydrocarbons

- Hydrocarbon poisonings are common
  - Account for 7 percent of all ingestions in children under 5 years of age
  - Most ingestions occur between May and September
    - Home use of petroleum products allows children the greatest opportunity for exposure (e.g., cleaning and yard machinery)
Hydrocarbons

• Viscosity
  – Most important physical characteristic in potential toxicity of ingested hydrocarbons
  – Lower viscosity = higher risk of aspiration and associated complications
  – Products with low viscosity spread rapidly over surface of mouth and throat
    • Gasoline
    • Turpentine

• Viscosity
  – More volatile components become gases on contact with warm mucous membranes
  – Exposure causes irritation, coughing, possible aspiration
    • Aspiration may allow toxic amount of hydrocarbons to enter lungs
  – Products with high viscosity are not aspirated or absorbed in GI tract, thus do not have significant toxicity

• Clinical features
  – Vary widely, depending on type of agent involved
  – May involve patient’s respiratory, gastrointestinal, and neurological systems
  – Clinical features may be immediate or delayed
Hydrocarbons

• Patient not displaying symptoms on EMS arrival
  – Chances of serious complications usually low
  • Patient generally observed in emergency department for several hours
  • Often requires no treatment
• Patient who coughs, chokes, cries, has spontaneous emesis on swallowing
  – Assume to have aspirated hydrocarbon until proved otherwise

Hydrocarbons

• Emergency care for symptomatic patients with ingestion
  – Ensure patent airway
  • Provide adequate ventilatory and circulatory support as needed
  – Identify substance
  • Contact medical direction or poison control center
  – Decontamination of stomach generally avoided due to risk of aspiration

Hydrocarbons

• Emergency care for symptomatic patients with ingestion
  – Activated charcoal or diluents not shown effective in managing hydrocarbon ingestion
  – Initiate intravenous fluid therapy
  – Monitor cardiac rhythm
  – Transport patient for physician evaluation
Will the potential lethal effects of this ingestion (hydrocarbons) always be visible on the scene?

Methanol

- Poisonous alcohol found in various products
  - Gas line antifreeze
  - Windshield washer fluid
  - Paints
  - Paint removers
  - Varnishes
  - Canned fuels such as Sterno
  - Many shellacs

Methanol

- Colorless liquid
- Odor distinct from ethanol (alcohol in alcoholic beverages)
Methanol

- Poisonings may result from
  - Intentional or unintentional ingestions
  - Absorption through skin
  - Inhalation
- Examples
  - Deliberate use by chronic alcoholics to maintain inebriated state
  - Unintentional ingestion from misuse or distribution of methanol for ethanol
  - Accidental ingestions in children

Methanol

- Metabolites of methanol
  - Extremely toxic
  - During absorption
    - Liver rapidly converts methanol to formaldehyde then to formic acid
- Formic acid in blood affects
  - Central nervous system
    - Lethargy
    - Confusion
    - Seizure

Methanol

- Formic acid in blood affects
  - GI tract
    - Abdominal pain
    - Nausea
    - Vomiting
  - Leads to development of metabolic acidosis
    - Shock
    - Multisystem failure
    - Death
Methanol

• Formic acid in blood affects
  – Patient’s vision
    • Blurred vision
    • Photophobia
  – Blindness
    • Caused by ingestion of as little as 4 mL
• Symptoms of methanol poisoning correlate with degree of acidosis
  – Onset after ingestion ranges from 40 minutes to 72 hours

Do you think this (methanol ingestion) could have been the origin of the expression “blind drunk”?

Methanol Emergency Care

• Supportive care
  – Secure patent airway and monitor pulse oximetry
  – Provide adequate ventilatory and circulatory support as needed
  – Adequate ventilation essential
    • Ensures adequate oxygenation
    • Helps correct profound metabolic acidosis
    • Maximizes respiratory excretion
  – Establish intravenous line
    • Place patient on cardiac monitor to detect rhythm disturbances
Methanol Emergency Care

• Gastrointestinal decontamination
  – If patient seen within 1 hour after ingestion, gastric lavage may be indicated
  – Activated charcoal is ineffective and should not be given
  – Consult medical direction or a poison control center

Methanol Emergency Care

• Correction of metabolic acidosis
  – Medical direction may recommend attempt to correct with sodium bicarbonate
    • Large or repeated doses may be necessary
    • Serum formic acid may be neutralized with bicarbonate administration
    • Hemodialysis likely necessary to remove toxic levels of methanol and formate

Methanol Emergency Care

• Prevent conversion of methanol to formic acid
  – May be prevented by the administration of ethanol
  – Ethanol has nine times greater affinity for enzyme that converts methanol to formic acid
  – If authorized by on-line medical direction or protocol, give conscious patient 30 to 60 mL of 80-proof ethanol by mouth or gastric lavage tube
  – Unconscious patients should have airway protected with endotracheal tube before gastric tube administration of ethanol
  – Transport patient rapidly to proper medical facility for definitive treatment
Ethylene Glycol

- Colorless, odorless, water-soluble liquid
- Commonly used in
  - Windshield deicers
  - Detergents
  - Paints
  - Radiator antifreeze
  - Coolants

Ethylene Glycol

- Accidental ingestion common in young children
  - Brilliant colors added to these preparations attract children
  - Widespread availability of products containing ethylene glycol
  - Warm, sweet taste
- Sometimes consumed by alcoholics as substitute for ethanol
- Without treatment, ingestion of 0.2 mL/kg reported to be lethal in adults

Ethylene Glycol

- Early symptoms of CNS depression caused by ethanol-like effects of ethylene glycol
- Toxicity caused by glycolic and oxalic acids accumulated after metabolism
  - Occurs primarily in liver and kidneys
- Metabolic molecules may affect
  - CNS
  - Cardiopulmonary system
  - Renal systems
Ethylene Glycol

- Initial signs and symptoms
  - Slurred speech
  - Nausea and vomiting
  - Hallucinations
  - Seizure
  - Stupor
  - Coma
  - Can be followed by pulmonary edema and cardiac failure

Ethylene Glycol

- Emergency care
  - Similar to that for methanol poisoning
  - Anticipate orders from medical direction or poison control center for administration of
  - Thiamine
    - Degrade glycolic acid to nontoxic metabolites
  - Calcium gluconate or calcium chloride
    - Manage hypocalcemia
  - Diazepam or lorazepam
    - Control seizure activity

For what could the effects in stage one of ethylene glycol poisoning be mistaken?
Isopropanol

- Volatile, flammable, colorless liquid
  - Has characteristic odor and bittersweet taste
  - Rubbing alcohol is most common household source
- Also is used in
  - Disinfectants
  - Degreasers
  - Cosmetics
  - Industrial solvents
  - Cleaning agents

Isopropanol

- Common routes of toxic exposure
  - Intentional ingestion as substitute for ethanol
  - Accidental ingestion
  - Inhalation of high concentrations of local vapor
- More toxic than ethanol, less toxic than methanol or ethylene glycol
- Lethal dose in adults rare
- Consider any amount of ingestion in children potentially toxic

Isopropanol

- Body systems affected
  - CNS
  - GI
  - Renal
Isopropanol

- Signs and symptoms often occur within 30 minutes
  - CNS and respiratory depression
  - Abdominal pain
  - Gastritis
  - Hematemesis
  - Hypovolemia
- Causes acids to accumulate in blood (acetonemia) and ketones to accumulate in urine (ketonuria)
  - Metabolic acidosis doesn’t usually occur unless patient develops hypotension

Emergency Care for Isopropanol Poisoning

- Emergency care
  - Mainly supportive
  - Airway and ventilatory support
    - Ensures adequate respiratory elimination of acetone
  - Fluid resuscitation as needed
  - Rapid transport to appropriate medical facility, where dialysis may be necessary
  - Gastric lavage and activated charcoal not effective
  - Ethanol not proven to inhibit toxic metabolite to same degree as in methanol or ethylene glycol poisoning

Metals

- Infants and children are high-risk groups for unintentional poisoning
  - Iron
  - Lead
  - Mercury poisoning
- Immature immune systems, increased absorption as function of age contribute to risk
Iron Poisoning

• 10 percent of ingested iron (mainly ferrous sulfate) absorbed daily from small intestine
  – Then converted and stored in iron storage protein
  – Then transported to liver, spleen, and bone marrow for incorporation into hemoglobin
  – When ingested iron is greater than body’s ability to store, free iron circulates in blood
    • Then deposited into other tissues

• Most result from ingestion of pediatric multivitamins by children under 6 years old

Iron Poisoning

• Unintentional or intentional ingestion may be fatal
  – Ingested iron corrosive to GI tract lining
  – Iron may produce
    • Gastrointestinal hemorrhage
    • Bloody vomitus
    • Painless bloody diarrhea
    • Dark stools
  – Severe cases involve ingestion of over 20 mg/kg
  – Ingestion of over 60 mg/kg can produce cardiovascular collapse and death

Iron Poisoning

• Prehospital care
  – Supportive measures and rapid transport for physician evaluation
  – Possible gastric decontamination to prevent further absorption
  – Activated charcoal generally not recommended (absorbs iron poorly)
  – Most patients survive episode
  – Long-term prognosis favorable
Lead Poisoning

• 1978, lead based paint recognized as major health hazard
  – Banned from household paints in U.S.
• Children most common victims
  – ≈ 250,000 children in U.S. have levels of lead in bloodstream of 10 mg/dL or greater
  – Most pediatric poisonings from ingesting lead-based paint chips and contaminated house dust

Lead Poisoning

• Children with high levels of lead can suffer from damage to brain and nervous system
  – Behavioral and learning problems
  – Hyperactivity
  – Slowed growth
  – Hearing problems
  – Headaches
• Children who appear healthy can have dangerous levels of lead

Lead Poisoning

• Adults with high levels of lead
  – Difficult pregnancies
  – Reproductive problems
  – Hypertension
  – Nerve disorders
  – Muscle and joint pain
  – Problems with memory and concentration
Lead Poisoning

• Toxicity in adults most commonly from exposure by inhalation
• Most poisoning from lead slow in onset
• Results from chronic ingestion or inhalation, eventually resulting in toxicity

Lead Poisoning

• Body excretes it slowly
  – Tends to accumulate in tissues, mainly bone
• Exposure resulting in acute intoxication may result in
  – Paralysis
  – Seizures
  – Coma
  – Death
• Survivors likely to sustain brain damage

Lead Poisoning

• Prehospital care
  – Focused on recognizing potential for lead poisoning
  – Transporting patient for physician evaluation
• If confirmed through x-ray and lab tests, care may include
  – Gastric decontamination
  – Whole-bowel irrigation
Lead Poisoning

• After treatment
  – Patients must be discharged to lead-free environment
  – Outpatient chelation therapy indicated to detoxify lead and excrete it from body

Paramedics play a key role in the emergency management of lead poisoning. What other role can they play in the management of this problem?

Mercury Poisoning

• Only metal liquid at room temperature
  – Used in
    • Thermometers
    • Sphygmomanometers
    • Dental fillings
Mercury Poisoning

• Various mercury compounds used in
  – Some paints
  – Pesticides
  – Cosmetics
  – Drugs
  – Certain industrial processes
• All forms except those in dental fillings are poisonous

Mercury Poisoning

• Inhalation of vapor most common route of mercury poisoning
  – May cause shortness of breath and lung damage
  – May be absorbed through skin, causing severe inflammation
  – Through intestines after ingestion
  – Passes into bloodstream after entering body
  – Accumulates in various organs, mainly brain and kidneys

Mercury Poisoning

• Symptoms
  – Malaise
  – Incoordination
  – Excitability
  – Tremors
  – Numbness in the limbs
  – Vision impairment
  – Nausea and emesis (symptoms of renal failure)
  – Mental status changes
Mercury Poisoning

- Prehospital care
  - Mainly supportive
  - Following physician evaluation, patients managed with
    - Gastrointestinal decontamination if recent ingestion
    - Chelating agents
    - Severe cases may require hemodialysis

Food Poisoning

- Any sudden-onset illness caused by food eaten within previous 48 hours
  - Usually associated with stomach pain, vomiting, and diarrhea
  - Classifications
    - Infectious (bacterial and viral)
    - Noninfectious

Infectious (Bacterial) Types

- One of most common
  - Salmonella
    - Organism found in
      - Animals (especially poultry)
      - Human beings
  - Salmonella bacteria may be transferred to food
    - From excrement of infected animals or humans
    - By infected person handling food
### Infectious (Bacterial) Types

- Other bacteria cause formation of toxins
  - May be difficult to destroy even with thorough cooking
- Other common bacterial causes of diarrhea
  - *Escherichia coli* (traveler’s diarrhea)
  - *Campylobacter* and *Shigella* organisms

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### Infectious (Bacterial) Types

- **Botulism**
  - Rare but life-threatening form of food poisoning
  - May result from eating improperly canned or preserved food contaminated with *Clostridium botulinum*

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### Infectious (Bacterial) Types

- **Botulism**
  - Organism found in soil and untreated water in most parts of world
  - Also harmlessly present in intestinal tracts of many animals, including fish
  - Spore-forming properties resist boiling, salting, smoking, and some forms of pickling
  - Bacterium thrives in improperly preserved or canned foods
  - Food-borne botulism rare (more common in U.S. due to home food preserving)
Infectious (Bacterial) Types

- Botulism associated with severe CNS symptoms
  - Symptoms appear in head-to-toe progression
    - Headache
    - Blurred or double vision
    - Dysphagia
    - Respiratory paralysis
    - Quadriplegia
  - Respiratory failure occurs in 50 percent of patients
  - Death rare with treatment

Infectious (Viral) Types of Food Poisoning

- Viruses that most often cause food poisoning
  - Norwalk (common contaminant of shellfish)
  - Rotavirus
- Both may cause illness when raw/partly cooked foodstuffs have contacted water contaminated by human excrement

Noninfectious Types

- May result from
  - Consuming mushrooms and toadstools
  - Eating fresh foods and vegetables accidentally contaminated with large amounts of insecticide
- Chemical food poisoning may result from eating food stored in contaminated container
  - Container previously used to store poison
  - Also can result from improperly preparing and cooking various exotic foods
### Management Guidelines

- **Factors affecting onset of signs and symptoms**
  - Cause of poisoning
  - Level of food's contamination

- **Development of symptoms**
  - Within 30 minutes for chemical poisoning
  - In 1 to 12 hours for bacterial toxins
  - In 12 to 48 hours with viral and bacterial infections

- **Avoid contamination of self and equipment**
  - Wear gloves, gown, or both if appropriate

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### Management Guidelines

- **Treatment**
  - Ensure adequate airway, ventilatory, and circulatory support
  - Gather complete history
    - Time and onset of symptoms
    - Recent travel
    - Relation of symptoms to ingestion of particular food
    - Effects on others who ate same food
    - Information on consistency, frequency, odor of stool (plus presence of mucus or blood)
    - Note fever
    - Include significant medical history, allergies, medications

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### Management Guidelines

- **Treatment**
  - Initiate IV therapy with crystalloid solution
    - Helps manage dehydration and electrolyte disturbances from vomiting and diarrhea
  - Transport patient for physician evaluation
Plant Poisoning

- Toxic plant ingestion frequently reported category of poisonings
  - Second only to ingestion of cleaning substances
- Majority of exposures occur in children under 6 years old

What features of a plant would make it attractive for children to eat?

