



Southern Regional Disaster
Response System
HHS Region 4

Chemical Hazard Clinical Care Guidelines

Table of Contents

Author Page	3
Crowd Control Agents	6
Cyanide	11
Hallucinogens	17
Hydrofluoric Acid Exposure	21
Hydrogen Sulfide Gas	26
Irritants	30
Opioid and Fentanyl Analogs	41
Organic Phosphorus Compounds	44
Phosphide Salts or Gas	54
Sedatives	58
Stimulant Poisoning or Overdose	61
Sulfur Mustard, Lewisite	67
Unknown Chemical Agents	72
White Phosphorus	82

Author Page

Lead Authors: Ziad Kazzi, MD, FAAEM, FACER, FACMT

Esther Hwang, DO, MPH, FACEP & Irfan Husain, MD, MPH, FACEP

Crowd Control Agents

Esther Hwang, DO, MPH, FACEP -Assistant Professor, Emory University School of Medicine
Department of Emergency Medicine Section of Prehospital and Disaster Medicine

Jessica Rivera, PharmD, DABAT -Clinical Director, Alabama Poison Information Center at
Children's of Alabama Assistant Professor, Office of Medical Toxicology, Department of
Emergency Medicine, University of Alabama at Birmingham

Cyanide

Irfan Husain, MD, MPH, FACEP Associate Medical Director, Sandy Springs Fire
Department Associate Medical Director, MetroAtlanta Ambulance Service Assistant
Professor of Emergency Medicine Emory University School of Medicine

Robert J. Geller, MD, FAAP, FAACT, FACMT -Administrative Medical Director, Georgia
Poison Center, Professor of Pediatrics Emory University School of Medicine

Hallucinogens (e.g., anticholinergics, PCP, ketamine)

Andre Pennardt, MD, FACEP, FAEMS, Professor of Emergency Medicine and Critical Care,
Florida International University Herbert Wertheim College of Medicine

Jeffrey N. Bernstein, MD, FACMT, FAACT, Medical Director, Florida Poison Information
Center, Miami Attending Physician, Emergency Care Center Jackson Memorial Hospital

Hydrofluoric Acid Exposure

Aaron R. Kuzel, DO, MBA- Assistant Professor, University of Louisville School of Medicine

Brent Morgan, MD, FACMR -Administrative Medical Director, Georgia Poison Center and
Emory University

Hydrogen Sulfide Gas

Malcolm Velasco, MD- Emergency Medical Services Fellow ,Emory University

Stephanie Hon, PharmD, DABAT, FAACT- Director, Georgia Poison Center, Grady Health
System

Irritants

Brent Morgan, MD, FACMR -Administrative Medical Director, Georgia Poison Center and Emory University

Opioid and Fentanyl Analogs

Peter Akpunonu, MD- Medical Director, Kentucky Poison Control Center

Ashley Webb, PharmD, FAACT- Executive Director, Kentucky Poison Center-Louisville, KY

Organic Phosphorus Compounds

Douglas Chesson, MD- Medical Director, Emory Johns Creek Hospital

Justin Arnold, DO- Medical Director, Florida Poison Information Center- Tampa

Alexandra Funk, PharmD- Managing Director, Florida Poison Information Center- Tampa

Phosphide Salts or Gas

Jonathan de Olano, MD, Assistant Professor of Emergency Medicine and Medical Toxicologist, Emory University School of Medicine, Georgia Poison Center

Sedatives

Sophia Sheikh MD-Associate Professor, Medical Director, Department of Emergency Medicine, University of Florida College of Medicine-Jacksonville

Dawn Sollee PharmD-Department of Emergency Medicine, University of Florida College of Medicine - Jacksonville

Nathan Roney MD- Assistant Professor, Department of Emergency Medicine, University of Florida College of Medicine - Jacksonville

Christine Gage DO-Assistant Professor, Department of Emergency Medicine, University of Florida College of Medicine-Jacksonville

Reeves Simmons PharmD-Assistant Director, Florida Poison Information Center- Jacksonville

Stimulant Poisoning/Overdose

Justin Arnold, DO- Medical Director, Florida Poison Information Center- Tampa

Alexandra Funk, PharmD- Managing Director, Florida Poison Information Center- Tampa

Rachel Semmons, MD – Associate Professor, Tampa Fire Rescue, Tampa General Hospital, University of South Florida

Sulfur Mustard, Lewisite

Patrick C Filkins, PharmD, BCPS, DABAT-Clinical Toxicologist, Operations and Special Projects Manager | Georgia Poison Center Emergency Medicine Clinical Pharmacist | Grady Memorial Hospital Adjunct Instructor of Emergency Medicine | Emory University School of Medicine

Irfan Husain, MD, MPH, FACEP-Associate Medical Director, MetroAtlanta Ambulance Service Associate Medical Director, Sandy Springs Fire Department Assistant Professor of Emergency Medicine Emory University School of Medicine

Unknown Chemical Agents

Michael Marlin, MD- Associate Professor, Department of EM Medical Director, Mississippi Poison Center University of Mississippi Medical Center

James Augustine, MD - Clinical Professor, Dept of Emergency Medicine Wright State University, Dayton, OH

White Phosphorus

Michael C Beuhler, MD-Adjunct Professor of Emergency Medicine, Wake Forest School of Medicine

Daniel Willner, MD, MPH- Assistant Professor of Emergency Medicine, UNC School of Medicine

Some physicians are Emergency Medicine Board Certified with EMS Fellowship Training

Toxicologists are affiliated with a poison control center.

Crowd Control Agents

Introduction

Toxin Name Aliases

- Chemical crowd/riot control agents
- 2-Chloroacetophenone (CN, Mace®) Harassing agents
- Incapacitating agents
- Lacrimators
- O-chlorobenzylidene malononitrile (CS)
- Oleoresin capsicum (OC, pepper spray)
- Tear gas

Patient Care Goals

1. Address side effects of exposed individuals
2. Decontamination of affected individuals
3. Minimize effects to clinician(s)

Patient Presentation

Inclusion Criteria

Exposure to identifiable agents that are not intended to cause significant injury or fatality.

Exclusion Criteria

1. Exposure to chlorine, phosgene, ammonia, nerve gases, or other agents that are intended to cause significant injury or fatality
2. Exposure to an unknown agent

Notes/Education Pearls

Key Considerations

1. CN, CS, and OC are the most encountered riot control agents
2. CN, CS, and OC have a high safety ratio and rapid onset. All three have a high median lethal concentration (LCt50) and a low median effective concentration (ECt50)
3. Toxicity is related to time of exposure and concentration of agent used (exposure in non-ventilated space)
4. Symptoms that may be experienced after exposure:
 - a. **Eyes:** tearing, pain, conjunctivitis, blurred vision, photophobia, blepharospasm
 - b. **Nose/mouth/throat:** rhinorrhea, sneezing, burning/pain, trouble swallowing, hypersalivation
 - c. **Lungs:** chest tightness, coughing, choking sensation, wheezing, dyspnea
 - d. **Skin:** burning, redness, dermatitis, “hot” sensation (with OC)
 - e. **GI:** nausea and vomiting are rare and may be post tussive. Diarrhea can be possible if an agent is accidentally ingested.

5. Symptoms begin within seconds of exposure, are self-limited and are best treated by removing patient from ongoing exposure. Symptoms frequently decrease over time (15–45 minutes) after exposure ends

Pertinent Assessment Findings

1. Riot control agent used
2. Symptoms of exposed
3. Lung sounds
4. Evidence of other traumatic injuries

Patient Management

Assessment

1. Assess scene safety: evaluate for hazards to EMS personnel, patient, bystanders
 - a. Determine number of patients
 - b. Observe symptoms exhibited by the exposed individual(s)
 - c. Determine riot control agent being used
 - d. Don appropriate PPE
 - e. If available, don an air purifying respirator and appropriate eye protection such as well fitted goggles. Skin should be covered with protective coveralls and gloves.
 - f. If PAPRs are not available, don an N95 respirator, appropriate eye protection and skin protection at a minimum.
2. Examine as appropriate to complaints

Treatments and Interventions

1. Move affected individuals from contaminated environment into fresh air if possible. These agents contain particles that are heavier than air and prompt removal of affected individuals to a well-ventilated area is a priority.
2. Remove contaminated clothing and place in airtight bags to prevent secondary exposures as able.
3. Have the patient remove contact lenses, or assist in their removal, if appropriate.
4. Irrigation with water or saline may facilitate resolution of symptoms and is recommended for decontamination of dermal and ocular exposure
5. If a patient is in respiratory distress, refer to your agency's guidelines for respiratory management.
 - a. Respiratory distress includes abnormal respiratory pattern or rate, abnormal depth or equality of respiratory effort, abnormal breath sounds (wheezing, rhonchi, rales or stridor), accessory muscle use, cyanosis.
 - b. Administer oxygen if needed for respiratory distress.
 - c. Provide appropriate maneuvers to maintain a patent airway if needed
 - d. Take into consideration other injuries such as trauma to safely manage an airway
 - e. Bronchodilators and steroids can assist in cases of severe respiratory distress.
6. If the patient is wheezing, refer to your agency's guidelines for bronchospasm.

- a. Inhaled medications for bronchospasm such as albuterol MDI or nebulized bronchodilator can aid in relieving bronchospasm.
 - b. Concurrent use of ipratropium (Atrovent) can be given for up to 3 doses with albuterol.
 - c. Steroid administration should be considered. If available, PO steroids can be given in more stable patients.
7. Dexamethasone 0.6 mg/kg to a max dose of 10 mg IV depending on EMS agency protocol
 8. Can also be given PO
 9. Methylprednisolone 2 mg/kg to a max dose of 125 mg IV
 10. Prednisone 1 mg/kg to a max dose of 60 mg PO
 11. Magnesium Sulfate should be administered for severe respiratory distress and impending respiratory failure (40 mg/kg to a max dose of 2 g IV)
 12. For persistent pain of the eye or skin, refer to your agency's guidelines for Topical Chemical Burns or Burns.
 13. If possible, obtain information on the agent
 14. Carefully remove contaminated clothing and contact lenses (if applicable)
 15. Calculate the estimated total body area involved
 16. Flush the patient's skin and eyes with copious amount of water or normal saline
 17. For eye exposure, if possible, administer continuous water or normal saline irrigation to eye until improvement of symptoms
 18. Provide analgesia per your agency's pain management guidelines
 19. Take measures to minimize hypothermia
 20. Exposed individuals who are persistently symptomatic warrant further evaluation and treatment per local standards. Please refer to your agency's guidelines.

Patient Safety Considerations

1. Toxicity is related to duration of exposure and concentration of agent used (exposure in non-ventilated space)
2. Patients with pre-existing pulmonary conditions (e.g., asthma, COPD) may be prone to more severe respiratory effects
3. Traumatic injuries may result when exposed individuals are in proximity to the device used to disperse the riot control agent (e.g., hose/stream under pressure, riot control agent projectile, grenade)

Quality Improvement

Associated NEMSIS Protocol(s) (eProtocol .01)

- 9914033 - Exposure - Airway/Inhalation Irritants
- 9914041 - Exposure - Chemicals to Eye
- 9914075 - General - Universal Patient Care/ Initial Patient Contact
- 9914111 - Medical - Allergic Reaction/ Anaphylaxis
- 9914139 - Medical - Respiratory Distress/ Asthma/ COPD/ Reactive Airway
- 9914185 - General - Law Enforcement - Assist with Law Enforcement Activity
- 9914191 - Injury - Mass/ Multiple Casualties
- 9914213 - Injury - Topical Chemical Burn

Key Documentation Elements

- Type of riot control agent if known
- Symptoms being treated
- Treatment provided
- Response to treatment

Performance Measures

- Riot control agent identified before making patient contact and providing treatment
- PPE used by responders
- Affected individuals removed from ongoing exposure
- Contaminated clothing and contact lenses removed as able

References

1. Barry JD, Hennessy R, McManus JG Jr. A randomized controlled trial comparing treatment regimens for acute pain for topical oleoresin capsaicin (pepper spray) exposure in adult volunteers. *Prehosp Emerg Care*. 2008 Oct–Dec;12(4):432–7
2. Dimitroglou Y, Rachiotis G, Hadjichristodoulou C. Exposure to the Riot Control Agent CS and Potential Health Effects: A Systematic Review of the Evidence. *Int. J. Environ. Res. Public Health* 2015, 12(2), 1397–1411
3. Menezes RG, Hussain SA, Rameez MA, Kharoshah MA, Madadin M, Anwar N, Senthilkumaran S, Chemical crowd control agents. *Med Leg J*. 2016 Mar;84(1):22–5
4. Riot-control agents. Army.mil. <https://medcoe.army.mil/borden-field-mgt-of-cb-casualties>. Accessed March 11, 2022
5. Riot control agents. Fas.org. <https://fas.org/nuke/guide/usa/doctrine/army/mmcch/RiotAgnt.htm>. Accessed August 29, 2017
6. Riot control agents/tear gas. CDC.gov. <https://emergency.cdc.gov/agent/riotcontrol/factsheet.asp>. Accessed March 11, 2022
7. Schep LJ, Slaughter RJ, McBride DI. Riot control agents: the tear gases CN, CS and OC- a medical review. *J R Army Med Corps*. 2015 Jun;161(2):94–9. <http://jramc.bmj.com/content/161/2/94.long>. Epub 2013 Dec 30. Accessed March 11, 2022
8. Akpunonu PD, Eagar H, Doty B. Managing the effects of riot control agents. EM Resident. <https://www.emra.org/emresident/article/riot-agents> Epub 2020 Jun 05. Accessed October 6, 2024
9. Grill W. Riot Control Agents. ACEP Toxicology Section. <https://www.acep.org/toxicology/newsroom/august-2022/riot-control-agents#:~:text=Pulmonary:%20patients%20with%20underlying%20reactive,10%2C11> Epub 2022 Sep 05. Accessed October 10, 2024
10. Frey AS, Maniscalco PM, Holstege CP. Chemical agents encountered in protests. *Emerg Med Clin of North America*, 2022 40(2):365-379. <https://www.sciencedirect.com/science/article/pii/S0733862722000074> Accessed October 10, 2024

Other Comments/Questions

Click or tap here to enter text.

Cyanide

Introduction

Toxin Name Aliases

- Blood agent
- Cyanide
- Hydrogen cyanide

Patient Care Goals

1. Remove patient from toxic environment.
2. Assure adequate ventilation and oxygen delivery. Oxygen utilization at the cellular level is impaired by cyanide. Delivery of an antidote is necessary to permit cellular metabolism to continue, so antidote administration is an urgent need.
3. Correct hypoperfusion if present.

Patient Presentation

Cyanide can be a colorless hydrogen cyanide gas or white crystal salt (e.g., sodium cyanide or potassium cyanide) which binds to the ferric ion in cells, blocking the enzyme cytochrome oxidase, thus preventing the use of oxygen by the cell's mitochondria, leading to cellular hypoxia. While hydrogen cyanide gas has a characteristic "bitter almond smell", only some people can smell it.

Signs and Symptoms by Body Systems:

- a. CNS - Typical progressive hypoxia including headache, anxiety, agitation, confusion, lethargy, seizures and coma.
- b. Cardiovascular - Initially bradycardia and hypertension may occur, followed by hypotension and tachycardia. The terminal event is consistently bradycardia and hypotension.
- c. Respiratory - Early findings in patients may include increased respiratory rate, shortness of breath, and chest tightness. As poisoning progresses, breathing may become slow and gasping. Central cyanosis may or may not be present, and pulmonary edema can occur.
- d. GI - Following ingestion, symptoms may include abdominal pain, nausea and vomiting.
- e. Skin - A cherry red coloration may appear due to elevated venous hemoglobin oxygen saturation. Cyanide does not directly cause cyanosis; if present, it is typically a result of shock.
- f. Ocular - Direct contact with liquid cyanide can cause eye irritation and swelling.
- g. Children and pregnant women are much more vulnerable than adults to cyanide agent toxicity.

Hydrogen cyanide is highly toxic through all exposure routes. The amount of cyanide, the duration of exposure, and the route of exposure all affect the onset time and severity of illness.

At higher doses, symptoms typically begin in seconds (such as following inhalation of gaseous hydrogen cyanide) and may cause sudden onset of profound CNS, cardiovascular, and respiratory effects, leading to death within minutes. Signs and symptoms may appear over an extended period if the poisoning occurs gradually or involves lower doses. In some circumstances, the patient may even remain asymptomatic.

Inclusion Criteria

1. Depending on its form, cyanide can enter the body through inhalation, ingestion, or absorption through the skin (e.g. acetonitrile). Cyanide should be suspected in occupational or other smoke exposures (e.g., firefighting), industrial accidents, natural catastrophes, suicide and murder attempts, chemical warfare, and terrorism (whenever there are multiple casualties of an unclear etiology). Cyanide is produced from combustion of many nitrogen-containing compounds, including many plastics and from many natural and synthetic fabrics, when combustion conditions permit.
2. **Non-specific and early signs** of cyanide exposure (inhalation, ingestion, or absorption) include the following signs and symptoms: anxiety, vertigo, weakness, headache, tachypnea, nausea, dyspnea, vomiting, and tachycardia.
3. High concentrations of cyanide will produce:
 - a. Markedly altered level of consciousness, including rapid collapse
 - b. Seizures
 - c. Respiratory depression or respiratory arrest
 - d. Cardiac dysrhythmias (other than sinus tachycardia)

Exclusion Criteria

None noted

Patient Management

Assessment

1. Staff must use appropriate PPE for the situation, to prevent them becoming victims as well. When there is risk of exposure to hydrogen cyanide gas, self-contained breathing apparatus is necessary
2. Remove patient from toxic environment.
3. Assess ABCDs and, if indicated, expose the patient, and then re-cover the patient to assure retention of body heat.
4. Assess vital signs (pulse, blood pressure, respiratory rate, neurologic status assessment) including temperature and pulse oximetry (which usually will not correlate with tissue oxygenation in cyanide/smoke exposure).
5. Attach a cardiac monitor and examine rhythm strip for arrhythmias.
6. Perform a 12-lead EKG.