Plant Poisoning

- Signs and symptoms
  - Signs of toxicity are predictable
  - Categorized by plant's chemical and physical properties
  - Most tend to be consistent with type of major toxic chemical component in plant
  - Symptoms usually appear within several hours, but may be delayed 1 to 3 days
  - Paramedics should be familiar with common poisonous plant life in their response area
Management
• In U.S., hundreds of green plant species and over 100 mushroom varieties have toxic compounds
  – Have widely varying potencies and combinations of toxins
  – Factors dictating severity of toxic symptoms
    • Age of plant
    • Soil conditions

Management
• Management guidelines should be customized to patient’s symptoms, not type of ingestion
  – Identification of plant important if possible
    • Inability to ID should not delay patient care
  – Consult medical direction or poison control center

Management
• Includes
  – GI decontamination
  – Activated charcoal in conscious patients
  – IV fluids
  – Ensure adequate airway, ventilatory, and circulatory support
  – Most patients hospitalized for observation and treatment as indicated for toxin involved
  – Dialysis not shown effective in removing most plant toxins
Poisoning by Inhalation

• Unintentional or intentional inhalation of poisons can lead to life-threatening emergency
• Type and location of injury depend on specific actions and behaviors of chemical involved
• Respiratory difficulty may not appear for several hours after exposure
• Encourage all patients to be evaluated by physician, including asymptomatic patients

Do you think that situations involving toxic gas inhalation are likely to involve single patients or multiple patients? Why?

Classifications

• Categories
  – Simple asphyxiants: methane, propane, inert gases
    • Cause toxicity by displacing or lowering amount of O₂ in air
  – Chemical asphyxiants (e.g., CO₂, cyanide)
    • Cause number of local and pulmonary reactions
    • Toxic systemic effects prevent uptake of O₂ by blood
    • Can interfere with tissue oxygenation
  – Irritants/corrosives (e.g., chlorine, ammonia)
    • Cause cellular destruction and inflammation as they contact moisture
General Management

• Same as for any other hazardous materials incident
  – Scene safety
  – Personal protective measures (clothing, respiratory protective apparatus)
  – Rapid removal of patient from poison environment
  – Surface decontamination

General Management

• Management principles
  – Adequate airway, ventilatory, and circulatory support
  – Initial assessment and physical examination
  – Irrigation of eyes as needed
  – IV line with saline solution
  – Regular monitoring of vital signs, ECG, and pulse oximetry
  – Rapid transport

Lesson 34.2

Cyanide, Ammonia, Hydrocarbon, Insects, Reptiles, and Aquatic Poisoning
Learning Objectives

• Describe the signs, symptoms, and management of patients who have inhaled cyanide, ammonia, or hydrocarbon.
• Describe the signs, symptoms, and management of patients injected with poison by insects, reptiles, and hazardous aquatic creatures.

Cyanide

• Highly toxic substances containing cyanogen chemical group
  – Few applications because of toxicity
  – In electroplating ore extraction
  – Fumigation of buildings
  – As a fertilizer
  – In gas chambers as means of execution
  – Released via combustion of products like nylon and polyurethane
    • A hazard in fire environments

Cyanide

• Poisoning may result from
  – Inhalation of cyanide gas
  – Ingestion of cyanide salts, nitriles, or cyanogenic glycosides
    • Amygdalin: in apple seeds and cherry, pear, and apricot pits
    • Infusion of nitroprusside
  – Absorbed across skin
Cyanide

• Regardless of entry route, rapidly acting poison
• Inhibits cellular oxygenation
  – By combining and reacting with ferric ions (Fe3) of respiratory enzyme cytochrome oxidase
  – Cytotoxic hypoxia produces rapid progression of symptoms
    • Dyspnea to paralysis, unconsciousness, death
    • Large doses usually fatal within minutes from respiratory arrest

Cyanide

• Management
  – Ensure personal safety
  – Secure open airway
  – Provide adequate ventilatory support with high-concentration O2
    • Displaces cyanide from cytochrome oxidase
    • Increases effectiveness of drug administration

Cyanide

• Management
  – One treatment converts (oxidizes) ferrous ions in hemoglobin (Fe2) to ferric ions (Fe3)
    • Result is methemoglobin: hemoglobin with ferrous ion in oxidized (Fe3) state
  – Cyanide, with greater attraction to Fe in ferric state, released from cytochrome oxidase
    • Combines with methemoglobin, allowing cytochrome oxidase to resume function in normal cellular respiration
  – Cyanide antidotes found in Pasadena cyanide antidote kit (Lilly Cyanide Poison Kit) and Taylor Kit thought effective through inducing methemoglobin
Cyanide

- Reconversion of methemoglobin to hemoglobin
  - Methemoglobin cannot transport $O_2$
    • Must be reconverted to hemoglobin by administration of sodium thiosulfate

Cyanide

- Conversion is three-part process
  - Goal of Parts 1 and 2: formation of methemoglobin through
    • Administration of amyl nitrate by inhalation
    • IV sodium nitrite
    • These convert ferrous hemoglobin into methemoglobin
  - Goal of Part 3: detoxify cyanide so it can be eliminated
    • Give IV sodium thiosulfate
    • Provides sulfur to help detoxify cyanide so kidneys can eliminate it

Cyanide

- Prehospital care
  - Don personal protective equipment as needed to help prevent rescuer contamination
  - Remove patient from cyanide source
  - Rapid decontamination and removal of patient's contaminated clothing essential
  - Ensure patent airway, provide adequate ventilatory support
  - Administer high-concentration $O_2$, monitor pulse oximetry
  - Administer cyanide antidote
  - Consult medical direction or poison control center and follow manufacturer's instructions
Cyanide Poisoning

- Prehospital care
  - IV fluid therapy with volume-expanding solution
  - Monitor cardiac rhythm by ECG
  - Rapidly transport
  - Anticipate hypotension as side effect of antidote therapy
    - Keep patient lying down, closely monitor BP
    - If hypotension develops, medical direction may recommend vasopressors

Ammonia Inhalation

- Toxic irritant causes local pulmonary complications after inhalation
  - Complications
    - Inflammation and irritation
    - Destruction of mucosal tissue of all respiratory structures (severe cases)
  - Injury occurs as ammonia vapor combines with H₂O into highly caustic alkaline

Ammonia Inhalation

- Symptoms
  - Coughing
  - Choking
  - Congestion
  - Burning and tightness in chest
  - Feeling of suffocation
    - These symptoms often associated with burning eyes and tearing
  - Bronchospasm and pulmonary edema (severe cases)
Ammonia Inhalation

- Treatment
  - General management principles
  - May also include positive-pressure ventilation
  - Administration of diuretics and bronchodilators

Hydrocarbon Inhalation

- Hydrocarbons posing greatest risk for injury have
  - Low viscosity
  - High volatility
  - High surface tension or adhesion of molecules along surface
    - Characteristics allow hydrocarbons to enter pulmonary tree
    - Causes aspiration pneumonitis
    - Creates potential for systemic effects
    - Examples: CNS depression and liver, kidney, or bone marrow toxicity

- Most result from “recreational use” of halogenated hydrocarbons
  - Carbon tetrachloride
  - Methylene chloride
  - Aromatic hydrocarbons
    - Benzene
    - Toluene
  - Produce inebriation or euphoria through “sniffing” or “huffing”
Hydrocarbon Inhalation

- Effects
  - Onset effects usually rapid, typically within seconds
    - May be followed by CNS depression
    - Respiratory failure
    - Cardiac dysrhythmias

- Signs and symptoms
  - Burning sensation on swallowing
  - Nausea and vomiting
  - Abdominal cramps
  - Weakness
  - Anesthesia
    - Hallucinations
    - Changes in color perception
    - Blindness
    - Seizures
    - Coma

- Emergency care
  - Generally supportive
    - Airway, ventilatory, and circulatory support
    - IV fluid therapy
    - Vital sign and ECG monitoring
    - Transport
Poisoning by Injection

- May result from
  - Arthropod bites and stings
  - Reptile bites
  - Hazardous aquatic life
- Poisons from envenomation mixtures of many different substances
  - May produce several different toxic reactions in humans
  - Be prepared to manage reactions in many organ systems at same time

Poisoning by Injection

- General management
  - Ensure personal safety
  - Provide adequate airway, ventilatory, and circulatory support
    - Watch for signs of serious allergic reaction

Poisoning by Injection

- General management
  - Clean affected area with saline
    - Cover with sterile dressing
    - Intermittently apply ice
  - Obstruction tourniquets/suction devices do not delay absorption, should not be used
  - Commercially prepared antivenin (if available) sometimes given in emergency department
Poisoning by Injection

- General management
  - Moderate to severe symptoms may require aggressive management
    - Per medical direction, muscle spasm, severe headache, vomiting, paresthesia managed with benzodiazepines, antiemetics, and pain medication
  - Transport patient for physician evaluation
    - Most recover fully
    - Those at greatest risk for morbidity: very young, older adults, underlying hypertension or other medical problems

Arthropod Bites and Stings

- Invertebrates with segmented bodies and jointed limbs
  - Some bite, some sting, a few bite and sting
- Arthropod venoms complex, diverse in chemistry and pharmacology
  - May produce major toxic reactions in sensitized persons
  - Such reactions include anaphylaxis and upper airway obstruction

Arthropod Bites and Stings

- Classifications of various reactions to venoms
  - Local
  - Toxic
  - Systemic
  - Delayed
Hymenoptera (Wasps, Bees, Ants)

- Large, highly-specialized order of insects including
  - Wasps
  - Bees
  - Ants

- Venom contains mixtures of
  - Toxins
  - Enzymes
  - Other compounds
    - Histamines
    - Serotonin
    - Acetylcholine
    - Dopamine

Hymenoptera (Wasps, Bees, Ants)

- Single wasp, bee, or ant sting in unsensitized person causes
  - Instant pain
  - Followed by wheal-and-flare reaction with variable edema
**Hymenoptera (Wasps, Bees, Ants)**

- Most serious complication
  - Anaphylaxis
    - Estimated 0.4 percent of U.S. population has some chemical allergy to insect venoms
    - 40 to 100 deaths caused by anaphylaxis from hymenoptera stings reported annually
    - Persons with history of allergic reactions to stings often wear medical alert ID
    - Those allergic often carry an emergency kit that contains Epi-Pen

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**Hymenoptera (Wasps, Bees, Ants)**

- Fire ants
  - Species of greatest concern in U.S.
    - Stings or bites may produce systemic reactions, including anaphylaxis

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**Hymenoptera (Wasps, Bees, Ants)**

- Honeybees
  - Often leave stingers in wound
    - Should be scraped or brushed off
    - Should not be removed with forceps
    - Squeezing attached venom sac may worsen injury
  - Treat hypovolemia in conventional manner with volume-expanding crystalloid infusion
Arachnida
(Spiders, Scorpions, Ticks)
• Large class of arthropods
• Usually have four pairs of legs
• Body divided into cephalothorax (combined head and thorax) and abdomen

Spider Bites
• Most spiders have venom glands
• Two major types of reactions to spider venom
  – Neurotoxic reactions resulting from black widow bite
  – Local tissue necrosis from most other spider bites

Black Widow Spider
• Female
  – Shiny and black with red hourglass marking on undersurface of abdomen
• Male
  – About 1/2 size of female, brown, and nonvenomous to humans
Black Widow Spider

- Generally found in undisturbed areas (under stones, logs, clumps of vegetation)
  - Rarely inhabit occupied dwellings
- Most bites occur in rural and suburban areas of southern and western states between April and October

Black Widow Spider

- Bite generally described by patients as slight pinprick
  - Initially painless
  - As a rule, only physical findings are two small fang marks
    - 1 mm apart and surrounded by small papule
    - Multiple bites usually rule out spider envenomation because spiders rarely bite more than once
Black Widow Spider

• Symptoms
  – Within 1 hour of envenomation
  – Neurotoxin produces characteristic muscle spasms and cramps
    • Abdominal rigidity and intense pain may result