7. Check blood glucose level.
8. Monitor EtCO₂.
9. Pulse oximetry will not accurately reflect patient oxygenation in the presence of cyanide in the body. (Cyanide and carbon monoxide both react with hemoglobin to create cyanohemoglobin and carboxyhemoglobin, and their presence in the circulation interferes with the calculations made by the pulse oximeter).
10. Monitor patient for signs of hypoxia and respiratory decompensation, regardless of pulse oximetry reading.
11. Identify the specific agent of exposure, time of ingestion/inhalation, and quantity/timing of exposure.
12. In a mass casualty incident, only three agents that can be dispersed as aerosols or gases have been identified as potential causes for a group of individuals to simultaneously collapse, lose consciousness, and experience seizures: nerve agents, cyanide, and hydrogen sulfide. In a fire scene, patients remaining hypoxic after delivery of high concentrations of oxygen should lead to suspicion of carbon monoxide and/or cyanide toxicity.
Where feasible, responders should seek help in identifying the chemicals by examining container shapes, placards, labels, shipping papers, and conducting analytical tests. General information on these identification methods can be found in the Emergency Response Guidebook.
13. Obtain patient history including cardiovascular history and prescribed medication.
14. Obtain other pertinent patient history.
15. Perform physical exam. Cherry red skin color if present is suggestive of cyanide toxicity, but its absence should not be used to exclude cyanide toxicity.

Treatments and Interventions

There is **no** widely available, rapid, confirmatory cyanide blood test. Therefore, prehospital treatment decisions must be made based on clinical history and signs and symptoms of cyanide intoxication. If the patient has a relevant exposure history and exhibits one or more significant signs or symptoms of cyanide toxicity, initiate treatment with:

1. 100% oxygen via non-rebreather mask, CPAP, or bag valve mask.
2. If a patient demonstrates cardiopulmonary arrest, ventilate using bag-valve mask; do not use mouth to mouth ventilation. Administer available antidote (antidote options below) as soon as possible. If an arrest occurs en-route and antidote is not available on the EMS unit, call ahead to arrange for antidote to be available for administration upon arrival at the hospital.
3. Collect a pre-treatment blood sample in the appropriate tube for lactate and cyanide levels, if feasible within local EMS agency guidelines and provider scope of practice. Very high serum lactate levels (>8 mmol/L) are highly suggestive of cyanide toxicity.
4. Administer one of the following antidote regimes:
 - a. Hydroxocobalamin (*the preferred agent*)
 - i. **Adult:** Administer hydroxocobalamin
 - a. Initial dose is 5 g administered over 15 minutes slow IV.

- b. Each 5 g vial of hydroxocobalamin is to be reconstituted with 200 mL of LR, NS, or D5W (25 mg/mL) and administered at 10–15 mL/minute.
- c. An additional 5 g dose may be administered with medical consultation.

ii. **Pediatric:** Administer hydroxocobalamin 70 mg/kg (reconstitute concentration is 25 mg/mL)

- a. Each 5 g vial of hydroxocobalamin is to be reconstituted with 200 mL of LR, NS, or DSW (25 mg/mL) and administered at 10–15 mL/minute.

iii. Maximum single dose is 5 g.

iv. Note that hydroxocobalamin is a deeply red colored medication, and its administration may temporarily lead to false results on lab analyses performed using colorimetric assays.

OR

b. Sodium thiosulfate

i. **Adult:** Sodium thiosulfate 12.5 g IVF (50 mL of 25% solution)

ii. **Pediatric:** Sodium thiosulfate 0.5 g/kg IV (2 mL/kg of 25% solution)

OR

c. Sodium Nitrite + Sodium thiosulfate (Nithiodote)

i. **Adult:**

- Sodium Nitrite – 10 mL of sodium nitrite at the rate of 2.5 to 5 mL/minute
- Sodium Thiosulfate – 50 mL of sodium thiosulfate immediately following administration of sodium nitrite

ii. **Pediatric:**

- Sodium Nitrite – 0.2 mL/kg (6mg/kg or 6-8 mL/m² BSA) of sodium nitrite at the rate of 2.5 to 5 mL/minute not to exceed 10 mL
- Sodium Thiosulfate – 1 mL/kg of body weight (250mg/kg or approximately 30-40 mL/m² of BSA) not to exceed 50 mL total dose immediately following administration of sodium nitrite

iii. Redosing: if signs of cyanide poisoning reappear, repeat treatment using one-half the original dose of both sodium nitrite and sodium thiosulfate

1. If seizure, treat per local EMS agency seizure protocol or refer to NASEMSO seizure guideline

Patient Safety Considerations

1. In the event of multiple casualties, be sure to wear appropriate PPE during rescue evacuation from the toxic environment.
2. Patients exposed only to cyanide gas do not need decontamination, as the gas will not further penetrate the body and will not off-gas once the patient is removed from further exposure.
3. If the patient ingests cyanide, it will react with the acids in the stomach generating hydrogen cyanide gas. Be sure to maximize air circulation in closed spaces (ambulance) as the patient's gastric contents may contain hydrogen cyanide gases when released with vomiting or belching.
4. Do not use nitrites in conjunction with suspected carbon monoxide poisoning as it worsens the hemoglobin oxygen carrying capacity even more than carbon monoxide (CO).
5. Hydroxocobalamin is the only agent safe for treatment of cyanide poisoning in pregnant patients.

Notes/Education Pearls

Key Considerations

1. Cyanide toxicity is primarily a clinical diagnosis
2. After hydroxocobalamin has been administered, skin, tears, and urine will all turn red. This flushing should not be interpreted as an allergic reaction.
3. If the patient ingests cyanide, it will react with the acids in the stomach generating hydrogen cyanide gas. Be sure to maximize air circulation in closed spaces (ambulance) as the patient's gastric contents may contain hydrogen cyanide gases when released with vomiting or belching.
4. **Pertinent Assessment Findings**

Early and repeated assessment is essential.

Quality Improvement

Associated NEMIS Protocol(s) (eProtocol .01)

9914043—Exposure - Cyanide

9914141—Medical - Seizure

Key Documentation Elements

- Repeat evaluation and documentation of signs and symptoms as the patient's clinical condition may deteriorate rapidly
- Identification of possible etiology of poisoning
- Time of symptom onset and time of initiation of exposure-specific treatments
- Therapy and response to therapy
- **Performance Measures**
- Early airway management in the rapidly deteriorating patient
- Accurate exposure history
- Time of ingestion/exposure
- Route of exposure
- Quantity of medication or toxin taken (safely collect all possible medications or agents)

- Alcohol or other intoxicant taken
- Appropriate protocol selection and management
- Multiple frequent documented reassessments

References

1. Amyl Nitrite—Medical Countermeasures Database. Chemm.nlm.nih.gov. <https://wwwn.cdc.gov/TSP/MMG/MMGDetails.aspx?mmgid=523&toxid=93>. Accessed March 11, 2022
2. Bebarta VS, Tanen DA, Lairet J, Dixon PS, Valtier S, Bush A. Hydroxocobalamin and sodium thiosulfate versus sodium nitrite and sodium thiosulfate in the treatment of acute cyanide toxicity in a swine (*Sus scrota*) model. *Ann Emerg Med*. 2010; 55(4):345–51
3. Cyanide Poisoning. UpToDate.com. https://www.uptodate.com/contents/cyanide-poisoning?source=search_result&search=cyanide%20and%20pulse%20oximetry&selecte_dTitle=3~150. Updated September 28, 2016. March 11, 2022
4. Geller RJ, Barthold C, Saiers J, Hall AH. Pediatric cyanide poisoning: causes, manifestations, management, and unmet needs. *Pediatrics* 2006;118:2146-2158
5. Hoffman RS, Howland MA, Lewin NA, Nelson LS, Goldfrank LR. *Goldfrank's Toxicologic Emergencies, 10th Edition*. China: McGraw-Hill Education; 2015
6. Marraffa JM, Cohen V, Howland MA. Antidotes for toxicological emergencies: a practical review. *Am J Health Syst Pharm*. 2012;69(3):199–212
7. Meridian Cyanokit (package insert). Semoy, France: Merck Sante. https://www.meridianmeds.com/sites/default/files/pi/CYANOKIT_PI.pdf. Accessed March 11, 2022
8. Roderique EJ, Gebre-Giorgis AA, Stewart DH, Feldman MJ, Pozez AL. Smoke inhalation injury in a pregnant patient: a literature review of the evidence and current best practices in the setting of a classic case. *J Burn Care Res*. 2012; Sep-Oct;33(5):624–33
9. Shepherd G, Velez LI. Role of hydroxocobalamin in acute cyanide poisoning. *Ann Pharmacotherapy*. 2008;42(5):661–9
10. Thompson JP, Marrs TC. Hydroxocobalamin in cyanide poisoning. *Clin Toxicol (Phila)*. 2012;50(10):875–85

Hallucinogens

Introduction

Substances and Common Aliases:

Psilocybin (4-phosphoryloxyN, N-dimethyltryptamine): Magic Mushrooms, Mushrooms, Shrooms, Boomers, and Little Smoke.

LSD (d-lysergic acid diethylamide): Acid, Blotter Acid, Dots, Hits, Microdot, Mellow Yellow, Sugar Cubes, Trips, Tabs, and Window Pane.

DMT (Dimethyltryptamine): Dimitri

Peyote (Mescaline): Buttons, Cactus, Mesc, and Peyoto.

Ayahuasca: (contains DMT): Hoasca, Aya, and Yagé.

Ketamine (Dissociative anesthetic with hallucinogenic effects): Cat Tranquilizer, Cat Valium, Jet K, Kit Kat, Purple, Special K, Special La Coke, Super Acid, Super K, and Vitamin K.

MDMA (3,4-methylenedioxymethamphetamine) (Stimulant with hallucinogenic effects): Ecstasy, Molly, Adam, Beans, Clarity, Disco Biscuit, E, Eve, Hug Drug, Lover's Speed, Peace, STP, X, and XTC.

Synthetic Cannabinoids: K2, Spice.

Patient Care Goals

- Ensure the safety of the patient, including prevention of self-harm, and responders.
- Provide a supportive environment with a minimum of external stressors.
- Stabilize any acute physical or psychological distress.
- Maintain clear communication with receiving facility about patient status.

Patient Presentation

Inclusion Criteria:

- Patient is known to have ingested or been exposed to a hallucinogen.
- Patient is experiencing changes in thought, mood, and perception consistent with a hallucinogen, including synesthesia (transposition of certain sensory nodes such as tasting colors or feeling sounds).
- Toxidrome consistent with hallucinogen exposure, including mydriasis, blurred vision, sweating, flushing, dry mouth, palpitations, tachycardia, hypertension, nausea, diarrhea, tremors, hyperreflexia, mild pyrexia, and impaired coordination.
- Consultation with Poison Control Center suggesting hallucinogen exposure.

Exclusion Criteria

- Symptoms suggestive of a pre-dominant severe stimulant exposure (e.g., severe hyperpyrexia requiring immediate cooling measures, hemodynamic instability such as hypertensive crisis, seizures, tachydysrhythmia, or chest pain).
- Evidence of acute head injury or other significant trauma.
- Known history of severe psychiatric disorders (e.g., schizophrenia, psychosis).
- Suicide attempt with suspected co-ingestants.

Patient Management

Assessment

- **Safety First:** Rapidly assess if the patient presents a threat to themselves and/or others. Request the assistance of law enforcement if necessary. Restrain the patient if necessary for safety. Ensure the patient does not have any weapons.
- Assess and monitor airway, breathing, and circulation (ABCs).
- Obtain and monitor vital signs (heart rate, blood pressure, respiration, oxygen saturation, end tidal CO₂, and temperature).
- Assess mental status: level of consciousness, orientation, and cooperation.
- Assess for hallucinations, agitation, anxiety, or other abnormal behaviors.
- Discuss presentation and management recommendations with Poison Center by calling 800-222-1222 (will connect to local Poison Control Center).

Treatments and Interventions

- If the patient is agitated or anxious, attempt to provide a stress-free environment (e.g., no bright lights or loud noises) and apply verbal de-escalation techniques in a calm and reassuring tone.
- Administer benzodiazepines (e.g., midazolam, lorazepam) if needed to control severe agitation or distress. Refer to NASEMSO's Agitated or Violent Patient/Behavioral Emergency Protocol or your local EMS agency's respective protocols.
- Provide supportive care such as anti-emetics for nausea and vomiting.
- **Physical Restraints:** If necessary to protect the patient and responders, restraints may be applied, following local protocols.
- Avoid restraining the patient in prone position due to risk of asphyxiation.
- **Transport Considerations:**
 - Continuous monitoring of vital signs during transport.
 - Ongoing assessment of mental status and comfort level.
 - Notify the receiving facility of the patient's status and interventions performed.

Transport to higher level of care:

- Patient is unstable or unable to maintain safety due to hallucinations, agitation, or psychosis.
- Abnormal vital signs requiring further evaluation or intervention.

- Concerns about co-ingestion of other substances (e.g., alcohol, drugs) complicating the clinical picture.
- Physical symptoms (e.g., vomiting, dehydration, hyperthermia) requiring medical attention.
- Lack of adequate supervision or safe environment at the scene.

Patient Safety Considerations

Hallucinogens may have profoundly disturbing effects in certain individuals, including panic attacks, amplification of unconscious fears, aggression toward self or others, and profound depression. The altered perception associated with hallucinogens may prevent the patient from ensuring their own safety in a potentially hazardous environment.

Notes/Education Pearls

Key Considerations

Certain stimulants such as MDMA, which may present with hallucinogenic symptoms, may also have significant physiological derangements that include severe hyperthermia. EMS personnel must identify and rapidly cool patients with this condition to reduce morbidity and mortality.

Pertinent Assessment Findings

- Mental status and behavior.
- Vital signs, including any abnormalities or instability.
- Absence of physical injuries.
- Presence of any toxidromes.
- Frequent reassessment of ABCs if sedated or restrained.

Quality Improvement

Associated NEMIS Protocol(s) (eProtocol .01) (www.nemsis.org)

9914225—Medical - Stimulant Poisoning/Overdose

9914135—General - Overdose/Poisoning/Toxic Ingestion

9914053 – General - Behavioral/Patient Restraint

Key Documentation Elements

If sedation or physical restraints are required, document in detail the justification, type and timing of medication or restraints, and ongoing reevaluation.

Performance Measures

- The safety of the patient and responders is protected.
- Vital sign abnormalities are appropriately identified, treated, and documented.
- Supportive care is provided in a low-stress environment.

References

- Baquiran M, Keyes D, Al Khalili Y. Lysergic Acid Diethylamide Toxicity. 2023 Dec 17. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan–. PMID: 31985997.
- Kupas DF, Wydro GC, Tan DK, Kamin R, Harrell IV AJ & Wang A (2021) Clinical Care and Restraint of Agitated or Combative Patients by Emergency Medical Services

Practitioners, Prehospital Emergency Care, 25:5, 721-723, DOI:
10.1080/10903127.2021.1917736

- Prybys KM, Hansen KN. Hallucinogens. Tintinalli JE, Stapczynski JS, Ma OJ, Yealy DM, Meckler GD, Cline DM, eds. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*. 8th ed. New York, NY: McGraw-Hill; 2016. Chapter 188.

Hydrofluoric Acid Exposure

Introduction

Toxin Name Aliases

Chemical Burn

Patient Care Goals

1. Rapid recognition of hydrofluoric acid topical chemical burn, inhalational, and/or oral exposure
2. Initiation of emergent and appropriate intervention and patient transport

Patient Presentation

Inclusion Criteria

1. Patients of all ages who have sustained exposure to hydrofluoric acid that can cause a topical chemical burn, inhalational or ingestion injuries, that may develop immediate or in some cases a delayed clinical presentation

Exclusion Criteria

None noted

Patient Management

1. Don the appropriate PPE
2. Remove the patient's clothing, if necessary
3. Contaminated clothing should preferably be placed in double bags
4. Information regarding hydrofluoric acid (e.g., concentration and amount), type of exposure, and routes of exposure should be gathered while on the scene including materials safety data sheet if available
5. Communicate all data regarding the chemical to the receiving facility
6. Transportation to a receiving facility with decontamination capabilities
7. Discuss presentation and management recommendations with Poison Center by calling 800-222-1222 (will connect to local Poison Control Center).

Assessment

1. Clinical effects and severity of a topical chemical burn is dependent upon:
 - a. Class of agent (alkali injury or acid injury)
 - b. Concentration of the chemical (the higher the concentration, the greater the risk of injury)
 - c. pH of the chemical
 - i. Alkali-increased risk with pH greater than or equal to 11
 - ii. Acid-increased risk with pH less than or equal to 3
 - d. Onset of burn
 - i. Immediate
 - ii. Delayed (e.g., hydrofluoric acid)
 - e. Calculate the estimated total body surface area that is involved
3. Prevent further contamination

4. Special attention to assessment of ocular, inhalational, or oropharyngeal exposure — evaluate for airway compromise secondary to spasm or direct injury associated with oropharyngeal burns
5. Exposure to hydrofluoric acid may manifest systemic effects

Treatments and Interventions

1. If dry chemical contamination, carefully brush off solid chemical prior to flushing the site as the irrigating solution may activate a chemical reaction
2. If wet chemical contamination, flush the patient's skin (and eyes, if involved) with copious amounts of water or normal saline
3. Provide adequate analgesia per the Pain Management Guideline
4. For eye exposure, administer continuous flushing of irrigation fluid to eye at minimum 15-30 minutes of continuous irrigation— Morgan lens may facilitate administration
5. Early airway intervention for airway compromise or bronchospasm associated with oropharyngeal burns or ingestion
6. Take measures to minimize hypothermia
7. Initiate intravenous fluid resuscitation if necessary to obtain hemodynamic stability

Hydrofluoric Acid

1. Hydrofluoric acid (HF) is a highly corrosive substance that is primarily used for automotive cleaning products, rust removal, porcelain cleaners, etching glass, cleaning cement or brick, or as a pickling agent to remove impurities from various forms of steel.
 2. The primary toxicity of hydrofluoric acid, hydrogen fluoride, and all soluble fluorides is due to the fluoride anion. The fluoride anion forms bonds with endogenous magnesium and calcium to produce insoluble forms of magnesium fluoride and calcium fluoride. This formation of insoluble magnesium fluoride and calcium fluoride results in hypocalcemia, hypomagnesemia, and hyperkalemia. These electrolyte abnormalities have a profound effect on the excitable tissues in the nervous system, skeletal system, and cardiac tissues.
 3. Hydrofluoric acid readily penetrates intact skin and there may be underlying tissue injury. It is unlikely that low concentration HF (<10%) will cause an immediate acid-like burn however there may be delayed onset of pain to the exposed area.
 4. Higher concentration HF (>50%) may cause immediate pain and a burn appearance that can range from mild erythema to an obvious burn.
 5. Toxic exposures such as oral, inhalational, or large dermal exposures may lead to seizure, muscle twitching, myocardial irritability, QT interval prolongation, and increased risk for Torsades de Pointes.
1. For all patients in whom a hydrofluoric acid exposure is confirmed or suspected (The implementation of the below specialized treatments will depend on your local EMS protocols and the scope of practice for EMS providers):
 - a. Remove the patient from the toxic atmosphere and initiate decontamination.
 - b. Vigorously irrigate all affected areas with water or normal saline for a minimum of 15 minutes.
 - c. Administer oxygen 100% by non-rebreather as needed/ Intubate if necessary

- d. Apply a cardiac monitor for oral or large dermal exposures significant HF exposures. Monitor ECG and obtain a 12 lead ECG.
 - i. Be prepared to manage ventricular dysrhythmia per current ACLS protocols.
 - ii. Consider Magnesium Sulfate as primary antidysrhythmic if hypomagnesemia is known or suspected.
- e. For patients who have ingested hydrofluoric acid or who have a large dermal exposure consider intravenous calcium gluconate, 1–2 grams of 10% solution, as symptomatic hypocalcemia can precipitate rapidly as manifested by muscle spasms, seizures, hypotension ventricular arrhythmias, and QT prolongation.
- f. Apply calcium preparation
 - i. Calcium prevents tissue damage from hydrofluoric acid.
 - ii. For local dermal exposures with severe pain, apply Calcium Gluconate gel or ointment to the burned area.
 - 1. Topically apply calcium preparation to exposed skin:
 - a. Commercially manufactured calcium gluconate gel
 - b. If commercially manufactured calcium gluconate gel is not available, a topical calcium gluconate gel preparation can be made by combining 150 mL (5 ounces) of a sterile water-soluble gel (e.g., Surgilube® or KY® jelly) with one of the following:
 - i. 35 mL of calcium gluconate 10% solution
 - ii. 10 g of crushed calcium gluconate tablets (e.g., Tums®)
 - iii. 3.5 g calcium gluconate powder or
 - c. If calcium gluconate is not available, 10 mL of calcium chloride 10% solution in 150 mL in sterile water-soluble gel (e.g., Surgilube® or KY® jelly).
 - 2. Apply generous amounts of calcium gluconate gel to the exposed skin sites to neutralize the pain of the hydrofluoric acid.
 - i. Leave in place for at least 20 minutes then reassess
 - ii. This can be repeated as needed
 - 3. Hydrofluoric acid exposure is very painful. Calcium gel is the foundation of pain control. While intravenous pain medications may be less effective, they should be added to calcium gel to assist with pain control. Hydrofluoric acid exposure typically causes pain out of proportion to the visible dermal effects. Minimal skin changes may exist with substantial exposures.
 - 4. If fingers are involved, apply the calcium gel to the hand, squirt additional calcium gel into a surgical glove, and then insert the affected hand into the glove.
 - 5. If a patient has moderate to severe irritation to the conjunctiva and eyes, a Morgan lens may be placed bilaterally and the eyes flushed with a solution of normal saline and 50 ml 10% Calcium Gluconate.
 - a. The therapeutic endpoint for irrigation is a pH of 7 in the conjunctival sac.

6. For patients who have ingested hydrofluoric acid or who have a large dermal exposure consider intravenous calcium gluconate, 1–2 grams of 10% solution, as symptomatic hypocalcemia can precipitate rapidly as manifest by muscle spasms, seizures, hypotension ventricular arrhythmias, and QT prolongation.
7. If concern for inhalational exposure of hydrofluoric acid or respiratory distress following an exposure, can consider the use of nebulized 2.5% calcium gluconate if in protocols or carried.
8. If the patient should develop seizures despite adequate oxygenation and blood glucose level within normal limits, administer benzodiazepines.

Patient Safety Considerations

1. Don PPE
2. Take measures to prevent the patient from further contamination through decontamination
3. Take measures to protect the EMS clinician and others from contamination
 - a. Patient should undergo appropriate decontamination prior to transportation
4. Do not attempt to neutralize an acid with an alkali or an alkali with an acid as an exothermic reaction will occur and cause serious thermal injury to the patient
5. Expeditious transport or transfer to a designated burn center should be considered for burns that involve a significant percentage of total body surface area or burns that involve the eyes, face, hands, feet, or genitals

Notes/Education Pearls

Key Considerations

1. IV fluid resuscitation should be guided by patient age, percentage of body surface area involved in burn, body habitus and calculated by the Parkland Formula [See Appendix VI. Burn and Burn Fluid Charts]
2. Since the severity of topical chemical burns is dependent upon the type, concentration, and pH of the chemical involved as well as the body site and surface area involved, it is imperative to obtain as much information as possible while on scene about the chemical substance by which the patient was exposed. The information gathering process will often include:
 - a. Transport of the original or a copy of the Material Safety Data Sheet (MSDS) of the substance to the receiving facility
 - b. Contacting the reference agency to identify the chemical agent and assist in management (e.g., CHEMTREC®)
3. Inhalation of HF should be considered in any dermal exposure involving the face and neck or if clothing is soaked in product. Can consider the use of nebulized 2.5% calcium gluconate if in protocols or carried.
4. Decontamination is critical for both acid and alkali agents to reduce injury — removal of chemicals with a low pH (acids) is more easily accomplished than chemicals with a high pH (alkalis) because alkalis tend to penetrate and bind to deeper tissues

5. Hydrofluoric acid exposure may manifest as immediate or delayed local and systemic signs, symptoms, and bodily damage

Pertinent Assessment Findings

1. An estimate of the total body surface area that is involved
2. Patient response to therapeutic interventions
3. Patient response to fluid resuscitation
4. Patient response to analgesia
5. ECG changes or new arrhythmia

Quality Improvement

Associated NEMESIS Protocol(s) (eProtocol .01)

- 9914213—Injury - Topical Chemical Burn

Key Documentation Elements

- Burn site
- Body surface area involved
- Identification of the chemical
- Reported or measured pH of the chemical
- Acquisition and transfer of MSDS, chemical container, or other pertinent substance information to the receiving the facility

Performance Measures

- Accurate (over triage/under triage) triage of patients to designated burn centers
- Early recognition of a hydrofluoric acid exposure with appropriate treatment
- Early recognition of hydrofluoric acid burns followed by expeditious initiation of treatment with calcium gluconate and/or calcium chloride and appropriate analgesia
- Measures taken to prevent further contamination

References

1. American Heart Association. Advanced Pediatric Life Support. Jones & Bartlett Learning LLC; 2013
2. Ferng M, Gupta R, Bryant SM. Hazardous Brick Cleaning. J Emergency Medicine. 2009;37(3):305–7
3. Hoffman RS, Howland MA, Lewin NA, Nelson LS, Goldfrank LR. Goldfrank's Toxicologic Emergencies, 10th Edition. China: McGraw-Hill Education;2015
4. Marx JA et al. Rosen's Emergency Medicine: Concepts and Clinical Practice, 2010 769–770
5. O'Sullivan SB, Schmitz TJ. Physical Rehabilitation, 5th Edition. F.A. Davis; 2007: 1098
6. Recommended Medical Treatment for hydrofluoric Acid Exposure. Morristown, NJ: Honeywell Performance Materials and Technologies; October 2012
7. Tintinalli JE, ed. Tintinalli's Emergency Medicine, 9th Edition. McGraw-Hill Education; 2021:35–40, 1391–96

Hydrogen Sulfide Gas

Introduction

Toxin Name Aliases

Hydrosulfuric acid

Sewer gas

Stink Damp

Sour Gas

Egg Gas

Patient Care Goals

1. Attempt rescue only if wearing appropriate respiratory protection (i.e., self-contained breathing apparatus or supplied air respirator)
2. Immediate removal of the victim to a fresh-air environment
3. High-flow oxygen as soon as possible
4. Evaluate for traumatic injuries from falls
5. ACLS Protocols

Patient Presentation

Inclusion Criteria

1. Patients who are found unconscious in an enclosed space, especially if bystanders note the smell of rotten eggs.
2. Patients with altered mental status, upper airway irritation, respiratory distress, or signs of cardiogenic shock following a suspected toxic inhalation. Patients can also exhibit minor effects such as headache, nausea, vomiting, chest pain, shortness of breath, weakness, and dizziness that could manifest into more severe symptoms.

Exclusion Criteria

1. None

Patient Management

Assessment

1. Remove the patient from the environment and move to fresh air.
2. Assess ABCs.
3. In cases of cardiac arrest, history may be limited and efforts should concentrate on the treatment of cardiac arrest.
4. Obtain vital signs including blood pressure, heart rate, pulse oximetry, respiratory rate, blood glucose.
5. Perform 12 lead ECG.
6. Obtain information from bystanders as to type of environment found, last seen normal, suspected time in toxic environment.
7. Obtain medical history including medications and allergies.
8. Perform a physical exam, looking for other injuries that may have occurred due to fall.

9. Discuss presentation and management recommendations with Poison Center by calling 800-222-1222 (will connect to local Poison Control Center).

Treatments and Interventions

1. Remove the patient from the environment and into fresh air is critical.
2. High-flow oxygen as soon as possible
3. The gas can cause eye or skin irritation, rinsing thoroughly with water is recommended. Eye exposure should be treated with sterile water or saline irrigation.
4. Apply ACLS Protocols.
5. If necessary, intubate to provide PEEP for potential ARDS.
6. Consider transport to hyperbaric capable facility but **DO NOT** divert or delay definitive care in order to reach a hyperbaric capable facility.
7. If available, consider treatment with sodium nitrite (3% NaNO₂)
 - a. Adult dose: 10 mL IV (300 mg) over 2-4 min
 - b. Pediatric dose: For children less than 25kg and assuming a hemoglobin level of 7g/dL (if hemoglobin unknown), administer 0.19 mL/kg IV
 - c. If nitrites are given pre-hospital, methemoglobin levels should be checked once the patient arrives in the ED. Patients with anemia will form more methemoglobin (as a percentage of total hemoglobin) than persons with normal red blood cell (RBC) volumes.
 - d. This treatment is not approved by the FDA and is based on animal studies. It could be considered in severe cases and if the antidote is available.
8. If available, consider treatment with hydroxocobalamin
 - a. Adult dose: 5g IV over 15 minutes
 - b. Pediatric dose: 70 mg/kg IV (maximum 5g) over 15 minutes
 - c. This treatment is not approved by the FDA and is based on animal studies. It could be considered in severe cases and if the antidote is available.
9. Crystalloids and vasopressors for hypotension.
10. Benzodiazepines for seizures.

Patient Safety Considerations

1. High concentrations of hydrogen sulfide have been known to cause injury to rescuers. In scenes where toxicity is suspected, only enter the scene with adequate respiratory protection.
2. The primary treatment is optimum supportive care. Do not delay transport for antidotes or hyperbaric oxygen at the expense of supportive care.

Notes/Education Pearls

Key Considerations

1. Hydrogen sulfide can cause olfactory nerve paralysis, leading to the false conclusion that the noxious smell is dissipating.
2. Inhaled agents are becoming more popular to commit suicide. Be aware of signage around a home where the patient may have left a warning to rescuers.

3. Hydrogen Sulfide is produced by bacterial decomposition and many industrial activities. It should be suspected in the decay of fish, sewage, and manure. It is also produced by pulp paper mills, leather industry, roofing asphalt tanks, vulcanizing of rubber, rayon production, and coke manufacturing from coal.

Pertinent Assessment Findings

1. The only findings that may suggest hydrogen sulfide are the smell of rotten eggs from clothing, blood, exhaled air, or gastric secretions.
2. Darkening of jewelry or silver coins near the victim could also heighten suspicion of hydrogen sulfide exposure, however, the absence of this finding should not exclude the presence of hydrogen sulfide.
3. There are no methods available to rapidly and accurately detect hydrogen sulfide exposure, so treatment may be based on history alone.

Quality Improvement

Associated NEMSIS Protocol(s) (eProtocol .01)

9914043—Exposure - Cyanide

9914033—Exposure - Airway/Inhalation Irritants

Key Documentation Elements

- Document elements of history that led to the suspicion of hydrogen sulfide
 - location
 - duration of exposure
 - last seen normal
 - type of industry
 - suspected suicide
- Document response to supportive care
 - ROSC vs continued cardiac arrest
 - improved mental status
 - improved cardiac output
 - improved respiratory status

Performance Measures

- Survival rates
- Clinical improvement in patient
- Long term sequelae
- Rescuer injury while managing dangerous scenes

References

1. Holstege CP, Kirk MA. Cyanide and Hydrogen Sulfide. In: Nelson LS, Howland M, Lewin NA, Smith SW, Goldfrank LR, Hoffman RS. eds. *Goldfrank's Toxicologic Emergencies*, 11e. McGraw-Hill Education; 2019. Accessed October 14, 2024. <https://accessemergencymedicine-mhmedical-com.proxy.library.emory.edu/content.aspx?bookid=2569§ionid=210264536>
2. National Center for Biotechnology Information. PubChem Compound Summary for CID 402, Hydrogen Sulfide. <https://pubchem.ncbi.nlm.nih.gov/compound/Hydrogen-Sulfide>. Accessed Oct. 14, 2024.
3. Beauchamp RO Jr, Bus JS, Popp JA, Boreiko CJ, Andjelkovich DA. A critical review of the literature on hydrogen sulfide toxicity. *Crit Rev Toxicol*. 1984;13(1):25-97. doi:10.3109/10408448409029321
4. Parra, O., Monso, E., Gallego, M., & Morera, J. (1991). "Inhalation of hydrogen sulfide: A case of subacute manifestations and long term sequelae." *British Journal of Industrial Medicine*, 48(4), 286-287 doi: [10.1136/oem.48.4.286](https://doi.org/10.1136/oem.48.4.286)

Irritants

Introduction

Common Irritant Gases

Ammonia
Chlorine
Chloramine
Phosgene
Sulfur dioxide

Patient Care Goals

Rapid recognition of the signs and symptoms of confirmed or suspected airway and respiratory system irritants.

Patient Presentation

Inclusion Criteria

1. Inhalation of a variety of gases, mist, fumes, aerosols, or dust that may cause irritation or injury to the upper and lower respiratory tract. They can also cause asphyxiation and/or other systemic effects.
2. Inhaled airway/respiratory irritant agents will interact with the mucous membranes, upper and lower airways based on solubility, concentration, particle size, and duration of exposure.
3. For irritant gas exposures, water solubility is a crucial factor in determining the location of the toxic effects. High water-soluble gases affect the moist mucous membranes of the upper airway while less water-soluble gases travel deeper into the lungs and affect the alveoli in the lower respiratory tract. The onset of symptoms caused by alveoli damage can be delayed. For aerosols and dust, the particle size of the substance is a crucial factor in determining the location of the toxic effects. Particles < 5 microns can travel deeper into the lower respiratory tract and like less water-soluble gases, cause a delayed clinical effect.

Signs and Symptoms

1. As the type, severity and rapidity of signs and symptom onset depends on agent, water solubility, concentration, particle size, and duration of exposure, the below signs and symptoms are often overlapping and escalating in severity
2. Many airway and respiratory irritant agents have "warning properties" such as identifiable or unpleasant smells or irritation to eyes or airways. Some agents do not have clear warning properties and can have delayed onset of signs and symptoms.