• Associated symptoms
  – Paresthesia (frequently a burning sensation in soles of feet or entire body)
  – Pain in shoulder, back, and chest muscles
  – Headache
  – Dizziness
  – Nausea and vomiting
  – Edema of eyelids
  – Increased perspiration and salivation
  – Most patients recover fully within 36 to 72 hours

Brown Recluse Spider

• Also known as fiddle-back spider
  – Most prevalent in Mississippi-Ohio-Missouri river basin
  – Southwestern U.S.
  – Prefers hot, dry, and abandoned environments such as vacant buildings
  – Often found in clothing closets
Brown Recluse Spider

• Identifying characteristics
  – Fawn to dark brown and 1 to 2 cm long
  – Six white eyes in semicircle on head (versus usual eight eyes of most other spiders)
  – Dark, violin-shaped marking on top of cephalothorax
  – Considered shy
    • Generally does not attack unless threatened
    – Most active from April to October

Brown Recluse Spider

• Symptoms
  – Venom initially causes little pain, often overlooked by victim
  – 1 to 2 hours later, localized pain and erythema
  – Followed within 2 days by blister or vesicle
Brown Recluse Spider

- Symptoms
  - Lesion may be surrounded by ischemic ring outlined further by irregular red halo
    - Classical bull’s-eye appearance seen with this bite
  - Over next 72 hours
    - Area often enlarges
    - Center of lesion may become purple or develop black eschar (dead tissue)
    - Eschar eventually sloughs, leaving ulcer of variable size and depth

Brown Recluse Spider

- Wound typically slow to heal, may be visible months to years after bite
  - Occasionally, excision and skin grafting necessary

Brown Recluse Spider

- Systemic involvement may occur with signs and symptoms
  - Fever
  - Chills
  - Malaise
  - Nausea and vomiting
  - Generalized rash
  - Development of hemolytic anemia, hemoglobinuria, and hypotension
  - Occasional death
    - Usually from disturbance of coagulation system or hepatic injury
For which type of spider bite is a patient most likely to call an ambulance? Why?

**Scorpion Stings**

• Of over 650 species of scorpions, only few produce human envenomation
  – Sculptured or bark scorpion only North American species dangerous to humans
    • Southwestern U.S. and Mexico
    • Nocturnal
    • Favors wooded areas along edge of desert washes
    • Occasionally invades homes, especially adobe houses
Scorpion Stings

- Small and yellow to brown
  - Some have tail stripes
- Most active from April to August, hibernating during winter

Scorpion Stings

- Venom delivered by stinger on telson
  - Mixture of proteins that causes release of acetylcholine
    - Stimulates sympathetic nerves
    - Directly stimulates CNS, causing hyperactivity
  - Venom has no enzymes that cause tissue destruction
    - Local inflammation thus not feature of this scorpion
  - If swelling, ecchymosis, or redness is present, scorpion not of neurotoxic type
  - Despite potential for life-threatening systemic effects, mild analgesics, cool compresses, and in-hospital observation adequate
Tick Bites

- Seldom require emergency care
  - Capable of causing human disease
  - Can transmit microorganisms or secrete toxins or venoms
- Hard ticks most familiar type in North America
  - Soft ticks also common to western states
  - Local reactions to bites vary
    - From small pruritic nodule to extensive ulceration

Rocky Mountain Spotted Fever

- Infectious disease
  - Transmitted from rabbits and small mammals to humans by
    - Wood tick bites
    - Dog tick bites
- More common on Atlantic seaboard
  - Accounts for ≈ 40 deaths in U.S. annually

Rocky Mountain Spotted Fever

- Signs and symptoms usually develop within 1 week of bite
  - Headache
  - High fever
  - Loss of appetite
  - May be followed with small pink spots on wrists and ankles
  - Eventually rash spreads over entire body
    - Spots darken, enlarge, become petechial
Rocky Mountain Spotted Fever

- In mild cases, recovery occurs within 20 days
- Mortality rate, if untreated, is between 8 and 25 percent

Lyme Disease

- Most commonly reported tick-borne disease in U.S.
  - Caused by bite of Ixodes tick known to infect deer and dogs

Lyme Disease

- Stages
  - 1st stage
    - Red dot appears at site of bite, gradually expands into reddened rash
    - Fever, lethargy, muscle pain, general malaise
  - 2nd stage
    - Cardiac abnormalities (including various AV blocks) and neurological effects, such as cranial nerve palsy
  - 3rd stage
    - Arthritis primary symptom
Tick Paralysis

- Results from prolonged bite by female wood tick
  - Occurs sporadically during spring and summer months
  - Caused by neurotoxin secreted from salivary glands after tick attaches to host
  - At first patient is restless and experiences paresthesia in hands and feet

- Over next 48 hours, flaccid paralysis may develop with loss of deep tendon reflexes
  - Begins at feet and travels upward, affecting both sides of body
  - Death may result from respiratory paralysis (severe cases)

- Removal of tick usually results in rapid improvement and complete resolution within several days
- May be fatal, especially in young and older patients, if undiagnosed

How can you distinguish tick paralysis from other conditions that cause progressive paralysis?
Management

• Principal treatment: proper removal of tick
  – Grasp tick as close to skin surface as possible with forceps, tweezers, or protected fingers
  – Pull it out with steady pressure
  – Take care not to crush or squeeze tick’s body
    • Can transmit disease from infective tick fluid

Reptile Bites

• American Association of Poison Control Centers National Data Collection System
  – Listed a total 6,343 bites from poisonous and nonpoisonous snakes in 2007
    • 3,267 were known to be poisonous
    • Rest from unidentified snakes
    • 2 deaths
    • Reflects high morbidity and low mortality rates associated with snake venom poisoning
Reptile Bites

• Of 115 species of snakes in U.S, only 19 are venomous
• Two main families of venomous snakes indigenous to U.S.
  – Pit vipers
  – Coral snakes

Pit Vipers

• In U.S. consists of
  – Rattlesnakes
  – Cottonmouth or water moccasin
  – Copperhead
  – Pigmy rattlesnake
  – Massasauga rattlesnake

• Vast majority of snakebites in U.S. caused by rattlesnake family
  – Identifying features
    • Vertical elliptical pupils
    • Triangular head distinct from rest of body
    • Interlocking horny segments (rattles) formed on tail that sometimes vibrate in direct relation to environmental temperatures
Pit Vipers

- Venom apparatus
  - Connected to one or more elongated hollow fangs on each side of head

Pit Vipers

- Venom
  - Designed to immobilize, kill, digest prey
  - Capable of producing various toxic effects on blood and other tissues
    - Hemolysis
    - Intravascular coagulation
    - Convulsions
    - Acute renal failure
  - Bleeding caused by coagulation defects and massive swelling can lead to hypovolemic shock
  - On any given strike, may release quantity varying from little or none to almost entire content of glands
Coral Snakes

- Two members found in U.S.
  - Arizona coral snake
  - Eastern coral snake
- Identifying features
  - Round pupils and small, fixed fangs located near anterior end of maxilla
  - Most have three-color pattern with red, black, and yellow or white bands that completely encircle body
  - Black snout

Coral Snakes

- Many nonpoisonous snakes in U.S. mimic appearance of coral snake
  - Identified by sequence of colors: red bands bordered by yellow indicate venomous species
  - Mnemonic: “red on yellow, kill a fellow; red on black, venom lack”
Coral Snakes

- Most are shy and docile and seldom bite unless threatened
  - Small mouth and fangs make it difficult to bite anything larger than finger, toe, or fold of skin
  - Tends to hang on and chew rather than to strike and release
  - Venom mainly is neurotoxic
  - Bite generally produces little or no pain, no necrosis or edema

Coral Snakes

- Early signs and symptoms
  - Slurred speech
  - Dilated pupils
  - Dysphagia (usually delayed several hours after bite)
- If untreated, venom produces flaccid paralysis and death within 24 hours
  - Death caused by respiratory failure, following nervous system dysfunction

Management of Snake Envenomation

- Venom phases
  - Absorption
  - Distribution
  - Elimination
- Tissue damage increases as venom spreads into lymphatics and blood
  - Emergency care directed at retarding systemic spread of venom
Management of Snake Envenomation

• Prehospital management
  – Stay clear of striking range of snake (about length of snake)
    • Move patient to safe area
    • If snake killed before EMS arrival, transport in closed container to emergency department with patient
    • Make no attempt to capture or kill snake; doing so may result in being bitten
    • Identification of snake is not necessary to manage patient appropriately

Management of Snake Envenomation

• Prehospital management
  – Provide adequate airway, ventilatory, and circulatory support
  – Continually monitor vital signs and ECG
  – Establish IV line in unaffected extremity with volume-expanding fluid

Management of Snake Envenomation

• Prehospital management
  – Medical direction may recommend that bitten extremity be immobilized in neutral position
    • May delay systemic absorption
    • May diminish local tissue necrosis
  – Keep the patient at rest
  – If coral snake inflicted bite, wrap bandage snugly around entire extremity
  – Prepare patient for immediate transport
Management of Snake Envenomation

- Prehospital management
  - Methods to remove venom or delay absorption are potentially harmful, should not be used
  - Avoid application of ice or chemical cold packs
    - May further damage tissue
  - Administration of antivenin to neutralize venom may be required
    - Provided in hospital after patient is tested for allergies to antivenin

What strategies can you use to calm the emotional state of a patient who has sustained a snakebite?

Hazardous Aquatic Life

- Marine animals involved in human poisonings in U.S. coastal waters
  - Coelenterates
  - Echinoderms
  - Stingrays
- Venom apparatuses
  - Used for defense
  - Capture prey
Hazardous Aquatic Life

- In addition to venom, may contain other poisonous substances as result of toxic ingestions
- Exposures to hazardous aquatic life result from recreational, industrial, scientific, military oceanic activities

Coelenterates (Jellyfish, Sea Anemones, Fire Coral)

- Group of species that may be encountered in ocean
  - Some carry venomous stinging cells (nematocysts)
    - Venom filled and contains long, coiled, hollow, threadlike tube that serves as tiny hypodermic needle
  - Severity of envenomation related to
    - Type and toxicity of venom
    - Number of nematocysts discharged
    - Physical condition of victim
Coelenterates (Jellyfish, Sea Anemones, Fire Coral)

- Jellyfish
  - Occur throughout Atlantic and Pacific oceans
  - Portuguese man-of-war is largest
    - Nematocyst-bearing tentacles may be up to 100 feet long
    - Single envenomation may involve several hundred thousand nematocysts
  - Swimmer who comes in contact may suffer enough envenomation to produce systemic signs and symptoms
  - Nematocysts often remain embedded in tissues of victim
  - Detached tentacle fragments can retain their potency for months

- Sea anemones
  - Colorful bottom dwellers
  - Sometimes found in tidal pools
  - Have flowerlike appearance
  - Possess slender projections used to sting and paralyze passing fish
    - Capable of producing mild to moderate pain in humans
Coelenterates (Jellyfish, Sea Anemones, Fire Coral)

• Fire corals
  – Not true, stony corals, but ocean-bottom dwellers
  – Often mistaken for seaweed
    • Commonly attached to rocks, shells, corals
  – May grow to 2 m in height
  – Have razor-sharp exoskeleton with thousands of protruding nematocyst-bearing tentacles

Management

• Coelenterate envenomation ranges in severity from
  – Irritant dermatitis to excruciating pain
  – Respiratory depression
  – Anaphylaxis
  – Life-threatening cardiovascular collapse

Management

• Envenomation
  – Most often is mild and usually characterized by
    • Stinging
    • Paresthesias
    • Pruritus
    • Reddish-brown linear wheals or “tentacle prints”
Management

- Envenomation
  - If potent venom or large body surface area involved, systemic symptoms may include
    - Nausea
    - Vomiting
    - Abdominal pain
    - Headache
    - Bronchospasm
    - Pulmonary edema
    - Hypotension
    - Respiratory arrest

Management

- Emergency care: directed at stabilizing patient and neutralizing effects of venom
  - Stabilize patient
    - Provide adequate airway, ventilatory, and circulatory support as needed
    - Continually monitor patient’s vital signs and ECG
    - Be prepared to provide advanced airway management if systemic reactions develop

Management

- Counteract effects of venom
  - Remove visible tentacle fragments with forceps
    - Avoid touching tentacles
  - Immediately rinse patient’s wound with seawater
    - Wet sand or freshwater usually causes nematocysts to discharge their venom, are contraindicated
  - Apply copious amounts of isopropanol and then baking soda paste to inactivate nematocysts
    - Isopropanol “fixes” nematocysts from firing
    - Baking soda helps remove them
Management

• Counteract effects of venom
  – Apply lather of shaving cream
    • Gently shave affected area to remove invisible nematocysts
  – Rinse again until pain is largely alleviated
    • If needed, consult medical direction regarding administration of analgesics
  – Transport

Echinoderms (Sea Urchins, Starfish, Sea Cucumbers)

• Marine animals with water-vascular system
  – Usually have
    • Hard, spiny skeleton
    • Radial body
Echinoderms (Sea Urchins, Starfish, Sea Cucumbers)

- Sea urchins
  - Globular, dome-shaped body
  - Found on rocky bottoms or burrowed in sand or crevices
  - Have tiny spines, some of which are venomous
  - Some also have small pincer-like organs thought to discharge poisonous substance
  - Spines are dangerous to handle
    - May break off easily in flesh
    - Lodge deeply, making removal difficult
Echinoderms (Sea Urchins, Starfish, Sea Cucumbers)

- Starfish
  - Covered with thorny spines that secrete toxins
  - As spine enters skin, carries venom into wound with
    - Immediate pain
    - Copious bleeding
    - Mild edema
  - Multiple puncture wounds may result in acute systemic reactions

- Sea cucumbers
  - Sausage-shaped animals found in shallow and deep water
  - Produce liquid toxin in tentacle-shaped organ
    - Organ can be projected and extended anally
    - Liquid is secreted into surrounding ocean
    - Usually produces only minor dermatitis or conjunctivitis in swimmers and divers

Management

- Emergency management for echinoderm envenomation
  - Usually involves caring for puncture wounds caused by spines and inactivating venom
  - Remove embedded spines with forceps
  - Wear protective gloves to avoid self-contamination
  - Larger spines may require surgical removal by physician
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<th>Management</th>
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<tbody>
<tr>
<td>• Echinoderm toxins may cause immediate</td>
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<tr>
<td>– Intense pain</td>
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<tr>
<td>– Swelling</td>
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<tr>
<td>– Redness</td>
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<tr>
<td>– Aching in affected extremity</td>
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<td>– Nausea</td>
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<th>Management</th>
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<tr>
<td>• Delayed toxic effects</td>
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<tr>
<td>– Respiratory distress</td>
</tr>
<tr>
<td>– Paresthesia of lips and face</td>
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<tr>
<td>– Respiratory paralysis</td>
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<tr>
<td>– Complete atonia</td>
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<th>Management</th>
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<tr>
<td>• Most marine venoms lose toxicity when exposed to changes in temperature or humidity</td>
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<tr>
<td>– Recommended management for stable patients</td>
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<tr>
<td>• Immerse affected area (usually foot or hand) in warm water before and during transport</td>
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<tr>
<td>• Water should be as hot as can be tolerated without scalding, but no warmer than 113°F (45°C)</td>
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<tr>
<td>• As safety precaution, recommended that both hands or feet not be immersed at same time</td>
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<td>• Protects against thermal injury that may go unnoticed by patient because of numbness or pain in affected part</td>
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Stingrays

• Stingrays
  – Responsible for about 1,800 injuries each year in U.S.
  – Vary in size from 2 inches to 14 feet
  – Often found half-buried in mud or sand in shallow water
  – Venom organ consists of two to four venomous barbs on dorsum of whip-like tail

Stingrays

• Envenomation generally occurs from stepping on sand-buried ray
  – Causes tail to thrust up and forward, driving barb into victim’s leg or foot
  – Defensive barb produces large, severe laceration that may be over 15 to 20 cm long
  – Entire barb tip of venom apparatus sometimes is broken off, embedded in tissue
Stingrays

- Venom has local and systemic complications
  - Locally, venom produces traumatic injury that causes
    • Immediate, intense pain
    • Edema
    • Variable bleeding
    • Necrosis

- Systemic manifestations
  • Weakness
  • Nausea
  • Vomiting
  • Diarrhea
  • Vertigo
  • Seizures
  • Cardiac conduction abnormalities
  • Paralysis
  • Hypotension
  • Death

Stingrays

- Prehospital care
  - Life support
  - Alleviation of pain
  - Inactivation of venom
  - Prevention of infection
  - Irrigate wound with normal saline or freshwater
  - If venom apparatus is visible, carefully remove
  - Immerse affected part in warm water, remain there until pains subside or until patient reaches emergency department
  - Analgesics may be needed to manage pain
Lesson 34.3
Organophosphate, Carbamate Poisoning, and Management of Drug Overdose

Learning Objectives
• Describe the signs, symptoms, and management of patients with organophosphate or carbamate poisoning.
• Outline the general principles of managing patients with drug overdose.

Poisoning by Absorption
• Many poisons can be absorbed through skin
• Two compounds responsible for large number of skin-absorbed poisonings each year
  – Organophosphates
  – Carbamates
    • Commonly available for commercial and public use in form of pet, home, commercial insecticides
Poisoning by Absorption

• Organophosphates used in development of military nerve agents such as sarin and soman
  – Because of widespread availability of insecticides that contain compounds, be aware of
    • Nature of these chemicals
    • Necessary precautions for personal safety
    • Immediate management that may be required before symptoms or signs of illness occur

Poisoning by Absorption

• Organophosphates and carbamates are highly toxic
  – Well absorbed by
    • Ingestion
    • Inhalation
    • Dermal routes
  – Both classes have similar pharmacological actions, inhibiting effects of acetylcholinesterase
    • Enzyme that degrades acetylcholine at nerve terminals
  – Acetylcholine is cholinergic neurotransmitter for
    • Preganglionic autonomic fibers
    • Somatic nerves to skeletal muscle
    • Many synapses in CNS

Poisoning by Absorption

• When acetylcholinesterase is inhibited, acetylcholine accumulates at synapses
  – Results in cholinergic “overdrive”
    • Signs and symptoms resulting from cholinergic overdrive are seen in organophosphate and carbamate poisoning
Signs and Symptoms

- Early signs and symptoms may be nonspecific
  - Headache
  - Dizziness
  - Weakness
  - Nausea
- As overstimulation and disruption of transmission in central and peripheral nervous systems occur, signs and symptoms begin to develop
  - Result from wide range of physiological and metabolic derangements

Signs and Symptoms

- Rapidity and sequence in which signs and symptoms develop depend on particular compound and on amount and route of exposure
  - Quickest after inhalation
  - Slowest after primary skin exposure and may be delayed for several hours

Signs and Symptoms

- Helpful mnemonics to recognize signs of poisoning
  - SLUDGE
    - Salivation
    - Lacrimation
    - Urination
    - Defecation
    - GI upset
    - Emesis
### Signs and Symptoms

- Helpful mnemonics to recognize signs of poisoning
  - DUMBELLS
    - Diarrhea or diaphoresis
    - Urination
    - Miosis or muscle fasciculations
    - Bronchospasm
    - Bradycardia
    - Emesis
    - Lachrimation
    - Lethargy
    - Salivation

- Rapidly changing pupils with miosis are common with vapor exposure of organophosphates
  - Muscle twitching (fasciculations) can follow rapidly
    - Individual muscle twitching can result from liquid contact and local skin absorption at site

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Consider a person who does not suspect poisoning. What condition might that person think he or she is suffering from with this clinical presentation?
Management

• Emergency care
  — Begins with scene safety
  — Personal protection
    • Wear protective clothing
    • Use respiratory protection
  — Decontamination procedures
    • Remove patient safely from contaminated area as soon as possible
  — Secure scene by qualified personnel
  — After these measures, patient care can be initiated

Management

• General principles
  — Respiratory support
  — Drug administration
  — ECG monitoring
• Organophosphates and carbamates produce similar physiological effects
  — Carbamates have shorter duration of action
    • More rapid decrease in effect

Respiratory Support

• Respiratory tract symptoms usually first to appear after exposure
  — Respiratory paralysis may occur suddenly without warning
  — Need for advanced airway management and ventilatory support should be anticipated
  — Copious bronchial secretions
    • May require suctioning
    • May necessitate positive-pressure ventilation and positive end-expiratory pressure
Drug Administration

• Directed at
  - Blocking effects of acetylcholine
  - Separating cholinesterase from chemical compound
  - Suppressing seizure activity if present

• Antidotes
  - Atropine
  - Pralidoxime chloride
  - Diazepam or lorazepam

Drug Administration

• Atropine
  - Reverses muscarinic effects of moderate to severe poisoning
    • Bradycardia
    • Bronchoconstriction
    • Respiratory secretions
    • Miosis

Drug Administration

• Atropine
  - Competitively antagonizes actions of acetylcholine
    • Results in decrease in hyperactivity of smooth muscles and glands
  - Indicated to dry patient’s secretions
  - Helps to decrease pulmonary resistance to ventilation
Drug Administration

• Atropine
  – Potentially hypoxic patients may require large doses
  – ECG should be monitored for dysrhythmias (other than tachycardia)
  – Supplemental O₂ given to minimize risk of ventricular fibrillation
  – Drug of choice for carbamate poisonings

Drug Administration

• Pralidoxime
  – Treatment of choice for organophosphate poisoning after administration of atropine
  – Should be used for nearly all patients with significant exposures, particularly those with muscular twitching and weakness
  – Primary effect of reactivating acetylcholinesterase
  – Adult and pediatric doses can be found in Emergency Drug Index

Drug Administration

• Diazepam or lorazepam
  – May be indicated if seizures present
  – Need for seizure control may arise before decontamination is complete
    • Drugs can be administered intramuscularly to control seizure activity
  – As safety precaution, IV therapy usually not initiated in contaminated area
  – Be alert to risk for respiratory and CNS depression
Consider that you give diazepam or lorazepam for seizures in this case. Will that eliminate the need for atropine?

ECG Monitoring

- May reveal variety of abnormalities
  - Idioventricular rhythms
  - Multifocal premature ventricular contractions
  - Ventricular tachycardia
  - Torsades de pointes
  - Ventricular fibrillation
  - Complete heart block
  - Asystole

ECG Monitoring

- Dysrhythmias usually occur in two phases
  - First phase begins with transient episode of intense sympathetic tone
    - Results in sinus tachycardia
ECG Monitoring

- Second phase: period of extreme parasympathetic tone
  - May manifest as
    - Sinus bradycardia
    - AV block
    - ST segment and T wave abnormalities
  - Slow ventricular dysrhythmias that do not respond to usual therapy may need to be treated with overdrive pacing

Drug Abuse

- Use of prescription drugs for nonprescribed purposes
  - Use of drugs that have no prescribed medical use
- Emergencies that result from drug abuse
  - Adverse effects caused by drug or impurities/contaminants mixed with drug
  - Life-threatening infections from IV or intradermal injection of drugs with unsterile equipment
  - Injuries during intoxication
  - Drug dependence or withdrawal syndrome resulting from habit-forming potential of many drugs

Drug Abuse

- Maintain high degree of suspicion and consider possibility for drug-related problem in any patient of any age who has
  - Seizures
  - Behavioral changes
  - Decreased level of consciousness
- Consideration of visibility, accessibility, careful handling of all medications carried on EMS vehicle should be part of any EMS policy and procedure
Why might a patient (or his or her friends) delay calling for help in a situation involving drug overdose?

Toxic Effects of Drugs

• EMS personnel often encounter persons who suffer from toxic effects of drugs
  – Toxicity may be result of
    • Overdose
    • Potential suicide
    • Polydrug administration
    • Accident (accidental ingestion, miscalculation, changes in drug strength)

Toxic Effects of Drugs

• Common drugs of abuse, along with their names and uses, vary widely in different geographical areas
  – Drugs of abuse often change over time
General Management Principles

- General principles for managing drug abuse and overdose
  - Ensure that scene is safe
    - Be prepared for unpredictable behavior from patient
    - Consider need for help from law enforcement
  - Ensure adequate airway, ventilatory, and circulatory support as needed

- General principles for managing drug abuse and overdose:
  - Obtain history of event
    - Include self-administration of other drugs that may have been taken by another route
    - Obtain any significant medical or psychiatric history
  - Identify substance
    - Consult with medical direction or poison control center
  - Perform a full, focused physical examination
    - Continually monitor patient’s vital functions and ECG

- General principles for managing drug abuse and overdose:
  - Start IV therapy
    - Draw blood sample for laboratory analysis and administer proper drug antidotes such as naloxone if opioid overdose is suspected
    - Pay special attention to personal protection because many of these patients are at high risk of harboring infectious disease
    - Nasal administration of naloxone should be considered in these patient groups
  - Administer activated charcoal (per protocol) for orally administered drugs taken within previous hour
General Management Principles

- General principles for managing drug abuse and overdose
  - Rapidly transport patient for physician evaluation
    - When examining any patient suspected of abusing drugs, always look for track marks
    - May be in antecubital space, under tongue, or on top of feet
    - Possibility of "body packing" (concealing packets of drugs in body cavities of stomach, rectum, vagina), "body stuffing" (swallowing drugs to avoid arrest) should be considered when person who abuses drugs appears ill for no apparent reason

For what illness is the patient who uses IV narcotics at risk?

Lesson 34.4

Drug Overdose
Learning Objective

• Describe the effects, signs and symptoms, and specific management for selected therapeutic and illegal drug overdoses.