3. Signs and Symptoms of Respiratory Irritant Exposure can include:
 - a. Unusual odor/smell
 - b. Tearing or itchy eyes
 - c. Burning sensation and burns to the nose, pharynx, and respiratory tract
 - d. Sneezing
 - e. General excitation
 - f. Cough
 - g. Chest tightness
 - h. Nausea
 - i. Shortness of breath/dyspnea
 - j. Wheezing
 - k. Stridor
 - l. Dyspnea on exertion
 - m. Dizziness Upper
 - n. Change in voice
 - o. Airway obstructions include laryngospasm and laryngeal edema
 - p. Pulmonary edema (non-cardiogenic)
 - q. Seizures
 - r. Cardiopulmonary arrest

4. High water solubility/highly irritating chemicals:
 - a. Acrolein
 - b. Ammonia
 - c. Chloramine
 - d. Ethylene oxide
 - e. Formaldehyde
 - f. Hydrogen chloride
 - g. Sodium azide
 - h. Sulfur dioxideIntermediate water solubility
 - a. Chlorine
 - b. Methyl bromide

5. Low water solubility/less irritating
 - a. Cadmium fume
 - b. Fluorine
 - c. Hydrogen sulfide (rotten egg odor; olfactory fatigue)
 - d. Mercury fume
 - e. Mustard gas (also delayed blistering skin manifestations)
 - f. Nickel carbonyl
 - g. Nitrogen Dioxide
 - h. Ozone
 - i. Phosgene

6. Asphyxia agents (two categories)

Simple Asphyxiants *Any gas that reduces oxygen fraction or displaces oxygen from the inspired air*

- i. Argon
 - ii. Carbon dioxide
 - iii. Ethane
 - iv. Helium
 - v. Methane
 - vi. Natural gas (e.g., heptane, propane)
 - vii. Nitrogen
 - viii. Nitrogen dioxide (delayed symptom onset)
- a. Systemic chemical asphyxiants: Chemical that interfere with oxygen delivery or utilization
- i. Carbon monoxide gas [See Carbon Monoxide/Smoke Exposure Guideline]
 - ii. Hydrogen cyanide gas [See Cyanide Exposure Guideline]
 - iii. Hydrogen sulfide gas
 - iv. Sodium Azide

7. Inhalants of abuse

- a. These agents or substances are a diverse class of substances that include volatile solvents, aerosols, and gases
- b. These chemicals are intentionally inhaled to produce a state that resembles alcohol intoxication with initial excitation, drowsiness, lightheadedness, and agitation
- c. Users of these inhaled agents are often called huffers, sniffers, baggers, or snorters
- i. These individuals often present after inhaling an aerosol or gas with a loss of consciousness and the presence of the aerosol can or residue/paint around or in the mouth, nose, and oral pharynx
- d. Common household products that are used as inhalants of abuse
- i. Volatile solvents
 - 1. Paint remover
 - 2. Degreasers
 - 3. Dry-cleaning fluids
 - 4. Gasoline
 - 5. Lighter fluid
 - 6. Correction fluid
 - 7. Felt tip markers
 - 8. Glue
 - ii. Cosmetic/paint spray
 - 1. Deodorant spray
 - 2. Vegetable oil spray
 - 3. Fabric protector spray
 - 4. Spray paint

iii. Propellants/asphyxiants/nitrous oxide

1. Propane gas
2. Balloon tanks (helium)
3. Computer keyboard cleaner
4. Ether
5. Halothane
6. Chloroform
7. Butane
8. Propane
9. Whipped cream dispensers

8. Riot Control Agents [See Riot Control Agent Guideline]

A prototype agent is identified with each region of the affected airway respiratory tract for mild to moderate exposures, as severe concentrated exposures of many of these agents overlap in signs and symptoms — the deeper the symptoms are in the respiratory tract and the slower the rate of symptom onset, the less water soluble the airway respiratory irritant. Common examples are listed below:

- a. Nasal and oral pharynx irritation: highly water-soluble agents (ammonia)
- b. Bronchial irritation (chlorine)
- c. Acute pulmonary edema/deep alveolar injury: poorly water soluble (phosgene)
- d. Direct neurotoxin (hydrogen sulfide)
- e. Asphyxia agent with additional symptoms (nitrogen dioxide — Silo Filler's disease)
- f. Inhalants of abuse (volatile solvents, cosmetics/paints, propellants/asphyxiants/nitrous oxide)
- g. Riot control agents [See Riot Control Agent Guideline]
- h. Anticholinesterase inhibitors [See Organophosphate/Nerve Agents Guideline]

1. Ammonia

- a. Immediate detection of unique sharp smell
- b. Nasal pharyngeal burning/irritation sensation
- c. Ocular tearing and irritation
- d. Sneezing
- e. Altered mental status — sleepy to agitated
- f. Cough
- g. Shortness of breath
- h. Chest tightness
- i. Bronchospasm and wheezing
- j. Change in voice
- k. Upper airway obstruction includes laryngospasm and laryngeal edema
- l. Corneal burns or ulcers
- m. Skin burns

- n. Pharyngeal, tracheal, bronchial burns
- o. Dyspnea/tachypnea
- p. High concentrations and or protracted exposure may develop non-cardiac pulmonary edema
- q. Esophageal burns
- 2. Chlorine
 - a. All the above like ammonia)
 - b. Increased likelihood of the following
 - i. Bronchiole burns
 - ii. Bronchospasm wheezing
 - iii. Non-cardiac pulmonary edema develops within 6–24 hours of higher exposures
- 3. Phosgene
 - a. Often have none of the above symptoms for first half hour to several hours then are much milder until more severe lower respiratory tract symptoms develop
 - i. Only warning is report of "fresh mowed hay" odor
 - ii. Mild airway irritation or drying
 - iii. Mild eye irritation
 - iv. Fatigue
 - v. Chest tightness
 - vi. Dyspnea/tachypnea
 - vii. Significant delay up to 24 hours for:
 - a. Exertional dyspnea
 - b. Bronchospasm and wheezing
 - c. Hypoxia
 - d. Severe non-cardiac pulmonary edema
 - e. Cardiopulmonary arrest
- 4. Hydrogen sulfide — A direct neurotoxin and is rapidly absorbed through lung generating systemic effects
 - a. Distinctive rotten egg smell which rapidly causes olfactory fatigue/loss of sense of smell
 - b. Skin and Conjunctival Irritation
 - c. Cough
 - d. Shortness of breath
 - e. Rapid alternations in cognition or consciousness
 - f. Bronchiole and lung hemorrhage/hemoptysis
 - g. Non-cardiac pulmonary edema
 - h. Hydrogen sulfide is known as the "knock down" gas because of near immediate and sudden loss of consciousness with high concentrations
 - i. Asphyxia
 - j. Death
- 5. Nitrogen dioxide (also called Silo Filler's disease)
 - a. Heavier than air displacing oxygen from low lying areas and closed spaces causing direct asphyxia

- b. Low concentrations may cause
 - i. Ocular irritation
 - ii. Cough
 - iii. Dyspnea/tachypnea
 - iv. Fatigue
- c. High concentrations:
 - i. Altered mental status including agitation
 - ii. Cyanosis
 - iii. Vomiting
 - iv. Dizziness
 - v. Loss of consciousness
 - vi. Cardiopulmonary arrest
- 6. Inhalants of abuse (i.e., felt tip markers, spray paint)
 - a. Physical presences of paint or residue on individual from the inhaled agent
 - b. Slurred speech
 - c. Altered mental status (excitation, drowsiness to unconsciousness)
 - d. Loss of consciousness
 - e. Cardiac dysrhythmias
 - f. Cardiopulmonary arrest

Patient Management

1. Don appropriate PPE — respiratory protection critical such as an air purifying respirator, appropriate eye protection and coverall if available.
2. Remove patient from the toxic environment
 - a. Remove the patient's clothing that may retain gases or decontaminate if liquid or solid contamination
 - b. Flush/ irrigate affected/areas where patient is complaining of symptoms of irritation and burning. This includes the skin and mucus membranes with attention to the eyes.
3. Rapidly assess the patient's respiratory status, mental status, and oxygenation
4. Administer oxygen (humidified, if available),
5. Establish intravenous access (if possible)
6. Apply a cardiac monitor (if available)
7. Continuous and ongoing patient reassessment is critical

Assessment

1. Make sure the scene is safe, as many gases are heavier than air and will build up in low lying areas. This is especially true of hydrogen sulfide and its "knock down" effect of the initial unprotected responder and subsequent casualties associated with unprotected rescuers attempting to save the first downed responder
2. Don appropriate Body Substance Isolation (BSI) or appropriate PPE such as an air-purifying respirator (if available) and body coverings including eye protection.
3. Remove patient from toxic environment

4. Decontaminate with water
5. Assess ABCDs and if indicated, expose the patient, and then cover the patient to assure retention of body heat
6. Obtain vital signs (pulse, blood pressure, respiratory rate, neurologic status assessment) which include temperature
7. Place patient on cardiac monitor and examine rhythm strip for arrhythmia potentials (consider 12-lead EKG)
8. Check blood glucose level
9. Monitor pulse oximetry and EtCO₂ for respiratory decompensation
10. Perform carboxyhemoglobin and cyanide device assessment, if available
11. Identify specific suspected agent if possible
12. Pertinent cardiovascular history or other prescribed medications for underlying disease
13. Patient pertinent history
14. Patient physical examination

Treatment and Interventions

1. Secure the airway
2. Administer oxygen, and if hypoventilation is present, toxic inhalation, or desaturation noted, support breathing
 - a. Maintain the airway and assess for airway burns, stridor, or airway edema and if indicated, perform intubation early (recommendation to avoid supraglottic airways— cricothyrotomy may be required in rare severe cases)
 - b. Non-invasive ventilation techniques :
Use continuous CPAP, BiPAP, intermittent positive pressure breathing (IPPB), HFNC, and/or bilevel nasal CPAP for severe respiratory distress or impending respiratory failure Use bag-valve-mask (BVM) ventilation in the setting of hypoventilation, respiratory failure, or arrest
3. While albuterol 2.5 mg nebulized is usually sufficient for mild wheezing without clinical distress, albuterol 5 mg nebulized (or 6 puffs metered dose inhaler) should be administered to all patients in respiratory distress with signs of bronchospasm as allowed by the EMS clinician's scope of practice. This medication should be repeated at this dose with unlimited frequency for ongoing distress
4. If there is evidence of bronchospasm, ipratropium 0.5 mg nebulized is recommended to be given up to 3 doses, in conjunction with albuterol.
5. Initiate IV access for infusion of lactated Ringer's or normal saline and obtain blood samples in effort to record pre-treatment levels, e.g., via point-of-care testing, associated with EMS management (e.g., glucose, lactate, cyanide)
6. Fluid bolus 1 liter (or 20 mL/kg for pediatric patients) if there is evidence of hypoperfusion
7. If the patient is experiencing significant pain, administer IV/IO analgesics per your agency's pain management guidelines. Please refer to your agency's maximum dose threshold.
8. Morphine sulfate 0.1 mg/kg IV or IO

9. Fentanyl 1 mcg/kg IV or IO
10. Eye irrigation early
11. Treat topical chemical burns [See appropriate Toxins and Environmental Section guideline(s)]
12. In severe respiratory irritation, in particular hydrogen sulfide, if there is altered mental status and no improvement with removal from the toxic environment, administer oxygen (humidified if available) as appropriate with a target of achieving 94–98% saturation. Consider a consultation for transfer to a tertiary care hospital and call the local Poison Center. If carbon monoxide is confirmed or suspected, administer high dose oxygen and consider transport to a facility with hyperbaric oxygen capabilities per local guidelines.

Medication Administration

1. If wheezing is present, consider administering inhaled albuterol (2.5–5 mg) as nebulized, or four to eight puffs metered dose inhaler
2. ipratropium 0.5 mg nebulized should be given in conjunction with albuterol, up to three doses

Patient Safety Considerations

1. Generally, speaking to patients with exposure to highly soluble airway/respiratory irritants you will find that they have self-extricated due to the warning properties such as the smell, rapidity of onset of irritation, and other symptoms
2. The less soluble agents may generate only an odor (e.g., mowed hay smell for Phosgene) and will have delayed serious symptoms such as acute pulmonary edema, hypoxia, and shortness of breath with minimal exertion

Notes/Educational Pearls

Key Considerations

1. Airway respiratory irritants can exacerbate underlying reactive airway diseases (e.g., asthma, chronic obstructive pulmonary disease (COPD)) and precipitate or exacerbate bronchospasm, respiratory distress, and hypoxia
2. Patients may be off gassing (particularly hydrogen sulfide and hydrogen cyanide) in the back of the transport vehicle. It is therefore important to have adequate ventilation of the patient compartment and for the EMS clinicians to do appropriate PPE.
3. Removal from the toxic environment, oxygen (humidified if available), general supportive therapy, bronchodilators, respiratory support, and rapid transport are core elements of care as there are no specific antidotes for any of these inhaled agents except for heavy metals that may be chelated in-hospital after agent identification
4. Hydrogen sulfide causes the cells responsible for the sense of smell to be stunned into inaction and therefore with a short exposure will shut down and the exposed victim will not perceive the smell, yet the victim continues to absorb the gas as it is still present

5. Inhaled agents have become popular as a means of committing suicide. If there is some form of suicide signage, hoses, or buckets of substances visible as you arrive at the vehicle or residence, immediately retreat to well-ventilated area and don self-contained breathing apparatus (SCBA) before opening the vehicle or making entry as these gases may be highly concentrated and potentially lethal to EMS responders
6. Household bathroom, kitchen, and oven cleaners when mixed can generate various airway respiratory irritants (ammonia, chloramine, and chlorine gas releases are particularly common). A quite common exposure is to chloramine, a gas liberated when bleach (hypochlorite) and ammonia are combined. Chloramine then hydrolyzes in the distal airways and alveoli to ammonia and hypochlorous acid
7. Sudden sniffing death can result from an individual use of inhalant of abuse
 - a. The cause of death can be from respiratory depression and asphyxia. In addition, some inhalants can cause cardiac arrest due to dysrhythmias from myocardial sensitization. Halogenated hydrocarbons are believed to have the highest risk of causing myocardial sensitization.

Pertinent Assessment Findings

1. Patient may describe a specific odor (chlorine swimming pool smell, ammonia smell, fresh mowed hay smell [phosgene]) which may be helpful but should not be relied upon as the human nose is a poor discriminator of scent
2. Respiratory distress (retractions, wheezing, stridor)
3. Decreased oxygen saturation
4. Skin color
5. Neurologic status assessment
6. Reduction in work of breathing after treatment
7. Improved oxygenation after breathing

Quality Improvement

Associated NEMIS Protocol(s) (eProtocol.01) (for additional information, go to www.nemsis.org)

- 9914033—Exposure - Airway/Inhalation Irritants
- 9914139—Medical - Respiratory Distress/Asthma/COPD/Reactive Airway

Key Documentation Elements

- Document key aspects of the exam to assess for a change after each intervention:
 - Respiratory rate
 - Oxygen saturation
 - Use of accessory muscles or tracheal tugging
 - Breath sounds
 - Air entry/stridor
 - Mental status
 - Color
 - Reduction of burning sensation in airway/pharynx

Performance Measures

- Clinical improvement in patient and response to therapy
- Survival rates of victims
- Long term sequelae of the victims
- No EMS clinicians injured while managing these incidents

References

1. Ainslie G. Inhalational injuries produced by smoke and nitrogen dioxide. *Respir Med.* 1993; 87:169–74
2. Arwood R, Hammond J, Ward GG. Ammonia inhalation. *J Trauma.* 1985; 25:444–7
3. Baydala L, Canadian Pediatric Society, First Nations, Inuit and Métis Health Committee. Inhalant Abuse. *Paediatr Child Health.* 2010;15(7):443–8
4. Chenuel B, Sonobe T, Haouzi P. Effects of infusion of human methemoglobin solution following hydrogen sulfide poisoning. *Clin Toxicol (Phila).* 2015;53(2):93–101
5. Chlorine Toxicity. Emedicine.medscape.com. <http://www.emedicine.com/emerg/topic851.htm> Updated Dec 11, 2015. Accessed March 11, 2022
6. D’Alessandro A, Kuschner W, Wong H, et al. Exaggerated responses to chlorine inhalation among persons with nonspecific airway hyperreactivity. *Chest.* 1996; 109:331–7
7. Douglas WW, Hepper NGG, Colby TV. Silo-filler’s disease. *Mayo Clin Proc.* 1989; 64:291–304
8. Fuller DC, Suruda AJ. Occupationally related hydrogen sulfide deaths in the United States from 1984 to 1994. *J Occup Environ Med.* 2000;42(9):939–42
9. Gorguner M, Akgun M. Acute Inhalation Injury. *Eurasian J Med.* 2010;42(1):28–35
10. Guloglu C, Kara IH, Erten PG. Acute accidental exposure to chlorine gas in the Southeast of Türkiye: a study of 106 cases. *Environ Res.* 2002; 88:89–93
11. Haouzi P, Chenuel B, Sonobe T. High-dose hydroxocobalamin administered after H₂S exposure counteracts sulfide poisoning induced cardiac depression in sheep. *Clin Toxicol (Phila).* 2015 Jan;51(1): 28–36
12. Hydrogen Sulfide Toxicity. Emedicine.medcape.com. <http://www.emedicine.com/emerg/topic258.htm> Updated December 29, 2016. Accessed March 11, 2022
13. Issley S, Lang E. Ammonia Toxicity. Emedicine.medscape.com. <http://www.emedicine.com/emerg/topic846.htm> Updated December 29, 2015. Accessed March 11, 2022
14. Leduc D, Gris G, Lheureux P, et al. Acute and long-term respiratory damage following inhalation of ammonia. *Thorax.* 1992; 47:755–7
15. Lim SC, Yang JY, Jang AS, et al. Acute lung injury after phosgene inhalation. *Korean J Intern Med.* 1996; 11:87–92
16. Mowry JB, Spyker DA, Brooks DE, Zimmerman A, Schauben JL. 2015 Annual Report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 33rd Annual Report. *Clin Toxicol (Phila).* 2016;(10):924–1109

17. Newman LS, Gottschall EB. Toxic Inhalational Lung Injury. In: Albert RK, Spiro SG, Jett JR, ed. Clinical Respiratory Medicine. 2nd Edition. Philadelphia, PA: Mosby; 2004:759–64
18. Noltkamper D, Burgher SW. Toxicity Phosgene 2006. Available at: <https://www.ncbi.nlm.nih.gov/books/NBK537213/>. Accessed March 11, 2022
19. Phosgene Toxicity. Emedicine.medscape.com. <https://www.ncbi.nlm.nih.gov/books/NBK537213/>. Accessed March 11, 2022
20. Reiffenstein RJ, Hulbert WC, Roth SH. Toxicology of hydrogen sulfide. Ann Rev Pharmacol Toxicol. 1992; 32:109–34
21. Sams RN, Carver HW 2nd, Catanese C, Gilson T. Suicide with hydrogen sulfide. Am J Forensic Med Pathol. 2013;34(2):81–2
22. Truscott A. Suicide fad threatens neighbors, rescuers. CMAJ. 2008 Aug 12;179(4):312–3
23. Weinberger B, Laskin DL, Heck DE, et al. The toxicology of inhaled nitric oxide. Toxicol Sci. 2001; 59:5–16

Opioid and Fentanyl Analogs

Introduction

Toxin Name Aliases

- Buprenorphine
- Carfentanil
- Dilaudid®
- Drug abuse
- Fentanyl
- Heroin
- Hydrocodone
- Hydromorphone
- Methadone
- Morphine
- Nitazenes
- Opiate
- Opioid
- Overdose
- Oxycodone
- Oxycontin®
- Percocet®
- Percodan®
- Suboxone
- Subutex
- U-47700
- Vicodin®
- *Note: opioids may be contaminated with additional substances that may or may not be known to the user. Nitazenes, for example, can be used alone or found with fentanyl analogs and further increase the potency of the product. Xylazine has also been found as a common contaminant that may cause further central nervous system depression and autonomic instability in which the patient may not respond to typical antidote therapy.