Opioid Overdose

• Heroin accounts for about 90 percent of opioid abuse in U.S.
  – Pure heroin is bitter-tasting white powder
    • Usually is adulterated or “cut” for street distribution
  – Typical “bag” is single-dose unit of heroin and may weigh 100 mg
  – On average, heroin is only 20 to 30 percent pure

• Other opioid drugs
  • Morphine
  • Hydromorphone
  • Methadone
  • Meperidine
  • Codeine
  • Oxycodone
  • Propoxyphene
  • Hydrocodone
  • "Designer opioids" that have been chemically modified such as alpha-methyl fentanyl (“China white”)
Opioid Overdose

- Depending on preparation, these drugs may be taken
  - Orally, injected intradermally
    - “Skin popping”
  - Intravenously
    - “Mainlining”
  - Intranasally
    - “Snorted”
  - Smoked

All opioids are CNS depressants
- Can cause life-threatening respiratory depression
- In severe intoxication, possible presence of
  - Hypotension
  - Profound shock
  - Pulmonary edema

Signs and symptoms of narcotic/opioid overdose
- Euphoria
- Arousable somnolence (“nodding”)
- Nausea
- Pinpoint pupils (except with meperidine, hypoxia, or in combination with other types of drugs)
- Slow and shallow respirations
- Coma
- Seizures
Antidote Therapy

• Naloxone
  – Pure opioid antagonist effective for virtually all opioid and opioid-like substances
  – Reverses the three major symptoms of opioid overdose
    • Respiratory depression
    • Coma
    • Miosis

Antidote Therapy

• Naloxone
  – Indicated for use when coma of unknown origin is present
  – Be prepared to restrain patient
  – Patient’s behavior may be unpredictable when effects of drug are reversed and patient experiences withdrawal symptoms
  – Medical direction may recommend it be given in small amounts
    • Amount should be enough to restore airway reflexes and adequate breathing, without fully awakening patient

Antidote Therapy

• Ventilate with bag mask prior to naloxone administration
• If patient does not respond to treatment, intubation should be considered
  – In absence of respiratory depression, use is controversial
  – Seizure activity is possible side effect of drug
Antidote Therapy

- Some opioids (e.g., heroin) have longer duration than naloxone
  - Monitor patient closely during antidote therapy
  - Repeated doses may be needed as well
  - In communities where abuse of naloxone-resistant opioids or use of China white is common, larger initial doses of naloxone may be needed
  - Desired signs of reversal of opioid intoxication
    - Adequate airway reflexes and ventilations, not complete arousal

Antidote Therapy

- Naloxone
  - Can cause withdrawal syndrome in opioid-dependent patients
    - Slowly administer smaller dose of drug for these patients
  - Withdrawal usually can be managed by symptomatic and supportive care

Sedative-Hypnotic Overdose

- Sedative-hypnotic agents
  - Benzodiazepines
  - Barbiturates
  - Usually taken orally
  - May be diluted and injected intravenously
  - Taking with alcohol greatly increases their effects
  - Commonly known as downers
Sedative-Hypnotic Overdose

- Benzodiazepines
  - Best-known to control symptoms of
    - Anxiety
    - Stress
    - Insomnia
  - Sometimes used to manage alcohol withdrawal and control seizure disorders
  - Promote sleep, relieve anxiety by depressing brain function
  - Often abused for sedative effects

Sedative-Hypnotic Overdose

- Benzodiazepines
  - Individually, somewhat nontoxic
  - May accentuate effects of other sedative-hypnotic agents
  - Common benzodiazepines
    - Diazepam (Valium)
    - Alprazolam (Xanax)
    - Lorazepam (Ativan)

Sedative-Hypnotic Overdose

- Barbiturates
  - General CNS depressants that inhibit impulse conduction in brainstem
  - Once were widely used to treat anxiety and insomnia
  - Addictive properties and potential for abuse have led to their replacement by benzodiazepines and other nonbarbiturate drugs
  - Commonly abused
    - Phenobarbital
    - Amobarbital
    - Secobarbital
Sedative-Hypnotic Overdose

- Signs and symptoms
  - Chiefly related to central nervous and cardiovascular systems
  - Excessive drowsiness
  - Staggering gait
  - Paradoxical excitability

- In severe toxicity, may become comatose with
  - Respiratory depression
  - Hypotension
  - Shock
  - Constricted pupils
    - More often become fixed and dilated even in absence of significant brain damage

- Airway control and ventilatory management are most important actions in managing significant overdose

Sedative-Hypnotic Overdose

- Flumazenil
  - Benzodiazepine antagonist
  - Used to reverse effects of benzodiazepines
  - Can produce seizure activity
  - Contraindicated in patients prone to seizures, those with tricyclic antidepressant overdose
  - Generally only used to reverse benzodiazepine effects after procedural sedation
Stimulant Overdose

- Commonly abused stimulant drugs are of sympathomimetic family
  - Amphetamine sulfate
  - Dextroamphetamine
  - Cocaine
  - Methamphetamine

Stimulant Overdose

- Sympathomimetic drugs
  - Produce general mood elevation
  - Improve task performance
  - Suppress appetite
  - Prevent sleepiness

Stimulant Overdose

- Structurally, amphetamines are similar to epinephrine and norepinephrine
  - Differ in their more pronounced effects on CNS
- Adverse effects
  - Tachycardia
  - Increased BP
  - Tachypnea
  - Agitation
  - Dilated pupils
  - Tremors
  - Disorganized behavior
  - In severe intoxication, patient may exhibit psychosis and paranoia and may experience hallucinations
Stimulant Overdose

- Sudden withdrawal or cessation of amphetamine use may result in "crash" stage
  - Patient becomes
    - Depressed
    - Suicidal
    - Incoherent
    - Near coma

Stimulant Overdose

- Drugs taken orally
  - May be smoked or injected for more rapid onset of action
  - Amphetamines commonly known as speed or uppers

Cocaine

- One of the most popular illegal drugs in U.S
  - Fine, white crystalline powder
  - Street forms usually are adulterated
    - Vary in purity from 25 to 90 percent
    - Doses vary from near 0 to 200 mg
    - This form generally is taken intranasally by snorting "line" containing 10 to 35 mg
    - After absorption through mucous membranes, effects begin within minutes
    - Peak effects occur 15 to 60 minutes after use, with half-life of 1 to 2 1/2 hours
Cocaine

• Other routes
  – Subcutaneous
  – Intramuscular
  – IV
    • Provides immediate absorption and intense stimulation
    • Peak occurs within 5 minutes, with half-life of about 50 minutes
    • Speed-balling refers to injection of cocaine-heroin combination

Cocaine

• Freebase or “crack” cocaine
  – More potent formulation of drug
  – Prepared by mixing powdered street cocaine with alkaline solution, then adding solvent such as ether
  – Combination separates into two layers
    • Top layer contains dissolved cocaine
    • Evaporation of solvent results in pure cocaine crystals, which are smoked and absorbed via pulmonary route

Cocaine

• Freebase or “crack” cocaine
  – Called rock or crack because of popping sound produced when crystals are heated
  – Freebase cocaine generally combined with marijuana or tobacco and smoked in pipe or cigarette
    • Reactions are similar to those experienced in IV use, with equal intensity and effects
Cocaine

• Major CNS stimulant
  – Causes profound sympathetic discharge
  – Increased levels of circulating catecholamines result in
    • Excitement
    • Euphoria
    • Talkativeness
    • Agitation

Cocaine

• Effects of the drug can cause significant cardiovascular and neurological complications
  – Cardiac dysrhythmias
  – MI
  – Seizures
  – Intracranial hemorrhage
  – Hyperthermia
  – Psychiatric disturbances

Cocaine

• Overdose can occur with any form, any route
  – Adult fatal dose is thought to be about 1200 mg (1.2 g)
    • Fatalities from cocaine-induced cardiac dysrhythmias have been reported with single doses of as little as 25 to 30 mg
Cocaine

• Prehospital management may be difficult
  – Toxicity may range from minor symptoms to life-threatening overdose
  – Emergency care may require full spectrum of basic and advanced life-support measures
    • Aggressive airway management
    • Ventilatory and circulatory support
    • Drug therapy
    • Rapid transport
    • Benzodiazepines are mainstay of treatment initially in cocaine toxicity

Phencyclidine Overdose

• Phencyclidine (PCP)
  – Dissociative analgesic originally used as veterinary tranquilizer
  – Has sympathomimetic and CNS stimulant, depressant properties
  – Potent psychoactive drug illegally sold in liquid, tablet, powder form to be taken
    • Orally
    • Intranasally
    • Intravenously/intramuscularly
    • Other drugs to be smoked ("Sherman")
  – Most tablets contain about 5 mg

Phencyclidine Overdose

• As rule, in powder form is purer (50 to 100 percent PCP)
• Chronic use can result in
  – Permanent memory impairment
  – Loss of higher brain functions
• Pharmacological effects are dose related
  – Can be divided into low-dose and high-dose toxicity
• Ketamine is derivative of PCP and has identical actions

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Low-Dose Toxicity

• In low doses under 10 mg, intoxication produces unpredictable state that can resemble drunkenness
  – Euphoria or confusion, disorientation
  – Agitation
  – Sudden rage

Low-Dose Toxicity

• Intoxicated patient often has blank stare and stumbling gait
  – Often in dissociative state
  – Pupils generally reactive
  – May experience
    • Flushing
    • Diaphoresis
    • Facial grimacing
    • Hypersalivation
    • Vomiting

Low-Dose Toxicity

• Nystagmus
  – Burst-like quality
  – Death usually related to behavioral disturbances resulting from
    • Spatial disorientation
    • Drug-induced immobility
    • Insensitivity to pain
    • Leads to bold acts of strength
    • Normal muscle activity limitation resulting from pain is inhibited
Why does low-dose toxicity put the patient at a high risk for injury?

Low-Dose Toxicity

• Sensory stimulation should be avoided
  – Verbal and physical stimuli will likely make clinical symptoms worse
  – Violent and combative patients require protection from self-injury
  – Safeguards must be provided for emergency crew and bystanders
  – Monitor vital signs and level of consciousness closely
  – Observe for increasing motor activity and muscle rigidity as well
  – May precede seizures

High-Dose Toxicity

• Patients with high-dose PCP intoxication from more than 10 mg may be in coma that can last from hours to several days
  – Often unresponsive to painful stimuli
  – Respiratory depression, hypertension, tachycardia also may be present, depending on dosage
  – In severe cases, hypertensive crisis causing cardiac failure, hypertensive encephalopathy, seizures, and intracerebral hemorrhage may result
High-Dose Toxicity

• Prehospital care
  – Manage respiratory and cardiac arrest
  – Manage status seizures
  – Rapidly transport

Phencyclidine Psychosis

• True psychiatric emergency that may mimic schizophrenia
  – Acute onset
  – May not become apparent until several days after drug ingestion
  – Can occur after single low-dose exposure to PCP
  – May last from several days to weeks
• Signs and symptoms
  – May range from catatonic and unresponsive state to bizarre and violent behavior

Phencyclidine Psychosis

• Often appears agitated and suspicious
  – Auditory hallucinations
  – Paranoia
• Management
  – Usually requires involuntary hospitalization
  – Control of violent behavior
  – Administration of antipsychotic agents
  – Personal safety is of prime importance
    • Law enforcement should be called on for assistance
Hallucinogen Overdose

- Substances that cause perceptual distortions
  - Most common: lysergic acid diethylamide (LSD)
  - Mescaline
    - Found in buttons of peyote cactus
  - Psilocybin mushrooms
    - Found in U.S. and Mexico

- Marijuana
  - Active agent of Cannabis sativa
- Morning glory plant
- Nutmeg
- Mace
- Methylenedioxymethamphetamine (Ecstasy)
- 3, 4-methylenedioxymphetamine (MDEA Eve)

- Depending on agent, effects may range from minor visual to more serious complications associated with LSD use
  - Respiratory and CNS depression (rare)
Hallucinogen Overdose

- Prehospital management
  - Usually is limited to supportive care
  - Minimal sensory stimulation
  - Calming measures
  - Transportation to medical facility
  - After arrival at emergency department, patients generally are placed in quiet environment for observation

Tricyclic Antidepressant Overdose

- Often are prescribed to help manage depression and certain pain syndromes
  - Work by blocking uptake of norepinephrine and serotonin into presynaptic neurons
  - Alter sensitivity of brain tissue to actions of these chemicals
  - Serious tricyclic antidepressant toxicity results in sodium-channel blockade in myocardium

- Other toxicities
  - Potassium efflux blockade and blockade of blood vessels
  - Anticholinergic effects
  - Seizures

- Commonly prescribed antidepressant drugs
  - Tricyclic antidepressants amitriptyline
  - Desipramine
  - Imipramine
  - Nortriptyline
Tricyclic Antidepressant Overdose

- Newer selective serotonin reuptake inhibitors chemically unrelated to tricyclic antidepressants
  - Fluoxetine
  - Sertraline
  - Paroxetine
  - Considered safe and effective compared with tricyclic antidepressants

Tricyclic Antidepressant Overdose

- Early symptoms of overdose
  - Dry mouth
  - Blurred vision
  - Confusion
  - Inability to concentrate
  - Occasionally visual hallucinations

Tricyclic Antidepressant Overdose

- More severe symptoms
  - Delirium
  - Depressed respirations
  - Hypertension
  - Hypotension
  - Hyperthermia
  - Hypothermia
  - Seizures
  - Coma
Tricyclic Antidepressant Overdose

• Cardiac effects
  – Tachycardia
  – Bradycardia
  – Dysrhythmias caused by AV block
  – Prolonged QRS complex, Glasgow Coma Scale lower than 8 are characteristic findings that should alert to major toxicity with potentially serious complications.
  – Sudden death from cardiac arrest may occur several days after overdose

Tricyclic Antidepressant Overdose

• Prehospital management
  – Basic supportive care
  – Rapid transport
    • 25 percent of patients who ultimately die as result of overdose are alert and awake
    • 75 percent have normal sinus rhythm when EMS personnel arrive
    • Tachycardia, especially with wide QRS complex greater than 100 ms, is early sign of toxicity
    • Sodium bicarbonate, per medical direction, may begin to reverse cardiac toxicity

Tricyclic Antidepressant Overdose

• Any patient with history of tricyclic antidepressant ingestion should receive
  – Airway ventilatory and circulatory support
  – IV access
  – ECK monitoring
  – Rapid transport
  – Treatment for specific problems such as seizures and ventricular dysrhythmias is complex
    • May require combination of alkalinization and anticonvulsants
    • Rapid transport to emergency department is most prudent course of action
How can you ensure rapid transport of these patients?