Patient Care Goals

- Rapid recognition and intervention of a clinically significant opioid poisoning or overdose
- Prevention of respiratory and/or cardiac arrest

Patient Presentation

Symptoms may range from general intoxication (incoordination, dizziness, drowsiness, mental confusion, sedation) to coma. Patients experience miosis and the pupils are

pinpoint in most, but not all cases. The respiratory rate is depressed and in severe poisoning, patients will experience apnea. Complications of severe poisoning/overdose include hypotension, bradycardia, hypothermia, cyanosis or mottled skin, pulmonary aspiration and/or pulmonary edema.

Patient Management

Poison Center Guidance

Pediatric cases:

Refer to the hospital for all unintentional ingestions. Refer to the hospital for all supratherapeutic ingestions.

Adult cases:

- All self-harm cases should be referred to the hospital.
- Accidental overdose cases need to be evaluated individually
- Factors such as patient tolerance need to be considered

Delayed symptoms may occur with polysubstance ingestions or large ingestions.

IF NO SYMPTOMS OCCUR

- For self-harm cases, monitor patients for 12-24 hours
- For an accidental overdose, observe patients for 4-6 hours
- For children, always monitor for 12-24 hours

IF SYMPTOMS OCCUR:

If symptoms occur, the patient will need to be observed until symptoms are resolved.

Suboxone (buprenorphine) ceiling effect:

- It has both opioid agonist and antagonist effects.
- In most cases, its opioid respiratory depression effects plateau or stop at high doses (ceiling effect). This limits the maximal analgesic effect and respiratory depression.
- Respiratory depression can still occur in adults if this drug (without the naloxone component Suboxone) is used intravenously.
- The ceiling effect does not apply to children. They are still at risk of respiratory depression.

Recommended Pre-Hospital Interventions

PPE:

- Although fentanyl and other opioid analogs are potent opioid receptor agonists, the risk of developing clinical symptoms from exposure is extremely low.
- Incidental dermal absorption from incidental contact with an opioid in the field is unlikely to cause toxicity.
- Nitrile gloves are sufficient to provide barrier protection for routine handling of opioids in the field (including fentanyl and analogs).
- In atypical and rare cases where they may be drug particles/droplets suspended in the air; an N-95 respirator should provide adequate protection from inhalation.
- Naloxone should only be administered to exposed first responders for objective signs of hypoventilation and CNS depression and not for vague symptoms of anxiety such as dizziness and tachycardia.

Patient Management:

- Assess the patient for symptoms of opioid intoxication (see clinical manifestations above).
- Activated charcoal should be avoided due to the risk for aspiration.
- Assess the airway and provide naloxone as indicated.
 - Provide adequate ventilation to reduce hypercapnia.
 - Naloxone should be provided to restore appropriate respiration to prevent intubation and should be administered for CNS depression only.
 - Administer Naloxone:
 - Adults: 0.1mg IV/SC/IM, titrated in rapid boluses to restore respiration. It is not necessary to reverse CNS depression if the patient is breathing at an adequate rate.
 - Pediatrics: The pediatric dose is essentially the same as adults, 0.1 mg/kg up to 2 mg adult dose, although there is no harm in giving higher doses if necessary.
 - Intranasal administration: This route is not ideal due to the difficulty in titrating doses, the delayed response due to more limited absorption, and the decreased ventilatory rate. However, this is a common acceptable strategy pre-hospital. Administer a 4 mg into the nostril. A second 4 mg dose may be given in the opposite nostril if there is no response after 2-3 minutes.
 - Intranasal nalmephe is approved for use in opioid overdose. It has a longer duration of action than naloxone. It is also more potent than naloxone at the opioid mu receptors.
- The American College of Medical Toxicology, the American Academy of Clinical Toxicology and the National Association of EMS [Physicians](#) have published a joint [position statement](#) encouraging the continued use of naloxone as a preferred and first-line agent while additional evidence becomes available to support the utility of nalmephe in an opioid overdose. This is due to the concern for its long duration of action requiring emergency departments to potentially observe patients for longer periods of time after its administration. There is also concern about its potency leading to a greater risk of precipitating opioid withdrawal.
 - If the total dose of naloxone exceeds 8 mg in rapid boluses with no response do not further delay intubation. In some cases, analogs may require higher than typical doses of naloxone or an additional sedative may be on board that is preventing an adequate response.
 - In some Suboxone ODs, the reversal effects of naloxone were not observed until 30 minutes later. (in contrast to other opiates, where its effect is usually obvious within 1-2 minutes)

Organic Phosphorus Compounds

Introduction

Toxin Name Aliases

- Acetylcholinesterase inhibitor
- Carbamate Insecticide
- Nerve Agent
- Organophosphate
- Pesticide
- Weapons of mass destruction (WMD)

Patient Care Goals

1. Rapid recognition of the signs and symptoms of confirmed or suspected acetylcholinesterase inhibitor (AChEI) agents such as carbamates, nerve agents, or organophosphates exposure followed by expeditious and repeated administration of atropine, the primary antidote
2. Carbamates and organophosphates are commonly active agents in commercial insecticides

Patient Presentation

Inclusion Criteria

DUMBELS is a mnemonic used to describe the muscarinic signs and symptoms of acetylcholinesterase inhibitor agent poisoning. All patient age groups are included where the signs and symptoms exhibited are consistent with the toxidrome of DUMBELS

- a. Diarrhea
- b. Urination
- c. Miosis
- d. Bronchospasm/Bronchorrhea/Bradycardia (the killer Bs)
- e. Emesis
- f. Lacrimation
- g. Salivation/Sweating

MTWThF is a mnemonic used to describe the nicotinic signs and symptoms of acetylcholinesterase inhibitor agent poisoning. All patient age groups are included where the signs and symptoms exhibited are consistent with the toxidrome of MTWThF

- h. Mydriasis or miosis
- i. Tachycardia
- j. Weakness
- k. Th for the H: hyperthermia or hypothermia
- l. Fasciculations

Exclusion Criteria

None noted

Patient Management

1. Don the appropriate PPE
2. Remove the patient's clothing and wash the skin with soap and warm water
 - a. Acetylcholinesterase inhibitor agents can be absorbed through the skin
 - b. Contaminated clothing can provide a source of continued exposure to the toxin
3. Rapidly assess the patient's respiratory status and mental status
4. Administer the antidote atropine immediately for confirmed or suspected acetylcholinesterase inhibitor agent exposure when patients are showing signs of muscarinic toxicity summarized in the DUMBELS mnemonic
5. Administer oxygen as appropriate with a target of achieving 94–98% saturation and provide airway management
6. Establish intravenous access (if possible)
7. Apply a cardiac monitor (if available)
8. The heart rate may be normal, bradycardic, or tachycardic
9. Clinical improvement should be based upon the drying of secretions and easing of respiratory effort rather than heart rate or pupillary response
10. Continuous and ongoing patient reassessment is critical

Assessment

1. Acetylcholinesterase inhibitor agents are highly toxic chemical agents and can rapidly be fatal
2. Patients with low-dose chronic exposures may have a more delayed presentation of symptoms
3. Antidotes (atropine and pralidoxime) are effective if administered before circulation fails
4. The patient may develop:
 - a. Miosis (pinpoint pupils)
 - b. Bronchospasm
 - c. Bradycardia
 - d. Vomiting
 - e. Excessive secretions in the form of:
 - i. Tearing
 - ii. Salivation
 - iii. Rhinorrhea
 - iv. Diarrhea
 - v. Urination
 - vi. Bronchorrhea
5. Penetration of an acetylcholinesterase inhibitor agent into the central nervous system (CNS) will cause:
 - a. Headache

- b. Confusion
 - c. Generalized muscle weakness
 - d. Seizures
 - e. Lethargy or unresponsiveness
6. Estimated level of exposure based upon signs and symptoms
- a. Mild
 - i. Miosis alone (while this is a primary sign in vapor exposure, it may not be present in all exposures)
 - ii. Miosis and severe rhinorrhea
 - b. Mild to moderate (in addition to symptoms of mild exposure)
 - i. Localized swelling
 - ii. Muscle fasciculations
 - iii. Nausea and vomiting
 - iv. Weakness
 - v. Shortness of breath
 - c. Severe (in addition to symptoms of mild to moderate exposure)
 - i. Unconsciousness
 - ii. Convulsions
 - iii. Apnea or severe respiratory distress requiring assisted ventilation
 - iv. Flaccid paralysis
7. Onset of symptoms can be immediate with an exposure to a large amount of the acetylcholinesterase inhibitor
- a. There is usually an asymptomatic interval of minutes after dermal or oral exposure before these symptoms occur
 - b. Effects from vapor exposure occur almost immediately
8. Signs and symptoms with large acetylcholinesterase inhibitor agent exposures (regardless of route)
- a. Sudden loss of consciousness
 - b. Seizures
 - c. Copious secretions
 - d. Apnea
 - e. Death
9. Obtain an accurate exposure history (as patient may become unconscious before arrival at the ED):
- a. Time of ingestion or exposure
 - b. Route of exposure
 - c. Quantity of medication or toxin taken (safely collect all possible medications or agents)
 - d. Alcohol or other intoxicant taken
 - e. Pertinent cardiovascular history or other prescribed medications for underlying disease

10. The patient can manifest any of the signs and symptoms of the toxidrome based on the route of exposure, agent involved, and concentration of the agent:
- a. Vapor exposures will have a direct effect on the eyes and pupils causing miosis
 - b. Patients with isolated skin exposures will have normally reactive pupils
 - c. Certain acetylcholinesterase inhibitor agents can place the patient at risk for both a vapor and skin exposure

Treatments and Interventions

1. Medications:

a. Atropine

- i. Atropine is the primary antidote for organophosphate, carbamate, or nerve agent exposures, and repeated doses should be administered liberally to patients who exhibit signs and symptoms of exposure or toxicity
- ii. Atropine may be provided in multi-dose vials, pre-filled syringes, or autoinjectors

b. Pralidoxime chloride (2-PAM)

- i. Pralidoxime chloride is a secondary treatment and should be given concurrently to reactivate acetylcholinesterase
- ii. Pralidoxime chloride may be provided in a single dose vial, pre-filled syringes, or auto-injectors
- iii. Auto-injectors typically contain 600 mg of pralidoxime chloride
- iv. To be beneficial to the victim, a dose of pralidoxime chloride should be administered shortly after the nerve agent or organophosphate poisoning as it has minimal clinical effect if administration is delayed

c. Benzodiazepines

- i. Benzodiazepines are administered as an anticonvulsant for those patients who exhibit seizure activity [See Seizures Guideline for doses and routes of administration]
- ii. Lorazepam, diazepam, and midazolam are the most frequently used benzodiazepines in the prehospital setting; midazolam may have the fastest onset of action
- iii. Benzodiazepines may be provided in multi-dose or single-dose vials, pre-filled syringes, or auto-injectors
- iv. CANA[®] (Convulsive 1. Antidote Nerve Agent) is a commercially available autoinjector that contains 10 mg of diazepam

d. Duodote[®]

- i. A commercially available auto-injector of nerve agent/organophosphate antidote
- ii. Duodote[®] is one auto-injector that contains 2.1 mg of atropine and 600 mg of pralidoxime chloride

e. ATNAA[®] (Antidote Treatment Nerve Agent Auto-injector)

- i. An auto-injector of nerve agent/organophosphate antidote that is typically in military supplies
- ii. ATNAA® is one auto-injector that contains 2.1 mg of atropine and 600 mg of pralidoxime chloride
- iii. ATNAA® may be seen in civilian supplies assets when Duodote® is unavailable or in short supply

f. CHEMPACK

- i. Federal cache of nerve agent antidotes that is managed by the Centers for Disease Control and Prevention (CDC) and offered to states that voluntarily agree to maintain custody and security of CHEMPACK assets
- ii. These are forward deployed at sites determined by states that are part of the program such as hospitals and EMS centers
- iii. Deployment of CHEMPACKs is reserved for events where the nerve agent/organophosphate exposure will deplete the local or regional supply of antidotes
- iv. There are two types of CHEMPACK containers:
 - 1. EMS Containers: CHEMPACK assets for EMS contain a large portion of autoinjectors for rapid administration of antidotes by EMS clinicians of all levels of licensure/certification. They contain enough antidote to treat roughly 454 patients
 - 2. Hospital Containers: CHEMPACK assets contain a large portion of multidose vials and powders for reconstitution — they contain enough antidote to treat roughly 1,000 patients

2. Medication Administration:

- a. Atropine, in large and potentially multiple doses, is the antidote for an acetylcholinesterase inhibitor agent poisoning
- b. Atropine should be administered immediately followed by repeated doses until the patient's secretions resolve
- c. Pralidoxime chloride (2-PAM) is a secondary treatment and, when possible, should be administered concurrently with atropine
- d. The stock of atropine and pralidoxime chloride available to EMS clinicians is usually not sufficient to fully treat the victim of an acetylcholinesterase inhibitor agent exposure; however, EMS clinicians should initiate the administration of atropine and, if available, pralidoxime chloride
- e. Seizures should be treated with benzodiazepines. There is some emerging evidence that, for midazolam, the intranasal route of administration may be preferable to the intramuscular route. However, intramuscular absorption may be more clinically efficacious than the intranasal route in the presence of significant rhinorrhea
- f. The patient should be emergently transported to the closest appropriate medical facility

3. Recommended Doses (See dosing tables) The medication dosing tables that are provided below are based upon the severity of the clinical signs and symptoms exhibited by the patient. There are several imperative factors to note:

- a. For organophosphate or severe acetylcholinesterase inhibitor agent exposure, the required dose of atropine necessary to dry secretions and improve the respiratory status may exceed 20 mg. Atropine should be administered rapidly and repeatedly until the patient's clinical symptoms diminish. Atropine must be given until the acetylcholinesterase inhibitor agent has been metabolized.
- b. Because Duodote® auto-injectors contain pralidoxime chloride, they should not be used for additional dosing of atropine beyond the recommended administered dose of pralidoxime chloride
- c. All the medications below can be administered intravenously in the same doses cited for the intramuscular route. However, due to the rapidity of onset of signs, symptoms, and potential death from acetylcholinesterase inhibitor agents, intramuscular administration is highly recommended to eliminate the inherent delay associated with establishing intravenous access
- d. The antidotes can be administered via the intraosseous route. However, due to the rapidity of onset of signs, symptoms, and potential death from acetylcholinesterase inhibitor agents, intramuscular administration remains the preferable due to the inherent delay associated with establishing intraosseous access and the limited use of this route of administration for other medications

Table 1. Mild Acetylcholinesterase Inhibitor Agent Exposure

Patient	Atropine Dose (Weight) IM or via Auto-injector
Infant: 0–2 years of age	0.05 mg/kg IM or via auto-injector (i.e., 0.25 and/or 0.5 mg auto-injector(s))
Child: 3–7 years of age (13–25 kg)	1 mg IM or via auto-injector (i.e., one 1 mg or two 0.5 mg auto-injectors)
Child: 8–14 years of age (26–50 kg)	2 mg IM or via auto-injector (i.e., one 2 mg or two 1 mg auto-injectors)
Adolescent/Adult	2 mg IM or via auto-injector
Pregnant Women	2 mg IM or via auto-injector
Geriatric/Frail	1 mg IM or via auto-injector
Adapted from: U.S. Department of Health and Human Services, ASPR, National Library of Medicine, Chemical Hazards Emergency Medical Management: Nerve Agents— Prehospital Management, https://www.cdc.gov/TSP/MMG/MMGDetails.aspx?mmgid=523&toxid=93	

Table 2. Mild to Moderate Acetylcholinesterase Inhibitor Agent Exposure

Patient (Weight)	Atropine Dose IM or via Auto-injector	Pralidoxime Chloride Dose IM or via 600 mg Auto-injector
Infant: 0–2 years of age	0.05 mg/kg IM or via auto-injector (i.e., 0.25 mg and/or 0.5 mg auto-injector)	15 mg/kg IM
Child: 3–7 years of age (13–25 kg)	1 mg IM or via auto-injector (i.e., one 1 mg auto-injector or two 0.5 mg auto-injectors)	15 mg/kg IM OR One auto-injector (600 mg)
Child: 8–14 years of age (26–50 kg)	2 mg IM or via auto-injector (i.e., one 2 mg auto-injector or two 1 mg auto-injectors)	15 mg/kg IM OR One auto-injector (600 mg)
Adolescent/ Adult	2–4 mg IM or via auto-injector	600 mg IM OR One auto-injector (600 mg)
Pregnant Women	2–4 mg IM or via auto-injector	600 mg IM OR One auto-injector (600 mg)
Geriatric/Frail	2 mg IM or via auto-injector	10 mg/kg IMOR One auto-injector (600 mg)
<p><i>Adapted from: U.S. Department of Health and Human Services, ASPR, National Library of Medicine, Chemical Hazards Emergency Medical Management: Nerve Agents— Prehospital Management, https://www.cdc.gov/TSP/MMG/MMGDetails.aspx?mmgid=523&toxid=93</i></p>		

Table 3. Severe Acetylcholinesterase Inhibitor Agent Exposure

Patient (Weight)	Atropine Dose IM or via 600 mg Auto-injector	Pralidoxime Chloride Dose IM or via Auto-injector
Infant: 0–2 years of age	0.1 mg/kg IM or via auto-injector (i.e., 0.25 mg and/or 0.5 mg auto-injector)	45 mg/kg IM
Child: 3–7 years of age (13–25 kg)	0.1 mg/kg IM OR 2 mg via auto-injector (i.e., one 2 mg auto-injector or four 0.5 mg auto-injectors)	45 mg/kg IM OR One auto-injector (600 mg)
Child: 8–14 years of age (26–50 kg)	4 mg IM or via auto-injector	45 mg/kg IM OR Two auto-injectors (1200 mg)

	(i.e., <i>two 2 mg auto-injectors or four 1 mg auto-injectors</i>)	
Adolescent: 14 years of age or older	6 mg IM or via auto-injector (i.e., <i>three 2 mg auto-injectors</i>)	Three auto-injectors (1800 mg)
Adult	6 mg IM or via auto-injector (i.e., <i>three 2 mg auto-injectors</i>)	Three auto-injectors (1800 mg)
Pregnant Women	6 mg IM or via auto-injector (i.e., <i>three 2 mg auto-injectors</i>)	Three auto-injectors (1800 mg)
Geriatric/Frail	2–4 mg IM or via auto-injector (i.e., <i>one to two 2 mg auto-injectors</i>)	25 mg/kg IM OR two to three auto-injectors (1200mg–1800 mg)
<p>Adapted from: U.S. Department of Health and Human Services, ASPR, National Library of Medicine, Chemical Hazards Emergency Medical Management: Nerve Agents— Prehospital Management, https://wwwn.cdc.gov/TSP/MMG/MMGDetails.aspx?mmgid=523&toxid=93</p>		

Patient Safety Considerations

1. Continuous and ongoing patient reassessment is critical
2. Clinical response to treatment is demonstrated by the drying of secretion and the easing of respiratory effort
3. Initiation of and ongoing treatment should not be based upon heart rate or pupillary response
4. Precautions for pralidoxime chloride administration:
 - a. Although Duodote® and ATNAA® contains atropine, the primary antidote for an acetylcholinesterase inhibitor agent poisoning, the inclusion of pralidoxime chloride in the auto-injector can present challenges if additional doses of atropine are warranted by the patient condition and other formulations of atropine are unavailable:
 - i. Pediatrics: an overdose of pralidoxime chloride may cause profound neuromuscular weakness and subsequent respiratory depression
 - ii. Adults: Especially for the geriatric victim, excessive doses of pralidoxime chloride may cause severe systolic and diastolic hypertension, neuromuscular weakness, headache, tachycardia, and visual impairment
 - iii. Geriatrics: victim who may have underlying medical conditions, particularly impaired kidney function or hypertension, the EMS clinician

should consider administering the lower recommended adult dose of intravenous pralidoxime chloride

5. Considerations during the use of auto-injectors
 - a. If an auto-injector is administered, a dose calculation prior to administration is not necessary
 - b. For atropine, additional auto-injectors should be administered until secretions diminish
 - c. Mark 1 kits, Duodote® and ATNAA® have not been approved for pediatric use by the Food and Drug Administration (FDA), but they can be considered for the initial treatment for children of any age with severe symptoms of an acetylcholinesterase inhibitor agent poisoning especially if other formulations of atropine are unavailable
 - d. Pediatric Atro-Pen® auto-injectors are commercially available in a 0.25 mg autoinjector (yellow) and a 0.5 mg auto-injector (red). Atro-Pen® auto-injectors are commercially available in a 1 mg auto-injector (blue) and a 2 mg auto-injector (green)
 - e. A pralidoxime chloride 600 mg auto-injector may be administered to an infant that weighs greater than 12 kg

Notes/Education Pearls

Key Considerations

1. Clinical effects of acetylcholinesterase inhibitor agents
 - a. The clinical effects are caused by the inhibition of the enzyme acetylcholinesterase which allows excess acetylcholine to accumulate in the nervous system
 - b. The excess accumulated acetylcholine causes hyperactivity in muscles, glands, and nerves
2. Organophosphates Insecticides
 - a. Can be legally purchased by the public
 - b. Organophosphate pesticides penetrate tissues and bind to the patient's body fat producing a prolonged period of illness and ongoing toxicity even during aggressive treatment
3. Nerve agents
 - a. Traditionally classified as weapons of mass destruction (WMD)
 - b. Not readily accessible to the general public
 - c. Extremely toxic and rapidly fatal with any route of exposure
 - d. GA (tabun), GB (sarin), GD (soman), GF, and VX are types of nerve agents and are WMDs

Pertinent Assessment Findings

The signs and symptoms exhibited with the toxidrome of DUMBELS [See Patient Presentation— Inclusion Criteria]

Quality Improvement

Associated NEMSIS Protocol(s) (eProtocol .01)

9914047—Exposure - Nerve Agents

Key Documentation Elements

- Time to recognize initial signs and symptoms
- Number of repeated doses of atropine required for the secretions diminish and respirations to improve
- Patient reassessments
- Patient responses to therapeutic interventions
- Measures taken to decontaminate the patient
- Measures taken to protect clean environments from contamination

Performance Measures

- Ability of the EMS system to rapidly locate additional and adequate antidote assets
- Ability of the EMS system to rapidly deploy additional and adequate antidote assets
- Survival rates of victims
- Complication rates from the toxin
- Complication rates from the antidotes
- Long-term clinical sequelae of the victims

References

1. Barkin RM, Rosen P, Seidel JS, Caputo GL, Jaffe DM. Pediatric Emergency Medicine: Concepts and Clinical Practice. St Louis, MO: Mosby; 1992:490–1
2. Burillo-Putze G, Nogue Xarau SN. In Tintinalli JE, ed. Tintinalli's Emergency Medicine, 8th Edition. McGraw-Hill Education; 2016:1318–21
3. Eddelston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus poisoning. *Lancet*. 2008;371(9612):597–607
4. Hoffman RS, Howland MA, Lewin NA, Nelson LS, Goldfrank LR. Goldfrank's Toxicologic Emergencies, 10th Edition. China: McGraw-Hill Education; 2015
5. Horowitz BZ, Hendrickson RG. Chemical disasters. In Tintinalli JE, ed. Tintinalli's Emergency Medicine, 8th Edition. McGraw-Hill Education; 2016:44–5
6. Marx JA et al. Rosen's Emergency Medicine: Concepts and Clinical Practice. 2014:825–6, 2057–60, 2476–7
7. Nelson LS. Goldfrank's Toxicologic Emergencies, 10th Edition. China: McGraw-Hill Education; 2015:1450–76
8. Nerve Agents—Prehospital Management. Chemm.nlm.nih.gov.

Phosphide Salts or Gas

Introduction

Toxin Name Aliases

Phosphide Salts, Metal Phosphide, Phosphine, Aluminum Phosphide, Zinc Phosphide, hydrogen phosphide, phosphorated hydrogen, phosphorus hydride, phosphorus trihydride

Patient Care Goals

1. Rapid recognition of the signs and symptoms of confirmed or suspected metal phosphide ingestion or phosphine inhalation and rapid removal of the patient from the source.
2. Maintain perfusion and hemodynamic stability.
3. Transfer to a higher-level care center with ECMO capabilities if available.

Patient Presentation

Inclusion Criteria

1. Intentional or unintentional ingestion of aluminum phosphide or zinc phosphide
2. Phosphine gas exposure
 - a. For greater than 0.3 ppm over 10 hours
 - b. For greater than 1 ppm more than 15 minutes
 - c. For greater than 50 ppm for any period of time
 - d. With any symptoms
3. Clinical manifestations can be broad and nonspecific:
 - a. Gastrointestinal symptoms including nausea, vomiting, diarrhea, and abdominal pains are often the first symptoms of toxicity. Both hypoglycemia and hyperglycemia can occur.
 - b. Dysrhythmias, acute respiratory distress syndrome (ARDS), shock and cardiovascular collapse can occur in rapid succession.
 - c. Electrocardiogram abnormalities are common and broad including sinus tachycardia, atrial flutter and fibrillation, SVT, ventricular tachycardia, ventricular fibrillation, and abnormalities with ST segment changes, QRS widening^[1]
4. Central nervous system manifestations including anxiety, headache, restlessness, ataxia, dizziness, paresthesias, and tremor may also be present, followed by more severe signs of toxicity with encephalopathy, seizures, and coma.

Exclusion Criteria

Absence of known or presumed exposure from phosphide salts and/or phosphine gas.

Patient Management

Assessment

The level of safety gear needed to access patients depends on known or unknown concentrations of phosphine gas—universal precautions, including gloves, goggles, and personal protective equipment is indicated.

Per NIOSH/OSHA^[ii]

1. Up to 3 ppm: APF (Assigned Protection Factor) = 10, any supplied-air respirator
2. Up to 7.5 ppm: APF = 25, any supplied-air respirator operated in a continuous-flow mode
3. Up to 15 ppm: APF = 50, any air-purifying, full facepiece respirator (gas mask) with a chin-style, front- or back-mounted canister providing protection against the compound. Any self-contained breathing apparatus with a full facepiece. Any supplied-air respirator with a full facepiece
4. Up to 50 ppm: APF = 1,000, any supplied-air respirator operated in a pressure-demand or other positive-pressure mode
5. Emergency or planned entry into unknown concentrations or IDLH conditions: APF = 10,000, Any self-contained breathing apparatus that has a full facepiece and is operated in a pressure-demand or other positive-pressure mode. Any supplied-air respirator that has a full facepiece and is operated in a pressure-demand or other positive-pressure breathing apparatus.

Discuss presentation and management recommendations with Poison Center by calling 800-222-1222 (will connect to local Poison Control Center).

Treatments and Interventions

1. Management:
 - a. Immediate removal from the area of exposure
 - b. Removal of clothes and immediate skin/eye decontamination with water
 - c. Manage airway as necessary
 - d. Administer oxygen as appropriate with target of achieving 94-98% saturation
 - e. Initiate monitoring and perform 12-lead EKG
 - f. Establish IV access
 - g. Check blood glucose and treat hypoglycemia per hypoglycemia guideline and hyperglycemia per hyperglycemia guideline
 - h. If hypotensive consider the following interventions:
 - i. 30 ml/kg IV fluid bolus of lactated ringers and/or normal saline
 - ii. Vasopressor medication, (in order of preference,) with goal to titrate to MAP of greater than 65 mm Hg (subject to EMS agency availability and provider scope of practice):
 1. Norepinephrine IV drip 0.02-0.4 mcg/kg/min

2. Phenylephrine IV drip 0.5 to 6 mcg/kg/min
 3. Epinephrine IV drip 0.02-0.2 mcg/kg/min
 4. Dopamine IV 2-10 mcg/kg/min
- If there is no pump available, use a standard drip rate formula to convert the above rates to drip rates per minute (gtt/min).*

iii. For cardiac dysrhythmias, treat according to ACLS guidelines

Patient Safety Considerations

There may be an increased risk for dysrhythmias in use of epinephrine and dopamine over norepinephrine and phenylephrine.^[iii]

Notes/Education Pearls

Key Considerations

1. Phosphide metals are uncommon in the U.S. because they are severely toxic, nonspecific pesticides. They are however available in the U.S. and are dispensed with appropriate permission. They are common in developing countries, particularly in India, Bangladesh, Sri Lanka, and Iran, and may be imported illegally.
2. Oral exposures to metal phosphides are highly toxic, even in small doses.
3. For intentional ingestions, tablet formulation (i.e., aluminum salts) are more toxic than the granule forms (zinc salts) because the granules need to be dissolved in water before ingestion.
4. Clinical effects of metal phosphides and phosphine
 - a. The clinical effects are caused by blocking the electron transport chain and inhibits the production of ATP and cellular respiration, leading to multisystem organ failure
 - b. Reduces glutathione concentration and produces free radicals leading to widespread cellular damage.
 - c. Phosphine gas is corrosive and can lead to direct alveolar damage causing ARDS.
5. Given majority of deaths occur within 12-24 hours from cardiovascular collapse, consider prehospital transport directly to a VA-ECMO capable center for extracorporeal life support when available with limited delay in care.^[iv]
6. There is no clear antidote for the management of metal phosphides and phosphine exposure. Primary goal is removal from source and supportive critical care.

Pertinent Assessment Findings

Smell of garlic or decaying fish on breath may be found on patients with oral or inhalational poisoning of metal phosphides.^[v]

Hypoxia from pulmonary edema, atelectasis, and/or ARDS may develop thereby needing O2 supplementation and positive pressure support.^[vi]

Quality Improvement

Associated NEMSIS Protocol(s) (eProtocol .01)

Key Documentation Elements

1. Time to recognition of phosphide metal or phosphine exposure
2. Protective measures for EMS
3. Form of ingestion/exposure (oral vs inhalation, intentional vs unintentional)
4. Cardiac rhythm/rate
5. Patient reassessments
6. Patient responses to therapeutic interventions
7. Measures taken to decontaminate the patient
8. Measures taken to protect clean environments from contamination

Performance Measures

1. Ability of the EMS system to rapidly locate and transport to ECMO capable center
2. Ability of the EMS system to rapidly escalate in case of mass exposure
3. Survival rates of victims
4. Complication rates from the toxin
5. Long-term clinical sequelae of the victims

References

- [i] Chugh SN, et al. Magnesium levels in acute cardiotoxicity due to aluminium phosphide poisoning. *Indian J Med Res.* 1991;94:437–439.
- [ii] <https://www.cdc.gov/niosh/npg/npgd0505.html>
- [iii] Gurjar M, Baronia AK, Azim A, Sharma K. Managing aluminum phosphide poisonings. *J Emerg Trauma Shock.* 2011 Jul;4(3):378-84. doi: 10.4103/0974-2700.83868. PMID: 21887030; PMCID: PMC3162709.
- [iv] Elabbassi W, et al. Severe reversible myocardial injury associated with aluminium phosphide toxicity: a case report and review of literature. *J Saudi Heart Assoc.* 2014;26:216–221.
- [v] Pepelko B, et al. Worker exposure standard for phosphine gas. *Risk Anal.* 2004;24:1201–1213
- [vi] Moghadamnia AA. An update on toxicology of aluminum phosphide. *DARU.* 2012;20:25

Sedatives

Introduction

Toxin Name Aliases

Barbiturates, benzodiazepines, Z-drugs, muscle relaxers, chloral hydrate, alpha 2 agonists (dexmedetomidine, xylazine, clonidine, guanfacine), baclofen

Patient Care Goals

1. Recognition of a clinically significant sedative-hypnotic exposure
2. Ensure adequate ventilation, oxygenation and hemodynamic support
3. Correct/prevent hypothermia

Patient Presentation

Inclusion Criteria

Patients of all age groups exhibit signs and symptoms consistent with a sedative-hypnotic toxidrome in the setting of suspected or known exposure to a sedative-hypnotic agent. Symptoms include central nervous system and respiratory depression, slurred speech, ataxia, incoordination, hypotension, and hypothermia. It is important to recognize that patients may have been exposed to more than one type of substance (for example, opioids or anticholinergics) and/or also display symptoms from a concomitant or underlying medical condition (i.e. hypoglycemia, stroke, etc).

Exclusion Criteria

Patients with symptoms entirely from other causes (e.g. head injury, opioids, post-ictal phase, or hypoglycemia).

Patient Management

1. Use appropriate PPE.
2. Implement interventions to support the patient's airway, breathing, and circulation.
3. If possible and the patient's condition allows, assess the scene and attempt to identify source of exposure (i.e. specific medication(s) or substance taken), time of ingestion, and quantity ingested. Bring pill bottles or source(s) of exposure with the patient to the treating facility, if they are available and safe to transport.
4. Identify and document relevant medical history and medications prescribed for underlying disease.
5. Always use caution when on scene. If the patient exposed themselves to a sedative-hypnotic via the intravenous route, there is a possibility that unsecured hypodermic needles may be present. This may result in a needle stick injury if the proper precautions are not employed. Additionally, populations using intravenous drugs have an increased incidence of bloodborne pathogens such as hepatitis and/or HIV.

6. Consider administration of naloxone (antidote for opioid overdose) as co-exposure to both a sedative-hypnotic substance AND an opioid medication may be possible. Naloxone will not reverse sedation caused by most sedative-hypnotics.
7. Consider consultation with Poison Center by calling 800-222-1222 (will connect to local Poison Control Center).