Lithium

- Mood-stabilizing drug sometimes prescribed for management of bipolar disorders
  - Has low toxic-to-therapeutic dose ratio
  - Overdose is common
  - Patients prescribed lithium have frequent blood tests to monitor level of lithium in body

- Helps to prevent mood swings
  - Interferes with hormonal responses to cyclic adenosine monophosphate and by increasing reuptake of norepinephrine
  - Produces antiadrenergic effect
  - As result, has many effects on body
    - Muscle tremor
    - Thirst
    - Nausea
    - Increased urination
    - Abdominal cramping
    - Diarrhea
Lithium

• Toxic ingestion signs and symptoms:
  – Muscle weakness
  – Slurred speech
  – Severe trembling
  – Blurred vision
  – Confusion
  – Seizure
  – Apnea
  – Coma

Lithium

• Prehospital care
  – Airway management
  – Ventilatory and circulatory support
  – Control of seizure activity if present
  – Activated charcoal does not effectively bind lithium and should not be given

Lithium

• In-hospital care
  – Restoring intravascular volume
  – Maintaining urine output
  – Correcting hyponatremia
  – Sometimes dialysis
Cardiac Medications

• Common cause of poisoning deaths in children and adults
  – Digoxin
  – Beta-blockers
  – Calcium channel blockers

• Treatment:
  – High-concentration O₂ administration
  – IV access
  – Careful monitoring of vital signs and ECG

Cardiac Medications

• Digoxin
  – Exerts direct and indirect effects on sinoatrial and atrioventricular nodal fibers
  – At toxic levels can
    • Halt impulses in sinoatrial node
    • Depress conduction through AV node
    • Increase sensitivity of SA and AV nodes to catecholamines
    • Affect Purkinje fibers, decrease resting potential and action potential duration of heart, and increase automaticity
    • Can cause increase in premature ventricular contraction formation

Cardiac Medications

• Can produce almost any dysrhythmia or conduction block
• Common signs and symptoms of toxicity
  – Nausea
  – Anorexia
  – Fatigue
  – Visual disturbances
  – Variety of disorders of GI, ophthalmological, and neurological systems
Cardiac Medications

• Oral overdoses sometimes are managed with activated charcoal and drugs to treat life-threatening dysrhythmias
• Severe overdoses are managed with IV digoxin-specific Fab
  – Decreases the morbidity and mortality associated with digoxin overdose

Why is it possible that this type of overdose (digoxin) would not be detected immediately?

Cardiac Medications

• Beta-blockers
  – Absorbed rapidly after ingestion
  – Toxicity impairs sinoatrial and atrioventricular node function
    • Leads to bradycardias and AV blocks
  – Associated depression in ventricular conduction and sodium channel blockade may cause QRS complex to widen
  – Occasionally, patients become susceptible to ventricular dysrhythmias
    • Rarely will they be ventricular tachycardia or ventricular fibrillation
Cardiac Medications

• Other signs and symptoms
  – CNS and respiratory depression
  – Hypotension
  – Seizures

Cardiac Medications

• Treatment for overdose
  – Activated charcoal administration
  – Drugs to manage hypotension and dysrhythmias
• In-hospital care
  – Infusions of glucagon and various catecholamines
  – Hemodialysis may be necessary, depending on particular agent involved

Cardiac Medications

• Toxic ingestion of calcium channel blockers can lead to
  – Myocardial depression
  – Peripheral vasodilation with negative effects
    • Inotropic
    • Chronotropic
    • Dromotropic
    • Vasotropic
Cardiac Medications

- Toxic ingestion of calcium channel blockers can lead to
  - Hypotension and bradycardia are early signs of toxicity
  - Overdose may result in serious dysrhythmias
    - AV block of all degrees
    - Sinus arrest
    - Atrioventricular dissociation
    - Junctional rhythm
    - Asystole

- Other signs and symptoms
  - Nausea and vomiting
  - Hypotension
  - CNS and respiratory depression

- Treatment
  - Airway, ventilatory, and circulatory support
  - Antidysrhythmics
  - Vasopressors
  - Calcium chloride
  - Activated charcoal

Monoamine Oxidase Inhibitors

- Block breakdown of monoamines
  - Norepinephrine
  - Dopamine
  - Serotonin
  - CNS transmitters are distributed throughout body
    - Highest concentration is in brain, liver, kidneys
Monoamine Oxidase Inhibitors

• Prescribed as
  – Antidepressants
  – Antineoplastics
  – Antibiotics
  – Antihypertensives

Monoamine Oxidase Inhibitors

• Some have active metabolites
  – Phenelzine
  – Tranylcypromine

• Signs of toxicity usually are delayed, presenting 6 to 24 hours after ingestion
  – Duration of effects also may last for several days
    • CNS depression
    • Various neuromuscular and cardiovascular system manifestations

Monoamine Oxidase Inhibitors

• Prehospital care is mainly supportive
  – Airway, ventilatory, and circulatory support
  – Cardiac medications as needed
  – Rapid transport
  – Activated charcoal may be indicated
Nonsteroidal Anti-inflammatory Drugs

- Group of drugs that have analgesic and antipyretic action
  - Reduce inflammation of joints and soft tissues, such as muscles and ligaments
  - Work by blocking production of prostaglandins
    - Chemicals that cause inflammation and trigger transmission of pain signals to brain

Nonsteroidal Anti-inflammatory Drugs

- Widely used to relieve symptoms caused by:
  - Types of arthritis
    - Rheumatoid arthritis
    - Osteoarthritis
    - Gout
  - Treat back pain
  - Menstrual pain
  - Headaches
  - Minor postoperative pain
  - Soft tissue injuries

Nonsteroidal Anti-inflammatory Drugs

- Common NSAIDS
  - Diflunisal
  - Fenoprofen
  - Ibuprofen
  - Naproxen
  - Ibuprofen and naproxen are available over the counter
Ibuprofen Overdose

- One of the most commonly ingested nonsteroidal anti-inflammatory drugs in overdose
  - Effects usually are reversible and seldom life threatening
  - Significant toxicity may result in
    - Coma
    - Seizure
    - Hypotension
    - Acute renal failure
- Chronic and acute ingestion is usually greater than 300 mg/kg
  - In such ingestion, common symptoms include mild GI and CNS disturbances
  - Usually resolve within 24 hours after ingestion

Ibuprofen Overdose

- Other less common effects
  - Mild metabolic acidosis
  - Muscle fasciculations
  - Chills
  - Hyperventilation
  - Hypotension
  - Asymptomatic bradycardia

Ibuprofen Overdose

- Emergency care
  - Gastric decontamination
  - Careful monitoring for secondary complications
    - Hypotension
    - Dysrhythmias
Salicylate Overdose

• Widely available in prescription and over-the-counter products such as
  – Acetylsalicylic acid (aspirin)
  – Cold preparations
  – Oil of wintergreen (methyl salicylate)
  – Combination with some analgesics such as propoxyphene and oxycodone

Salicylate Overdose

• Process of toxicity with salicylate poisoning is complex
  – Toxicity includes
    • Direct CNS stimulation
    • Interference with cellular glucose uptake
    • Inhibition of Krebs cycle enzymes that affect energy production and amino acid metabolism
  – Volume of distribution is dose dependent and usually small
  – With toxic ingestion, redistribution of drug into CNS occurs
    • Prolongs elimination of drug from body

Salicylate Overdose

• Complications
  – CNS stimulation
  – GI irritation
  – Glucose metabolism
  – Fluid and electrolyte imbalance
  – Neurological symptoms
  – Coagulation defects
Salicylate Overdose

• Symptoms
  – Confusion
  – Lethargy
  – Convulsions
  – Respiratory arrest
  – Coma
  – Brain death can occur in severe salicylate poisoning

Salicylate Overdose

• Prehospital care
  – Supportive
  – Activated charcoal for decontamination of GI tract
  – IV administered glucose to manage hypoglycemia
  – Salicylates are weak acids that can be excreted by kidney
    • Medical direction may recommend sodium bicarbonate in effort to produce alkaline urine

Salicylate Overdose

• Definitive care
  – In-hospital intensive care observation
  – Continued support of vital functions
  – Hemodialysis
Would you predict a tachypnea or bradypnea in patients with salicylate overdose? Why?

Acetaminophen Overdose

- Commonly prescribed analgesic and antipyretic agent
  - Available in many prescription and nonprescription preparations
  - Widespread availability accounts for its high incidence in unintentional and intentional poisoning
  - 1 of 10 most commonly used drugs for intentional self-poisoning
    - Associated with significant morbidity and mortality

- Overdose can cause life-threatening liver damage from toxic metabolites if not managed within 16 to 24 hours of ingestion
  - As few as 30 standard-size (325-mg) acetaminophen tablets are toxic in an average adult
  - Present in many drug combinations
    - Darvocet-N
    - Excedrin
    - Sinutab
Acetaminophen Overdose

- Acute ingestion = doses of 140 mg/kg or greater
- Toxic effects can be classified in four stages
  - Begins with mild symptoms that may be overlooked or masked by more dramatic effects of other agents
  - Temporary clinical improvement
  - Peak liver damage
  - If antidote management is started within 8 hours of ingestion, full recovery should occur

Do you think most nonmedical personnel realize that acetaminophen overdose can be lethal?

Acetaminophen Overdose

- Emergency care
  - Respiratory, cardiac, hemodynamic support
  - If ingestion is within 1 hour and patient is alert, medical direction may recommend activated charcoal
  - Patients with progressive toxicity require in-hospital administration of antidote, N-acetylcysteine
Drugs Abused for
Sexual Purposes/Sexual Gratification

• Some drugs are abused for sexual purposes or for sexual gratification
  – Commonly classified by users as “uppers,” “downers,” “all-arounders”
  – Taken alone or in combination to produce one or more of the following effects
    • Sense of euphoria
    • Excitation (“rush”)
    • Relaxation (“blissed out”)
    • Loss of inhibition

Drugs Abused for
Sexual Purposes/Sexual Gratification

• Each has different
  – Chemical structures
  – Mechanisms of action
  – Side effects
    • Problems associated with use can vary greatly

Drugs Abused for
Sexual Purposes/Sexual Gratification

• Signs and symptoms
  – Mild nausea and vomiting
  – Life-threatening respiratory depression
  – Hypotension
  – Methemoglobinemia (elevated serum hemoglobin)
  – Coma
  – Death
Drugs Abused for Sexual Purposes/Sexual Gratification

- Emergency care
  - Mainly supportive
  - Airway, ventilatory, and circulatory support
  - Rapid transport
- Personal safety is primary importance

Lesson 34.5
Ethanol, Alcohol Poisoning, and Toxins

Learning Objectives

- Describe the short- and long-term physiological effects of ethanol ingestion.
- Describe signs, symptoms, and management of alcohol-related emergencies.
- Identify general management principles for the most common toxic syndromes based on knowledge of the characteristic physical findings associated with each syndrome.
Alcoholism

- Alcohol and related illnesses are major problems in U.S.
  - In 2008, 51.6 percent of Americans age 12 and older reported current use of alcohol
  - 23.3 percent of population admitted to binge drinking (5 or more drinks within 2 hours)
  - 23.3 percent of population reported drinking 5 or more drinks per occasion on 5 or more days/month

Alcoholism

- Alcohol and related illnesses are major problems in U.S.
  - Latest statistics report that alcohol is a key factor in
    - 41 percent of vehicle fatalities
    - 68 percent of manslaughters
    - 62 percent of assaults
    - 54 percent of murder attempts
    - 48 percent of robberies
  - Economic cost of alcohol and other drug-related crime is $61.8 billion annually

How many calls have you been on that involved patients intoxicated with alcohol? What kinds of calls were they?
Alcohol Dependence

• Alcohol dependence characterized by
  – Chronic, excessive consumption of alcohol that results in
    • Injury to health
    • Inadequate social function
    • Development of withdrawal symptoms when the patient stops drinking suddenly

Alcohol Dependence

• Estimated 5 million alcohol-dependent persons (1 in 50) live in U.S.
  – Another 10 million have difficult time controlling their consumption of drug
  – Considered chronic, progressive, potentially fatal disease characterized by
    • Remissions
    • Relapses
    • Cures

Alcohol Dependence

• Development can be divided into four main stages that merge imperceptibly
  – Stages time frame may range from 5 to 25 years, average is about 10 years
  – First stage: tolerance of drug develops in heavy social drinker
    • Allows person to consume larger quantities of alcohol before experiencing its ill effects
  – Second stage: Drinker experiences memory lapses relating to events occurring during drinking episodes
Alcohol Dependence

- **Stages**
  - Third stage: loss or lack of control over alcohol
    - Drinker can no longer be certain of discontinuing alcohol consumption at will
    - Begins with prolonged binges of intoxication
    - Associated with mental and physical complications
  - Some drinkers halt their consumption for brief time or permanently during one of first three stages
  - Active ingredient in all alcoholic beverages is ethanol: colorless, flammable liquid produced from fermentation of carbohydrates by yeast

Metabolism

- 80 to 90 percent of ingested alcohol is absorbed within 30 minutes
  - 20 percent absorbed in stomach, rest in small intestine
  - Once absorbed, distributed rapidly throughout vascular space
  - Alcohol reaches virtually every organ system
    - About 3 to 5 percent is excreted unchanged via lungs and kidneys
    - Rest is metabolized in liver to CO₂ and water
    - Actual rate at which alcohol is metabolized depends on individual
    - Metabolism also depends on whether drinker is alcohol dependent

Blood Alcohol Content

- Alcohol content of blood is measured in terms of mass (milligrams) of alcohol per given volume of blood (deciliter)
  - Blood alcohol content used widely to evaluate CNS status of intoxicated person
  - In most states, legal limit of intoxication is 80 mg/dL (equivalent to 0.08 percent)
    - Some states have laws that allow paramedics to assist in conducting breathalyzer or blood tests to detect alcohol or drug intoxication
    - Be well versed in laws of state before assisting with tests
    - Follow established protocols carefully
Can you use an alcohol prep to prepare the site before drawing a blood alcohol specimen? Why?

Medical Consequences of Chronic Alcohol Ingestion

- Alcohol affects nearly every organ system of body
  - Consumption of large amounts of alcohol poses risk for physical and mental disorders
    - Neurological disorders
    - Nutritional deficiencies
    - Fluid and electrolyte imbalances
    - GI disorders
    - Cardiac and skeletal muscle myopathy
    - Immune suppression
    - Affect patient’s ability to tolerate traumatic injury

Neurological Disorders

- Alcohol is potent CNS depressant
  - When consumed in moderate amounts, reduces anxiety and tension
  - Gives most drinkers feeling of relaxation and confidence
  - Initial feelings of well-being progress to
    - Impaired judgment and discrimination
    - Prolonged reflexes
    - Incoordination and drowsiness
    - Ultimately may progress to stupor and coma
Neurological Disorders

- Long-term neurological effects
  - Short-term memory deficit
  - Problems with coordination
  - Difficulty with concentration and abstraction

Nutritional Deficiencies

- Alcohol can satisfy caloric requirements for brief time
  - Does not have essential vitamins, proteins, fats
  - Alcohol-dependent persons may have decreased dietary intake and malabsorption
  - Leads to multiple vitamin and mineral deficiencies, which can cause
    - Altered immunity
    - Poor wound healing
    - Anorexia
    - Cardiac dysrhythmias
    - Seizures

Wernicke-Korsakoff Syndrome

- Alcohol-dependent persons at risk of developing Wernicke-Korsakoff syndrome
  - Results from reduction in intestinal absorption and metabolism of thiamine caused by alcohol
  - Affects brain and nervous system
  - Disrupts central and peripheral nerve function
  - Consists of two stages
    - Wernicke's encephalopathy
    - Korsakoff's psychosis
    - Combination
Wernicke-Korsakoff Syndrome

• Wernicke's encephalopathy
  – Usually develops suddenly with clinical manifestations of
    • Ataxia
    • Nystagmus
    • Disturbances of speech and gait
    • Signs of neuropathy (paresthesias, impaired reflexes)
    • Stupor
    • Rarely, coma

Wernicke-Korsakoff Syndrome

• Wernicke's encephalopathy
  – Because body needs thiamine to metabolize sugar, may be caused by IV administration of glucose or glucose-containing fluids in malnourished patient
  – Coma may be sole manifestation of Wernicke's encephalopathy
    • Medical direction may recommend IV administration of thiamine before giving IV glucose in patients with altered mental status or coma of unknown origin

Why do you think recognizing Wernicke-Korsakoff syndrome is delayed in alcoholic patients?
Wernicke-Korsakoff Syndrome

• Korsakoff's psychosis
  – Mental disorder often found with Wernicke's encephalopathy
  – Signs
    • Apathy
    • Poor retentive memory
    • Retrograde amnesia
    • Confabulation (invention of stories to make up for gaps in memory)
    • Dementia
  – Usually considered irreversible
    • Leaves patient permanently handicapped by memory loss, requiring continual supervision

Fluid and Electrolyte Imbalances

• Urinary output increases after ingesting alcohol, over and above that expected from amount of fluid ingested
  – Diuresis results because alcohol blocks secretion of antidiuretic hormone
    • Can lead to dehydration and electrolyte imbalances

GI Disorders

• Effects of alcohol on GI system can produce alcohol-related illnesses and diseases
  – GI hemorrhage
  – Cirrhosis
  – Acute or chronic pancreatitis
GI Hemorrhage

• Primary causes of GI hemorrhage in patients who drink alcohol
  – Gastritis and ulcer formation
  – Esophageal tear (Mallory-Weiss syndrome)
  – Variceal hemorrhage

GI Hemorrhage

• Gastritis results from toxic effects of ethanol on gastric mucosa
  – Leads to diffuse or localized areas of erosion
  – In chronic form, blood may ooze continually from mucosal lining, ulcers may develop

GI Hemorrhage

• Esophageal tears of the gastroesophageal junction, stomach, or esophagus usually follow severe or protracted vomiting or retching
  – Injury results when gastric contents are forced against unrelaxed gastroesophageal junction
    • Produces sudden increase in pressure and mucosal tear with subsequent bleeding
    • Bleeding can be worsened by clotting abnormalities
    • Abnormalities are common in patients with alcoholic liver disease
GI Hemorrhage

- Varices are result of portal hypertension caused by cirrhosis
  - Thin-walled, blood-engorged veins subject to rupture and hemorrhage
  - Most common site is varices of esophagus
    - One of most difficult conditions to manage
    - Severe blood loss through vomiting requires aggressive supportive care with large-bore IV lines and fluid resuscitation
    - Permissive hypotension to maintain systolic BP of 80 to 90 mm Hg prudent
    - Consult with medical direction or follow established protocol

Cirrhosis

- Cirrhosis of liver
  - Caused by chronic damage to liver cells and eventual necrosis
  - Bands of fibrous scar tissue develop and break up normal structure of liver
  - Leads to portal hypertension
  - Results in complications
    - Ascites
    - Splenomegaly
    - Bleeding esophageal and gastric varices

Cirrhosis

- May lead to hepatic encephalopathy
  - Caused by accumulation of toxic metabolic waste products that normally would be detoxified by healthy liver
- 12th leading cause of death by disease
  - 27,000 deaths each year
Acute or Chronic Pancreatitis

• Alcohol is most common cause of acute and chronic pancreatitis
  – Usually produces same symptoms as acute form
  – Pain may last from several hours to several days
  – Attacks also become more frequent as condition progresses

• Effects
  – Malabsorption
  – Electrolyte imbalances
  – Diabetes mellitus may develop from insufficient insulin production

• Complications
  – Hemorrhagic pancreatitis
  – Sepsis
  – Pancreatic abscess
  – Associated with high mortality

Cardiac and Skeletal Muscle Myopathy

• Cardiac and skeletal muscle damage related to alcohol abuse
  – Thought to result from direct toxic effect of alcohol or its metabolites
  – In heart muscle, can result in
    • Decreased force of contraction
    • Dysrhythmias
    • Tendency to develop congestive heart failure
  – In skeletal muscle, major symptoms are
    • Weakness and muscle wasting
Immune Suppression

• Long-term alcohol abuse
  – Renders immune system less effective
  – Suppresses bone marrow production of white blood cells
  – RBCs and platelet production often decreased
  – Has direct, specific effects on lung tissue
    • May impair macrophage mobilization and protective ciliary function
    • Ability of body to fight pulmonary infection is lowered
    • Makes alcoholic more susceptible to viral and bacterial pneumonia

For what other pulmonary disease is the immune-suppressed alcoholic patient at risk?

Trauma

• Alcohol suppresses clotting factors produced in liver
  – Blood-clotting deficiency makes alcoholics prone to bruising and internal hemorrhage
  – Adds to frequency of subdural bleeding, even after relatively minor head trauma
Alcohol Emergencies

- Several other conditions caused by consumption or abstinence from alcohol may require emergency care
  - Acute alcohol intoxication
  - Alcohol withdrawal syndromes
  - Disulfiram-ethanol reaction

Acute Alcohol Intoxication

- Ingestion may cause acute poisoning if consumed in large amounts over short period
  - At toxic levels, may develop
    - Hypoventilation (including respiratory arrest)
    - Hypotension
    - Hypothermia

Acute Alcohol Intoxication

- Evaluate for hidden trauma and coexisting medical conditions
  - Hypoglycemia
  - Cardiac myopathy and dysrhythmias
  - GI bleeding
  - Polydrug abuse
  - Ethylene glycol or methanol ingestion
  - Never assume that intoxicated patient is merely inebriated
Management

• Mildly intoxicated patient may need to be transported for physician evaluation
  – In most cases, management requires patient observation in emergency department only until patient is sober
  – Monitor vital signs and level of consciousness carefully en route
  – Physical examination is warranted to rule out illness or injury masked by alcohol ingestion

Management

• Care of acutely intoxicated patient aimed at protecting patient from further injury and maintaining vital functions
• If patient is conscious and agitated, restraints may be necessary
  – If physical restraint becomes necessary, police should be summoned

Management

• Primary survey and resuscitation
  – Rapidly evaluate airway patency with spinal precautions
  • Assess ventilatory and hemodynamic status while obtaining history
  • Patient’s account of event may be unreliable because of alcohol ingestion
  – Initiate IV therapy
  • Draw blood samples for laboratory analysis
  • If hypoglycemia is confirmed, administer thiamine, dextrose 50 percent per protocol
  • Give naloxone if opioid overdose is suspected
Management

- Primary survey and resuscitation
  - Continually monitor patient's airway and provide adequate ventilatory and circulatory support as needed
  - Be prepared to provide suction and aggressive airway management
  - Monitor ECG for dysrhythmias
  - Rapidly transport

Alcohol Withdrawal Syndromes

- Period of relative or full abstinence from alcohol may cause withdrawal in alcoholic
  - Mediated by several mechanisms that result in CNS hyperexcitability
  - Biochemical changes such as respiratory alkalosis and hypomagnesemia may also play role
  - Syndromes can be divided into four general categories
    - Minor reactions
    - Hallucinations
    - Alcohol withdrawal seizures
    - Delirium tremens

Minor Reactions

- Minor reactions begin about 6 to 12 hours after cessation or reduction of alcohol intake
  - Symptoms peak within 24 to 36 hours
    - May persist for 10 to 14 days
    - When alcohol withdrawal is confined to minor reactions, prognosis for full recovery is excellent with proper management
Minor Reactions

- Minor reactions
  - Facial flushing
  - Diaphoresis
  - Nausea and vomiting
  - Slight disorientation
  - Generalized tremor made worse by agitation
  - Mild tachycardia
  - Hypertension
  - Hyperreflexia

Hallucinations

- Usually occur 12 to 24 hours after patient stops drinking alcohol
- Disorders of perception are common
  - May vary from auditory and visual illusions to frank hallucinations
  - Latter can produce agitation, fear, panic
  - During this period, patient may show signs of suicidal and homicidal tendencies, minor reactions may be more pronounced
  - Prognosis is same as for minor reactions with appropriate care

Alcohol Withdrawal Seizures

- Alcohol withdrawal seizures ("rum fits") usually occur 24 to 48 hours after ethanol cessation
  - Most often are grand mal of short duration
  - Status seizures are rare
  - Category of withdrawal may be self-limiting or may progress to delirium tremens with or without lucid interval
Alcohol Withdrawal Seizures

- Alcohol withdrawal seizures ("rum fits") usually occur 24 to 48 hours after ethanol cessation
  - Because of high drug tolerance level of alcoholic, seizure activity may require IV administration of large doses of diazepam or lorazepam
    * May synergistically interact with any ethanol still in patient’s system
    * Vital signs, respirations, mental status should be monitored closely

Delirium Tremens

- Most dramatic and serious form of alcohol withdrawal
  - Affects about 5 percent of all alcoholics hospitalized for withdrawal
  - Usually occurs 48 to 72 hours after cessation of alcohol
    * May be delayed up to 14 days

Delirium Tremens

- Most dramatic and serious form of alcohol withdrawal
  - Characterized by
    * Psychomotor, speech, and autonomic hyperactivity
    * Profound confusion
    * Disorientation
    * Delusional
    * Vivid hallucinations
    * Tremor
    * Agitation
    * Insomnia
Delirium Tremens

- Single episode may last 1 to 3 days and, with multiple recurrences, may last up to 1 month
- True medical emergency
  - Mortality rate that approaches 15 percent
  - Associated alcohol-related illnesses are frequent contributing causes of death
    - Pneumonia
    - Pancreatitis
    - Hepatitis

Management

- Prehospital care
  - Mainly supportive
  - Scene safety
  - Monitor patient’s airway, ventilatory, and circulatory status
  - IV therapy should be initiated with saline solution for rehydration
  - Pharmacological therapy may be indicated for
    - Altered level of consciousness
    - Dysrhythmias
    - Seizure activity

Management

- Need calm reassurance and frequent reorientation
  - All patients with signs and symptoms require physician evaluation
  - Benzodiazepines (e.g., oxazepam, lorazepam, and diazepam) often prescribed at regular intervals to help control withdrawal symptoms
Disulfiram-Ethanol Reaction

• Disulfiram (tetraethylthiuram disulfide [Antabuse]) is medication prescribed to some alcoholic patients to help them abstain
  – Drug works by inhibiting ethanol metabolism and by allowing accumulation of metabolite acetaldehyde
  – Acetaldehyde produces ill effects on GI, cardiovascular, ANS
    • Metabolic product thought to be responsible for common "hangover"
    • Patients who take disulfiram and then drink alcohol experience unpleasant and potentially life-threatening physiological response

Disulfiram-Ethanol Reaction

• Reaction begins 15 to 30 minutes after ingestion of 2 to 5 alcoholic drinks
  – Continues for 1 to 2 hours
  – Causes patient to experience
    • Vertigo
    • Headache
    • Vomiting
    • Flushing, which may give skin "lobster-red" appearance

Disulfiram-Ethanol Reaction

• Reaction begins 15 to 30 minutes after ingestion of 2 to 5 alcoholic drinks
  – Other effects
    • Dyspnea
    • Diaphoresis
    • Abdominal pain
    • Chest pain
Disulfiram-Ethanol Reaction

• More serious reactions
  – Hypotension
  – Shock
  – Dysrhythmias
  – Sudden death
  – Myocardial and cerebral infarction
  – Cerebral hemorrhage
• Reactions have been reported after as little as one drink of ethanol in patients taking high doses of disulfiram
• Acute overdose of disulfiram is now uncommon with current dosing regimens

Management

• Prehospital care
  – Airway, ventilatory, and circulatory support
  – IV fluids to manage hypotension
  – Pharmacological therapy as needed to manage dysrhythmias
  – Rapid transport
  – Most patients recover from episodes
  – Supportive care and in-hospital observation are usually all that are required