Assessment

1. Assess airway, breathing, circulation, and mental status
2. Maintain an open airway and consider oxygen support with a goal of 94-98% and if needed provide ventilation assistance with a bag valve mask
3. Assess vital signs including temperature
4. Evaluate for other potential contributors/causes of the patient's symptoms

Treatments and Interventions

1. Maintain airway support by positioning the patient to reduce aspiration risk, providing supplemental oxygen, assisting with a bag-valve mask, and securing the airway with an endotracheal tube when needed
2. Intravenous access should be obtained with two large-bore intravenous lines when possible.
3. For patients who are altered, obtain a blood glucose level and administer 2 mg of naloxone IV (when co-exposure to opioids is suspected or known) according to agency protocols.
4. Naloxone administration can occur via the IV, IM, or IN routes. Doses can be incrementally titrated until respiratory depression is reversed.
5. Volume resuscitation should be initiated in cases of hemodynamic instability.
6. Initiate re-warming measures to correct hypothermia.

Patient Safety Considerations

Measures to minimize aspiration risk should be implemented (such as patient positioning).

Notes/Education Pearls

Key Considerations

- Death usually results from cardiopulmonary collapse.
- It is not recommended to administer flumazenil in the pre-hospital setting.

Pertinent Assessment Findings

1. The primary clinical indications for the use of sedative-hypnotics are sedation, anxiety, muscle relaxation, analgesia, and seizure management.
2. In the sedative-hypnotic overdose scenario, signs and symptoms include:
 - Decreased mental status
 - Respiratory depression
 - Hypotension
 - Hypothermia
 - Slurred speech
 - Ataxia and incoordination

3. The risk of coma and respiratory arrest (with subsequent cardiac arrest) from a sedative-hypnotic overdose may increase with concomitant use of other medications that also impair the CNS and respiratory drive, such as opioids and ethanol.

Quality Improvement

Associated NEMIS Protocol(s) (eProtocol .01)

None

Key Documentation Elements

- Rapid and accurate identification of signs and symptoms of sedative-hypnotic poisoning
- Airway management
- Pulse oximetry (oxygen saturation) and capnometry or capnography
- Blood glucose assessment
- Hypothermia assessment

Performance Measures

- The performance and ongoing assessment of airway management
- Frequency of patients who develop adverse effects or complications (aspiration pneumonia or pulmonary edema, hypothermia, hypotension)

References

Nelson LS, Howland M, Lewin NA, Smith SW, Goldfrank LR, Hoffman RS. eds. Goldfrank's Toxicologic Emergencies, 11e. McGraw-Hill Education; 2019. Accessed October 22, 2024. <https://accessemergencymedicine.mhmedical.com/content.aspx?bookid=2569§ionid=210256528>

Murphy, C.M. (2021). Principles of toxicology. In Emergency Medical Services (eds D.C. Cone, J.H. Brice, T.R. Delbridge and J.B. Myers). <https://doi.org/10.1002/9781119756279.ch46>

Stimulant Poisoning or Overdose

Introduction

Toxin Name Aliases

- **Amphetamine Salts** (Prescription drugs such as amphetamine, dextroamphetamine, lisdexamphetamine, brand names such as Adderall, Desoxyn, Vyvanse, etc.)
- **Cocaine & Crack Cocaine** (Coke, blow, candy, gravel, etc.)
- **Ketamine** (Kit kat, K, K-hole, Special K, etc.)
- **MDMA** (Ecstasy, Molly, X, etc.)
- **Methamphetamine** (Ice, Meth, Speed, Uppers, etc.)
- **Synthetic Cannabinoids** (Spice, K2, etc.)
- **Synthetic Cathinones** (Bath Salts, Methylone, Methcathinone, alpha-PVP, etc.)
- **Prescription Stimulants** (methylphenidate, brand names such as Concerta, Ritalin; modafinil, brand name Provigil)
- **Phencyclidine** (PCP, angel dust, etc.)
- **Miscellaneous** (Tusi/2C/pink cocaine)
- **Methylxanthines** (Caffeine, theophylline, etc.)

Patient Care Goals

1. Identify intoxicating agent.
2. Protect organs at risk for injury such as heart, brain, liver, kidneys.
3. Determine if there is an antidote.
4. Treat the symptoms, which may include severe tachycardia and hypertension, hyperthermia, diaphoresis, agitation, hallucinations, chest pain, arrhythmia, and seizures.
5. Discuss presentation and management recommendations with Poison Center by calling 800-222-1222 (will connect to local Poison Control Center).

Patient Presentation

Inclusion Criteria

1. Tachycardia/tachydysrhythmias
2. Hypertension
3. Diaphoresis
4. Delusions/paranoia
5. Agitation
6. Seizures
7. Hyperthermia
8. Mydriasis (dilated pupils)
9. Exposure (e.g. ingestion, inhalation) to stimulant/hallucinogenic (with stimulant properties) agents:

- a. Amphetamine/methamphetamine
- b. Cocaine & Crack Cocaine
- c. Ketamine
- d. MDMA (“Ecstasy”)
- e. Synthetic Cannabinoids (“K2 or spice”)
- f. Synthetic Cathinones (“bath salts”)
- g. Prescription Stimulants (Adderall, Concerta, Modafinil)
- h. Phencyclidine (PCP)
- i. Misc. (2C/Tusi)

See more comprehensive list of examples above

Exclusion Criteria

None noted

Patient Management

Assessment

1. Begin with the **ABCDs**:
 - a. **A**irway is patent
 - b. **B**reathing is present and patient is ventilating well
 - c. **C**irculation is intact and patient is perfused well
 - d. **D**isability/neuro/mental status
 - e. Treat any compromise of these parameters
 - f. Ask about chest pain, difficulty breathing, any agitation or seizures.
2. Vital signs including temperature, if possible, when hyperthermia is considered
3. Apply a cardiac monitor and examine rhythm strip for arrhythmias
4. Check blood glucose level
5. Monitor EtCO₂ for respiratory decompensation
6. Check a 12-lead EKG when possible, with particular attention to potential tachydysrhythmias, ischemic changes or QRS or QTc prolongation
7. Check for trauma, self-inflicted injury
8. Law enforcement should have checked for weapons and drugs, but you may need to repeat the inspection
9. Discuss presentation and management recommendations with Poison Center by calling 800-222-1222 (will assist caller by connecting to local Poison Control Center)

Treatments and Interventions

1. IV access for any fluids and medications. If unable to start IV, the intranasal and intramuscular routes can be used. If obtunded or the patient is in critical condition and IV access cannot be obtained, IO access should be obtained.
2. If available, administer activated charcoal 1gm/kg PO once, for patients who have had a recent oral ingestion within the past 1-2 hours, with a patent airway and appropriate mental status. Communicate to the hospital activated charcoal was administered.

3. Treat chest pain as acute coronary syndrome (ACS) and follow your local ST-Elevation Myocardial Infarction (STEMI) Guideline if the EKG is consistent with STEMI
4. Treat shortness of breath as atypical ACS vs pulmonary edema. Administer oxygen as appropriate with a target of achieving 94–98% saturation. Positive pressure ventilation may be required if pulmonary edema is present.
5. Give IV fluids for poor perfusion, hypotension, or dehydration; cool fluids for hyperthermia
6. Treat seizures with benzodiazepines per local guidelines or protocols
7. Consider medications to reduce agitation and other significant sympathomimetic findings (significant hypertension or tachycardia):
Benzodiazepines are first line and can be given intravenously, intranasally, or intramuscularly. Repeat and high doses may be required, and airway/breathing should be carefully monitored with pulse-oximetry and end-tidal CO₂ monitoring. For complete list of medications and recommended dosing, please refer to NASEMSO's Agitated or Violent Patient/Behavioral Emergency Protocol or your local EMS agency's respective protocols. Below are a few options from NASEMSO dosing guidelines:

- i. Diazepam

1. Adults:

- a. 5 mg IV; 2–5 minute onset of action

OR

- b. 10 mg IM; 15–30 minute onset of action

2. Pediatrics:

- a. 0.05–0.1 mg/kg IV (maximum dose is 5 mg)

OR

- b. 0.1–0.2 mg/kg IM (maximum dose is 10 mg)

Note: IM not recommended due to erratic absorption

- ii. Lorazepam

1. Adults:

- a. 2 mg IV; 2–5 minute onset of action

OR

- b. 4 mg IM; 15–30 minute onset of action

2. Pediatrics:

- a. 0.05 mg/kg IV (maximum dose is 2 mg)

OR

- b. 0.05 mg/kg IM (maximum dose is 2 mg)

- iii. Midazolam

1. Adults:

- a. 5 mg IV; 3–5 minute onset of action

OR

- b. 5 mg IM; 10–15 minute onset of action

OR

c. 5 mg IN; 3–5 minute onset of action

2. Pediatrics:

a. 0.05–0.1 mg/kg IV (maximum dose 5 mg)

OR

b. 0.1–0.15 mg/kg IM (maximum dose is 5 mg)

OR

c. 0.3 mg/kg IN (maximum dose is 5 mg)

Note: IN or IM administration is very effective if IV access cannot be quickly established. nb: IN dosage is typically only given once.

a.

Ketamine may be considered only for patients with the most severe presentations of delirium and agitation. Avoid this therapy if the exposure and cause of patient's symptoms is due to ketamine. Below dosage reflects NASEMSO guidelines; however, please refer to your EMS agency's clinical care guidelines/ protocols and follow maximum dosage limits set forth by your own agency's protocols.

i. Adults:

a. 1-2 mg/kg IV; 1 minute onset of action

OR

b. 4-5 mg/kg IM; 3-5 minute onset of action

ii. Pediatrics:

a. 1mg/kg IV

OR

b. 3 mg/kg IM

Note: utilize capnography for monitoring due to increased risk of respiratory depression requiring intubation/mechanical ventilation.

Antipsychotics like Haloperidol or Droperidol may be considered as well after or in conjunction with benzodiazepines, however, cardiac monitoring for QT-interval prolongation should be utilized.

7. Consider soft physical management devices (e.g. restraints) especially if law enforcement is involved in getting patients to cooperate. **Chemical sedation should be considered in patients who require soft physical restraints, particularly if the patient remains visibly agitated.**

8. If hyperthermia is suspected, begin cooling measures (e.g., remove clothing, apply cold or ice packs to axilla/groin, cold IV fluids).

Patient Safety Considerations

1. Calming patient as soon as possible for patient and pre-hospital personnel safety
2. Apply the least amount of physical management devices that are necessary to protect the patient and the clinicians. De-escalation techniques should be considered prior to restraint, if possible. Staff should have adequate manpower to

safely restrain a patient, and patient should be positioned supine (never prone). Capnography is recommended for monitoring, and frequent pulse, motor, and sensory exams of the extremities should be performed while restraints are in place.

3. Assessment for potential weapons or additional drugs is very important since these items can pose a threat not just to the patient but also to the EMS crew

Notes/Education Pearls

Key Considerations

1. Recognizing and treating hyperthermia, along with using sedatives to reduce heat production from muscle activity and implementing aggressive cooling, is crucial, as many deaths are attributable to hyperthermia
2. If law enforcement has placed the patient in handcuffs, this patient needs both adequate chemical sedation and ongoing physical security for safe transport. Have law enforcement in back of ambulance for the handcuffed patient or make sure proper non-handcuff physical management devices are in place before law enforcement leaves and ambulance departs from scene.
3. If patient has signs and symptoms of ACS, consider giving up to three doses of nitroglycerin sublingual (SL) every 3–5 minutes if no concern for inferior STEMI and SBP greater than 110 mmHg, until pain resolves.
4. Maintaining IV access, cardiac monitor, and SPO₂/EtCO₂ monitors are key to being able to catch patient decompensation and intervene in a timely manner.
5. The risk of monitor and device displacement increases with increased agitation. Chemical and physical sedation should be considered in these scenarios to reduce this risk.
6. Cocaine has sodium channel blocking effects and can cause significant cardiac conduction abnormalities with a widened QRS. Treatment is with sodium bicarbonate similar to a tricyclic antidepressant. Check a 12-lead EKG to assess for these complications and consider providing sodium bicarbonate
 - a. Sodium bicarbonate dosage administration: 1 – 2 amps (50 – 100 mEq) IV/IO once for QRS > 100ms.

Pertinent Assessment Findings

1. History is as important as the physical examination. Look around the scene for clues of the exposure nature such as empty pill bottles or drug paraphernalia.
2. If the patient is found improperly dressed or undressed, this should increase the suspicion for stimulant overdose and possibly hyperthermia. These substances increase the risk for sudden death secondary to delirium with associated hyperthermia. Neuroleptic malignant syndrome or serotonin syndrome can also present with similar signs and symptoms.
3. Be prepared for the potential of cardiovascular collapse as well as respiratory arrest.
4. If a vasopressor is needed, norepinephrine is considered the first line, followed by epinephrine. Dopamine is not recommended due to increased risk of atrial dysrhythmias.

Quality Improvement

Associated NEMSIS Protocol(s) (eProtocol .01)

9914053 – General - Behavioral/Patient Restraint

Key Documentation Elements

- Reason for chemical sedation and physical restraint procedures used and neurologic/circulatory exams before and during their use
- Reason for medications selected
- Documentation of vital signs, including temperature
- Documentation of QT and QTc interval when antiemetic medications, haloperidol or droperidol is used and result conveyed to ED staff
- Documentation of QRS interval with suspected cocaine abuse
- Documentation of Poison Center contact and recommendations

Performance Measures

- Recognition and treatment of hyperthermia
- Recognition of need for monitoring cardiovascular and respiratory status of patient with stimulant toxicity
- ACS evaluation and treatment considered for chest pain and shortness of breath
- Respiratory compromise quickly recognized and treated
- Cardiovascular compromise quickly recognized and treated
- Patient and medics did not suffer any harm
- Vascular access and monitoring were not lost during transport
- Poison Center was contacted for recommendations

References

1. Kupas, D, Wydro, G, Tan, D, Kamin, R, Harrell, A, Wang, A, NASEMSO Position Paper 2020 Clinical Care and Restraint of Agitated or Combative Patients by Emergency Medical Services Practitioners <https://nasemsso.org/wp-content/uploads/Clinical-Care-and-Restraint-of-Agitated-or-Combative-Patients-by-Emergency-Medical-Services-Practitioners.pdf>. Accessed March 11, 2022
2. Silbergleit R, Lowenstein D, Durkalski V, Conwit R; Neurological Emergency Treatment Trials (NETT) Investigators. RAMPART (Rapid Anticonvulsant Medication Prior to Arrival Trial): a double-blind randomized clinical trial of the efficacy of intramuscular midazolam versus intravenous lorazepam in the prehospital treatment of status epilepticus by paramedics. *Epilepsia*. 2011 Oct;52 Suppl 8(Suppl 8):45-7. doi: 10.1111/j.1528-1167.2011.03235.x. PMID: 21967361; PMCID: PMC3211107.
3. Warrcik BJ, Hill M, Hekman K, et al. A 9-state analysis of designer stimulant, "bath salt," hospital visits reported to poison control centers. *Ann Emerg Med*. 2013;62(3):244–51
4. *White Paper Report on Excited Delirium Syndrome*. ACEP Excited Delirium Task Force, *American College of Emergency Physicians*; September 10, 2009

Sulfur Mustard, Lewisite

Introduction

Toxin Name Aliases

Sulfur Mustard, H, HD, Bis-(2-chloroethyl) sulfide

Lewisite, L, 2-Chlorovinyl dichloroarsine

Mixture of Sulfur Mustard and Lewisite, HL

Patient Care Goals

1. Remove patients from hazardous environments. Decontaminate to remove continued sources of absorption, ingestion, or inhalation
2. Identify intoxicating agent through the following means:
 - examination of container shapes, placards, labels, shipping documents or analytical testing
 - toxidrome
3. Assess risk for organ system impairments (eyes, skin, respiratory, gastrointestinal, neurologic)
4. Identify antidote (if Lewisite) or mitigating agent
5. Treat signs and symptoms in effort to stabilize patient, and understand likelihood for delayed clinical effects, especially in the case of sulfur mustard exposure.

Patient Presentation

Sulfur Mustard and Lewisite are both blister agents (vesicants). Sulfur mustard causes severe, delayed burns to the eyes, skin, and respiratory tract, with symptoms appearing hours after exposure. This chemical damages cells within minutes of contact, and in high doses, it can be lethal. Sulfur mustard also impacts the nervous system that regulates routine bodily functions, leading to "cholinergic toxicity," which is characterized by excessive salivation, tearing, miosis, and urination; gastrointestinal cramping, vomiting and less commonly, diarrhea. Historically, sulfur mustard has been deployed as a chemical weapon to inflict delayed injuries.

Lewisite is a highly toxic blister agent containing arsenic that impacts the lungs and causes systemic effects throughout the body. Although developed as a potential chemical warfare agent, it has not been used in combat. High levels of exposure can be fatal. Unlike mustard agents, lewisite causes immediate pain and irritation upon contact with its liquid or vapor form, and the resulting tissue damage appears rapidly. Lewisite also produces a burning sensation in the respiratory tract at concentrations too low to be detected by odor alone. This ability to cause prompt irritation makes the eye and respiratory injuries less severe than the ones with sulfur mustard. Victims will tend to close their eyes or hold their breath when exposed to Lewisite.