General Management Principles for Toxic Syndromes

• Most poisoned patients require only supportive therapy to recover, regardless of toxic agent
  – Grouping toxic agents and physical findings into toxic syndromes or toxidromes can give important clues to what type of poison or toxin involved
  – Will aid in remembering assessment and management strategies
General Management Principles for Toxic Syndromes

• Five toxic syndromes
  – Cholinergic
  – Anticholinergic
  – Hallucinogenic
  – Opioid
  – Sympathomimetic

Cholinergics

• Exposure to cholinergics uncommon
  – Important to recognize cholinergic poisoning so that lifesaving care can be initiated
  – Causative agents
    • Pesticides: organophosphates, carbamates
    • Nerve agents: sarin and soman

Cholinergics

• Assessment findings
  – Headache
  – Dizziness
  – Weakness
  – Bradycardia
  – Nausea
• “Wet” presentation manifested by profound salivation, lacrimation, urination, defecation, gastrointestinal upset, and emesis
  • SLUDGE
Cholinergics

- In severe cases, coma and convulsions may be present
  - Treatment
    - Airway, ventilatory, and circulatory support
    - Decontamination
    - Atropine
    - Pralidoxime
    - Diazepam or lorazepam
    - Activated charcoal

Anticholinergics

- Exposure fairly common because so many medications and plants have anticholinergic properties
  - Antihistamines
  - Antipsychotics
  - Antispasmodics
  - Cyclic antidepressant
  - Plants
    - Jimson weed
    - Night blooming Jessamine
    - Panther mushroom
    - Angel’s trumpet

Anticholinergics

- Signs and symptoms
  - Tachycardia
  - Dry, flushed skin
  - Dilated pupils
  - Facial flushing
- “Dry” patient presentation usually managed with airway, ventilatory, circulatory support
  - Physostigmine may be given as antidote in rare cases, in absence of tricyclic antidepressant overdose
What kinds of feelings do you think the patient and the patient’s family may be having during withdrawal reactions?

### Hallucinogens
- Common hallucinogens
  - LSD
  - PCP
  - Peyote
  - Mushrooms
  - Mescaline

### Hallucinogens
- Depending on agent and dose, signs and symptoms
  - CNS stimulation and/or depression
  - Behavioral disturbances
  - Delusions
  - Hypertension
  - Chest pain
  - Tachycardia
  - Seizures
  - Respiratory and cardiac arrest
Hallucinogens

- Prehospital care
  - Focused on ensuring personal safety
  - Providing airway, ventilatory, and circulatory support

Opioids

- Opioid syndrome carries a hallmark triad of
  - Depressed level of consciousness
  - Respiratory depression
  - Pinpoint pupils

Opioids

- Common causative agents
  - Heroin
  - Morphine
  - Codeine
  - Meperidine
  - Propoxyphene
  - Oxycodone
  - Hydrocodone
  - Fentanyl
Opioids

• Drugs in this class often are mixed with alcohol or other drugs
  – Leads to
    • Increased respiratory depression
    • Hypotension
    • Bradycardia

• Other signs and symptoms
  – Euphoria
  – Nausea
  – Pinpoint pupils
  – Seizures

• Treatment
  – Ensure airway, ventilatory, and circulatory support
  – Naloxone or another opioid-specific antidote agent

Sympathomimetics

• Sympathomimetic syndrome
  – Usually results from acute overdose of amphetamines or cocaine
  – Signs and symptoms
    • Elevated BP
    • Tachycardia
    • Dilated pupils
    • Altered mental status, including paranoid delusions
    • In severe cases, cardiovascular collapse
Sympathomimetics

• Management
  – Personal safety
  – Airway, ventilatory, and circulatory support

Why is it important to be able to identify these toxic syndromes?

Summary

• Poison is any substance that produces harmful physiological or psychological effects
• Goal of managing serious poisonings by ingestion is to prevent the toxic substance from reaching the small intestine, which limits its absorption
Summary

• Toxic effects of ingested poisons may be immediate or delayed
  – Depends on substance ingested
  – Main goal is to identify effects on three vital organ systems most likely to produce immediate morbidity and mortality
    • Respiratory system, cardiovascular system, central nervous system

Summary

• Strong acids and alkalis may cause burns to the mouth, pharynx, esophagus, and sometimes upper respiratory and GI tracts
  – Prehospital care is usually limited to airway and ventilatory support, IV fluid replacement, and rapid transport to appropriate medical facility

Summary

• Most important physical characteristic in potential toxicity of ingested hydrocarbons is its viscosity
  – Lower the viscosity, the higher the risk of aspiration and associated complications
  – Hydrocarbon ingestion may involve the patient’s respiratory, gastrointestinal, and neurological systems
  – Clinical features may be immediate or delayed in onset
Summary

• Methanol is a poisonous alcohol
  – Found in a number of products
  – Methanol itself is no more toxic than ethanol
    • Metabolites (formaldehyde and formic acid) are very toxic
  – Ingestion can affect central nervous system, gastrointestinal tract, and eyes
    • Can cause development of metabolic acidosis

Summary

• Ethylene glycol toxicity is caused by the buildup of toxic metabolites, especially glycolic and oxalic acids after metabolism
  – Occurs mainly in liver and kidneys
  – Toxicity may affect central nervous system and cardiopulmonary and renal systems
  – May result in hypocalcemia

Summary

• Majority of isopropanol (isopropyl alcohol) is metabolized to acetone after ingestion
  – Isopropanol poisoning affects several body systems, including central nervous, gastrointestinal, and renal systems
Summary

- Infants and children are high-risk groups for accidental iron, lead, and mercury poisoning
  - Due to their immature immune systems or increased absorption as a function of age
  - Ingested iron is corrosive to gastrointestinal tract mucosa
  - May produce lethal GI hemorrhage, bloody vomitus, painless bloody diarrhea, and dark stools

Summary

- Food poisoning is a term used for any illness of sudden onset (usually associated with stomach pain, vomiting, and diarrhea) suspected of being caused by food eaten within the previous 48 hours
  - Food poisoning can be classified as infectious
    - Results from bacterium or virus
  - Can be classified as noninfectious
    - Results from toxins and pollutants

Summary

- Toxic effects of major poisonous plant ingestions are predictable
  - Categorized by chemical and physical properties of plant
  - Most responses are consistent with type of major toxic chemical component in plant
Summary

• Concentration of a chemical in the air helps to predict the severity of an inhalation injury
  – Duration of exposure helps to determine this as well
  – Solubility also influences extent of inhalation injury
  – Highly reactive chemicals cause more severe and rapid injury than less-reactive chemicals
  – Properties that determine chemical reactivity are chemical pH; direct-acting potential of chemicals; indirect-acting potential of chemicals; and allergic potential of chemicals

Summary

• Cyanide refers to number of highly toxic substances that contain the cyanogen chemical group
• Regardless of route of entry, cyanide is rapidly acting poison
  – Combines and reacts with ferric ions of respiratory enzyme cytochrome oxidase
  – Inhibits cellular oxygenation
  – Can produce rapid progression from dyspnea to paralysis, unconsciousness, and death

Summary

• Ammonia is toxic irritant
  – Causes local pulmonary complications after inhalation
  – In severe cases, bronchospasm and pulmonary edema may develop
• Hydrocarbon inhalation may cause aspiration pneumonitis
  – Has potential for systemic effects such as CNS depression and liver, kidney, or bone marrow toxicity

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Summary

• Simple asphyxiants cause toxicity by lowering ambient oxygen concentration
  – Chemical asphyxiants possess intrinsic systemic toxicity
  – Toxicity occurs after absorption into circulation
  – Irritants or corrosives cause cellular destruction and inflammation as they come into contact with moisture in respiratory tract

Summary

• General principles of managing inhaled poisons are same as for any other hazardous materials incident
• Hymenoptera and arachnida cause highest incidence of need for emergency care
  – Arthropod venoms are complex and diverse in chemistry and pharmacology
  – May produce major toxic reactions in sensitized persons
  – Such reactions include anaphylaxis and upper airway obstruction

Summary

• Two main families of venomous snakes indigenous to U.S. are pit vipers and coral snakes
  – Pit viper venom can produce various toxic effects on blood and other tissues
    • Include hemolysis, intravascular coagulation, convulsions, and acute renal failure
  – Venom of coral snake is mainly neurotoxic
    • Signs and symptoms range from slurred speech, dilated pupils, and dysphagia to flaccid paralysis and death
Summary

• Marine animals most likely to be involved in human poisonings in U.S. coastal waters are coelenterates, echinoderms, and stingrays
  – Coelenterate envenomation ranges in severity from irritant dermatitis to excruciating pain, respiratory depression, and life-threatening cardiovascular collapse

Summary

• Echinoderm toxins may cause immediate intense pain, swelling, redness, aching in affected extremity, and nausea
  – Delayed effects may include respiratory distress, paresthesia of lips and face, and, in severe cases, respiratory paralysis and complete atonia
  – Locally, stingray venom produces painful traumatic injury
  – May cause bleeding and necrosis
  – Systemic manifestations range from weakness and nausea to seizures, paralysis, hypotension, and death

Summary

• Organophosphates and carbamates inhibit effects of acetylcholinesterase
  – Mnemonic aid that may help paramedic recognize this type of poisoning is SLUDGE (salivation, lacrimation, urination, defecation, gastrointestinal upset, and emesis)
  – Most specific findings, however, are miosis, rapidly changing pupils, and muscle fasciculation
Summary

• General principles for managing drug abuse and overdose include scene safety; ensuring adequate airway, breathing, and circulation; history; substance identification; focused physical exam; initiation of IV; administration of antidote if needed; prevention of further absorption; and rapid transport

Summary

• Narcotics are CNS depressants
  – Can cause life-threatening respiratory depression
  – In severe intoxication, hypotension, profound shock, and pulmonary edema may be present
  – Naloxone is a pure narcotic antagonist effective for virtually all narcotic and narcotic-like substances

Summary

• Sedative-hypnotic agents include benzodiazepines and barbiturates
  – Signs and symptoms of sedative-hypnotic overdose are chiefly related to central nervous system and cardiovascular symptoms
  – Flumazenil (Romazicon) is a benzodiazepine antagonist
  – Useful in reversing effects of these agents if they were given in clinical setting
Summary

• Commonly used stimulant drugs are those of amphetamine family
  – Adverse effects include tachycardia, increased blood pressure, tachypnea, agitation, dilated pupils, tremors, and disorganized behavior
  – With sudden withdrawal, patient becomes depressed, suicidal, incoherent, or near coma

Summary

• Phencyclidine (PCP) is a dissociative analgesic with sympathomimetic and CNS stimulant and depressant effects
  – In low doses, PCP intoxication produces unpredictable state that can resemble drunkenness (and rage)
  – High-dose intoxication may cause coma
    • May last from several hours to days
    • Respiratory depression, hypertension, and tachycardia may be present
    • PCP psychosis is psychiatric emergency and may mimic schizophrenia

Summary

• Hallucinogens are substances that cause distortions of perceptions
  – Depending on agent, overdose may range from visual hallucinations and anticholinergic syndromes to more serious complications, including psychosis, flashbacks, and respiratory and CNS depression
Summary

- Tricyclic antidepressant toxicity is thought to result from central and peripheral atropine-like anticholinergic effects and direct depressant effects on myocardial function
  - Prolonged QRS complex, a GCS score lower than 8, or both should alert paramedic to major tricyclic antidepressant toxicity

Summary

- Lithium is a mood-stabilizing drug
  - Toxic ingestion can include CNS effects that can range from blurred vision and confusion to seizure and coma
- Cardiac drugs are common cause of poisoning deaths in children and adults
  - Drugs responsible for majority of these fatalities are digitalis, beta blockers, and calcium channel blockers

Summary

- MAO inhibitors block or diminish activity of monoamines (norepinephrine, dopamine, serotonin)
  - Toxic effects include CNS depression and various neuromuscular and cardiovascular system manifestations
Summary

• Nonsteroidal anti-inflammatory drugs (NSAIDs) work by blocking production of prostaglandins
  – Effects of overdose of ibuprofen are usually reversible, are seldom life-threatening, and include mild GI and CNS effects
  – Salicylate poisoning may cause CNS stimulation, GI irritation, glucose metabolism, fluid and electrolyte imbalance, and coagulation defects

Summary

• Acetaminophen overdose may cause life-threatening liver damage
  – Results from formation of hepatotoxic intermediate metabolite if it is not managed within 16 to 24 hours of ingestion
• Some drugs are abused for sexual purposes or for sexual gratification
  – Commonly classified by users as “uppers,” “downers,” and those that have more than one primary effect (“all-arounders”)
  – Problems associated with their use vary widely

Summary

• Alcohol dependence is disorder characterized by chronic, excessive consumption of alcohol that results in injury to health or in inadequate social function and development of withdrawal symptoms when patient stops drinking suddenly
Summary

- Alcohol causes multiple systemic effects
  - include neurological disorders, nutritional deficiencies, fluid and electrolyte imbalances, gastrointestinal disorders, cardiac and skeletal muscle myopathy, and immune suppression
  - Several conditions caused by consumption or abstinence from alcohol that may require emergency care are acute alcohol intoxication, alcohol withdrawal syndromes, and disulfiram-ethanol reaction

Summary

- Most common toxic syndromes are cholinergic, anticholinergic, hallucinogenic, opioid, and sympathomimetic
  - Using these classifications allows paramedic to group similar toxic agents together
  - Enables paramedic to more easily remember how to assess and treat poisoned patient

Questions?