Inclusion Criteria

Presentation may vary depending on the concentration and duration of exposure. Both Sulfur Mustard and Lewisite can enter the body through inhalation, ingestion, or contact

with the skin or eyes. Inhalation is an important route of exposure, while ingestion is less common. Signs and symptoms vary, and may include, but are not limited to, the following:

1. Dermal (dose-related, latent period of 4-12 hours with Sulfur Mustard)
 - a. Pain (immediate with Lewisite)
 - b. Itching
 - c. Burning
 - d. Erythema
 - e. Hyperpigmentation (less with Lewisite)
 - f. Vesicles
 - g. Bullae
 - h. Skin necrosis
 - i. First/second/third degree burns (depending on vapor versus liquid contact)

2. Eye
 - a. Ocular pain
 - b. Miosis
 - c. Photophobia
 - d. Lacrimation
 - e. Blurred vision to blindness
 - f. Blepharospasm
 - g. Corneal damage

3. Respiratory
 - i. Hoarseness
 - j. Cough
 - k. Sore throat
 - l. Chest pressure
 - m. Bronchospasm / wheezing
 - n. Respiratory depression
 - o. Airway obstruction (hemorrhagic pulmonary edema)
 - p. Chemical burns to respiratory tract

4. Gastrointestinal
 - a. Nausea
 - b. Vomiting
 - c. Diarrhea (less common with sulfur mustard)

5. Neurologic
 - a. insomnia
 - b. hyperexcitability
 - c. convulsions

6. Sulfur mustard may emit a smell similar to garlic, onion, horseradish, or mustard. However, odor alone is an unreliable indicator of its presence and should not be relied

upon to detect sulfur mustard exposure. Lewisite has a distinct geranium-like odor. Sulfur mustard, unlike Lewisite, can cause a delayed suppression of the bone marrow with resulting neutropenia.

Exclusion Criteria

None noted.

Patient Management

Assessment

1. Don appropriate PPE – Level A
2. Remove patient from contaminated area into decontamination corridor and decontaminate
 - Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag
 - Thoroughly wash and rinse (using tepid water) the contaminated skin of the patient/victim using a soap and water solution.
 - For sulfur mustard: The sulfur mustard binds rapidly to the skin and effective decontamination would need to have been started by the victim or soldier on the scene. Since EMS arrival will likely be delayed, the EMS decontamination is primarily to decrease the risk of secondary exposure. This is because it is likely that the victim's skin damage would have already occurred.
 - Blisters form from 2-18 hours after exposure and vary in size. The blisters do not contain sulfur mustard liquid and should be kept intact in the prehospital setting. In the hospital, blisters larger than 1 cm in diameter will be unroofed.
3. Irrigate eyes with water for at least 15 minutes
4. Cover the patient/victim to prevent shock and loss of body heat.
5. Move the patient/victim to an area where emergency medical treatment can be provided.
6. Assess ABCDE (airway, breathing, circulation, disability).
7. Vital signs - pulse, temperature, O₂ saturation, blood pressure, respiratory rate, and neurologic status assessment.
8. Attach cardiac monitor and examine rhythm strip for arrhythmias (consider 12-lead EKG)
9. Check blood glucose level
10. Monitor pulse oximetry and end-tidal capnography (EtCO₂) for respiratory
11. decompensation
12. Perform carboxyhemoglobin device assessment, if available
13. Obtain pertinent cardiovascular history and other prescribed medications
14. Check for evidence of agent involved in exposure or trauma
15. Obtain any other pertinent patient history
16. Perform remainder of physical examination
17. Discuss presentation and management recommendations with Poison Center by calling 800-222-1222 (will connect to local Poison Control Center)

Treatments and Interventions

1. Mainstay of therapy is decontamination followed by supportive care
2. Ensure a patent airway, support ventilation with BVM if needed, and administer oxygen for hypoxia
3. Early intubation in patients with impending airway compromise
4. Establish IV access and administer fluids/vasoactive medications for cardiovascular collapse
5. BAL could be administered intramuscularly in the healthcare setting for systemic toxicity from Lewisite exposure. Pre-notify hospital of concern for Lewisite exposure so antidote can be obtained promptly.

Patient Safety Considerations

1. Scene/environmental safety is of primary importance for prehospital providers and patients
2. Early and adequate decontamination is the mainstay of therapy. Prehospital providers should wear appropriate PPE.
3. Remainder of treatment is primarily supportive care. For lewisite exposure, administer rapidly BAL topically to the exposed eyes or skin but this is unlikely to be available with EMS and the topical preparations are no longer being manufactured. Intramuscular BAL formulations are available and can be used for the systemic effects of Lewisite. This is also unlikely to be available in the prehospital setting.
4. Continuously reassess ABCs.
5. Maintain or normalize patient temperature
6. Call 1-800-222-1222 to be connected to the nearest regional poison center for additional assistance

Notes/Education Pearls

Key Considerations

1. Lewisite symptoms will occur immediately in contrast to delayed symptoms with sulfur mustard
2. Both agents are vesicants and will cause damage to skin and mucous membranes
3. BAL may be considered in patients exposed to Lewisite

Pertinent Assessment Findings

Frequent reassessment (vitals, signs, and symptoms) is essential as patient deterioration can be rapid and catastrophic

Quality Improvement

Associated NEMSIS Protocol(s) (eProtocol .01)

N/A

Key Documentation Elements

1. Repeat evaluation and documentation of vitals, signs and symptoms as the patient's clinical condition may deteriorate rapidly

2. Identification of possible etiology of poisoning
3. Time of symptom onset and time of initiation of exposure-specific treatments
4. Therapy and response to therapy

Performance Measures

1. Early and adequate decontamination
2. Early airway management in the rapidly deteriorating patient
3. Accurate exposure history
 - Time of ingestion/exposure
 - Route of exposure
 - Quantity of medication or toxin taken (safely collect all possible medications or agents)
 - Alcohol or other intoxicant taken
4. Appropriate protocol selection and management
5. Multiple frequent documented reassessments

References

1. Chemical Weapons. In: Nelson LS, Howland M, Lewin NA, Smith SW, Goldfrank LR, Hoffman RS. eds. Goldfrank's Toxicologic Emergencies, 11e. McGraw-Hill Education; 2019.
2. Mustard-Lewisite Mixture (HL): Blister Agent. Published 2024. https://www.cdc.gov/niosh/ershdb/emergencyresponsecard_29750007.html
3. NASEMSO Medical Directors Council. National Model EMS Clinical Guidelines NASEMSO National Model EMS Clinical Guidelines.; 2022. https://nasemso.org/wp-content/uploads/National-Model-EMS-Clinical-Guidelines_2022.
4. Dacre JC, Goldman M. Toxicology and pharmacology of the chemical warfare agent sulfur mustard. *Pharmacol Rev.* 1996 Jun;48(2):289-326.
5. "Lewisite (L): Blister Agent." *Centers for Disease Control and Prevention*, Centers for Disease Control and Prevention, 12 May 2011, www.cdc.gov/niosh/ershdb/emergencyresponsecard_29750006.html.
6. McManus J, Huebner K. Vesicants. *Crit Care Clin.* 2005 Oct;21(4):707-18, vi.
7. "Mustard - Prehospital Management." *Mustard Prehospital Management*, chemm.hhs.gov/mustard_prehospital_mmg.htm. Accessed 3 Nov. 2024.
8. "Sulfur Mustard: Blister Agent." *Centers for Disease Control and Prevention*, Centers for Disease Control and Prevention, 12 May 2011, www.cdc.gov/niosh/ershdb/emergencyresponsecard_29750008.html.

Unknown Chemical Agents

Introduction

Toxin Name Aliases

None Noted

Patient Care Goals

1. Removal of patients from hazardous environments. Decontamination when appropriate.
2. Stabilize patient and assess disabilities
3. Identify agent responsible for exposure by toxidrome and / or environmental testing
4. Identify antidote or mitigating agent
5. Symptomatic and supportive care

Patient Presentation

Inclusion Criteria

Suspected chemical exposure

1. Presentation may vary depending on the chemical, concentration, nature, and duration of exposure. Signs and symptoms vary, and may include, but are not limited to, the following:
 - a. Ears, Nose, Throat
 - i. Eye pain or redness
 - ii. Tearing
 - iii. Tinnitus
 - iv. Visual disturbances
 - v. Stridor
 - vi. Drooling
 - b. Cardiovascular
 - i. Rapid or slow heart rate
 - ii. Dysrhythmias
 - iii. Hypotension
 - c. Respiratory
 - i. Dyspnea
 - ii. Wheezing
 - iii. Respiratory depression or arrest
 - iv. Pulmonary edema
 - d. Gastrointestinal
 - i. Abdominal pain

- ii. Nausea
- iii. Vomiting
- iv. Diarrhea
- v. Defecation
- vi. Urinary incontinence or retention
- e. Neurological
 - i. Altered mental status
 - ii. Seizures
 - iii. Dilated or constricted pupils
 - iv. Hyperreflexia / myoclonus
- f. Dermal
 - i. Thermal or caustic burns
 - ii. Skin sloughing
 - iii. Rashes
 - iv. Sweating

2. Signs and symptoms may manifest differently depending on the route of exposure. While all routes have the potential to cause systemic symptoms, ingestion and inhalation have the highest risks. The following are special considerations given based on the route of exposure.

- a. Ingestion
 - i. Direct gastrointestinal injury (perioral, oral, esophageal, gastric)
- b. Inhalation
 - i. Direct lung injury
 - ii. Thermal or caustic glottic, subglottic or tracheal injury
 - iii. Stridor
 - iv. Wheezing
 - v. Sooty sputum
 - vi. Respiratory distress
- c. Injection
 - i. Localized pain
 - ii. Puncture wound
 - iii. Skin discoloration
 - iv. Tingling or numbness
 - v. Localized muscle twitching
 - vi. Reddening skin
 - vii. Rapid local swelling
 - viii. Metallic taste

3. Toxidromes (constellations of signs and symptoms that assist in the identification of certain classes of medications and their toxic manifestations). Toxidromes may be incomplete due to exposure to toxins with counteracting effects.

- a. Antimuscarinic (i.e., "anticholinergic" like diphenhydramine or jimson weed)
 - i. Red as a beet (flushed skin)

- ii. Dry as a bone (dry skin)
- iii. Mad as a hatter (altered mental status)
- iv. Blind as a bat (mydriasis)
 - v. Hot as a pistol (hyperthermia)
 - vi. Full as a flask (urinary retention)
 - vii. "Tacky" like a pink flamingo (tachycardia)
- b. Cholinergic (**DUMBELS**) **DUMBELS** is a mnemonic used to describe the signs and symptoms of acetylcholinesterase inhibitor agent poisoning.
 - i. **D**iarrhea
 - ii. **U**rination
 - iii. **M**iosis/Muscle weakness
 - iv. **B**ronchospasm/**B**ronchorrhea/**B**radycardia (the killer Bs)
 - v. **E**mesis
 - vi. **L**acrimation
 - vii. **S**alivation/Sweating
- c. Opioids
 - i. Respiratory depression
 - ii. Miosis (pinpoint pupils)
 - iii. Altered mental status
- d. Sedative Hypnotic
 - i. Central nervous system depression
 - ii. Ataxia (unstable gait or balance)
 - iii. Slurred speech
 - iv. Normal or depressed vital signs (pulse, blood pressure, respiratory rate, neurologic status assessment)
- e. Stimulants (Sympathomimetic)
 - i. Tachycardia, tachydysrhythmias
 - ii. Hypertension
 - iii. Diaphoresis
 - iv. Delusions/paranoia
 - v. Seizures
 - vi. Hyperthermia
 - vii. Mydriasis (dilated pupils)
- f. Serotonin Syndrome - definitive diagnosis based on altered mental status, autonomic instability, and neuromuscular findings after exposure to serotonergic agent.
 - i. Altered mental status
 1. Agitation
 2. Confusion
 3. Coma
 - ii. Autonomic Instability
 1. Tachycardia
 2. Hypertension / hypotension
 3. Hyperthermia / hypothermia

4. Diaphoresis
- iii. Neuromuscular findings
 1. Hyperreflexia
 2. Clonus
 3. Tremors / shivering
 4. Abnormal eye movements

Exclusion Criteria

None noted

Patient Management

Assessment

1. Make sure the scene is safe. Recommend ambient air testing (e.g., carbon monoxide, hydrogen sulfide) when available.
2. Consider body substance isolation (BSI) or appropriate PPE based on degree of contamination and suspected toxins if known
3. Remove patient's clothing, jewelry, and other attachments, which may pose secondary risks to patient and rescuers. If these items are heavily contaminated, leave at the scene to reduce contamination of transport vehicles and the Emergency Department.
4. Assess ABCDs and, if indicated, perform wet or dry decontamination to make the patient as clean as possible. Expose patient for assessment and then re-cover to ensure retention of body heat.
5. Vital signs - pulse, blood pressure, respiratory rate, neurologic status assessment, temperature, and oxygen saturation
6. Attach cardiac monitor and examine rhythm strip for arrhythmias (consider 12-lead EKG)
7. Check blood glucose level
8. Monitor end-tidal capnography (EtCO₂) for ventilatory compromise, and pulse oximetry for pulmonary decompensation
9. Perform carboxyhemoglobin device assessment, if available
10. When indicated, identify specific medication taken (including immediate release vs sustained release), time of ingestion, dose, and quantity. When appropriate, bring all medications (prescribed and not prescribed) found in the environment. For in home or occupational non-pharmaceutical exposure, obtain brand name and chemical information, if known.
 - a. Utilize secondary sources at scene for further history or detail. Also obtain history of alcohol or another co-toxicant taken if applicable.
11. If bringing in the possible exposure agent, consider the threat to rescuers and treatment personnel and the destination facility. Do not move the patient into the ED unless the facility and the staff are appropriately protected. Some EDs will perform decontamination outside of the physical department or hospital. Only transport medications and safely secured, non-volatile and non-

reactive chemicals. That substance may need to be kept safely outside the doors of the ED

12. Law enforcement should have checked for weapons and drugs, but you may need to re-check

13. Check for needle marks, paraphernalia, bites, bottles, or evidence of agent involved in exposure, self-inflicted injury, or trauma

14. Obtain any other pertinent patient history

15. Perform remainder of physical examination

Treatments and Interventions

1. Ensure that airway is patent

2. Administer oxygen as appropriate with a target of achieving 94–98% saturation. If available, administer humidified oxygen. If there is hypoventilation noted, support breathing with BVM.

3. Obtain blood samples for POC testing when indicated (e.g., COHb, glucose, lactate)

4. Initiate IV access for infusion of treatment medication using lactated Ringer's or normal saline if indicated.

5. Consider fluid bolus (20 mL/kg) if evidence of hypoperfusion

6. Administration of appropriate antidote or mitigating medication if available to agency (refer to specific agent guideline if not listed below)

a. Acetaminophen overdose:

i. If available, consider activated charcoal without sorbitol (1 g/kg) PO only if within the first hour of ingestion *and* prolonged transport to definitive care

ii. Based on suspected quantity and timing, consider acetylcysteine (pediatric and adult), if available

1. Loading dose is acetylcysteine 150 mg/kg IV; mix in 200 mL of dextrose 5% in water (D5W) and infuse over 1 hr

2. After loading dose, give acetylcysteine 50 mg/kg IV in 500 mL D5W over 4 hrs.

3. Note and record the time that acetylcysteine IV administration was started.

4. If IV is not available, acetylcysteine 140 mg/kg PO

iii. If risk of rapidly decreasing mental status, do not administer oral agents

b. Salicylate (Aspirin) overdose:

i. Consider activated charcoal without sorbitol (1 gm/kg) PO only if within the first hour of ingestion

1. As ASA is erratically absorbed, charcoal is highly recommended to be administered early

2. If altered mental status or risk of rapid decreasing mental status from polypharmacy, do not administer oral agents including activated charcoal

- ii. In salicylate poisonings, let the patient breathe on their own, even if tachypneic, until there is evidence of decompensation or hypoxia. Acid/base disturbances and outcomes worsen when the patient is manually ventilated. If patient requires BMV, hyperventilate to maintain initial respiratory rate.
- c. Benzodiazepine overdose:
 - i. Respiratory support
 - ii. Monitor neurological status, ensure airway protection
 - iii. Position to prevent aspiration
- d. Caustic substances ingestion (i.e., acids and alkali):
 - i. Evaluate for airway compromise secondary to spasm or direct injury associated with oropharyngeal burns
 - ii. Secure airway early if there are signs of upper airway edema (i.e., stridor)
- e. Dystonia (symptomatic), extrapyramidal signs or symptoms, or mild allergic reactions
 - i. Consider administration of diphenhydramine
 - 1. **Adult:** diphenhydramine 25–50 mg IV or IM
 - 2. **Pediatric:** diphenhydramine 1–1.25 mg/kg IVP/IO or IM (maximum single dose of 25 mg)
- f. Monoamine oxidase inhibitor overdose (symptomatic, e.g., MAOI; isocarboxazid, phenelzine, selegiline, tranylcypromine)
 - i. Consider administration of midazolam for temperature control
 - ii. **Adult and Pediatric:** Midazolam 0.1 mg/kg in 2 mg increments slow IV push over one to two minutes per increment with maximum single dose 5 mg — reduce by 50% for patients 69 years old or older
- g. Opioid overdose, treat per the Opioid Poisoning/Overdose Guideline
 - i. Assess breathing
 - 1. Pulse oximetry
 - 2. End-tidal CO₂
 - ii. Naloxone IN, IM, IV or IO for respiratory depression or severe CNS depression with failure to protect to airway
 - iii. Monitor for pulmonary edema
 - iv. Monitor for withdrawal or recurrence of opioid intoxication
- h. Organophosphates, carbamates, and nerve agents
 - i. See Acetylcholinesterase inhibitor guidelines
- i. Selective serotonin reuptake inhibitors (SSRIs)
 - i. Consider early airway management
 - ii. Non-perfusing dysrhythmias resulting in cardiac arrest should be treated with Advanced Cardiac Life Support (ACLS)
 - iii. Aggressively control hyperthermia
 - 1. Control psychomotor agitation with benzodiazepines
 - 2. Active cooling measures
 - iv. Consider fluid challenge (20 mL/kg) for hypotension

- v. Consider vasopressors after adequate fluid resuscitation (1–2 liters of crystalloid in adult) for the hypotensive patient
- vi. For agitation, consider midazolam
 - 1. **Adult:** midazolam 0.1 mg/kg in 2 mg increments slow IV push over one to two minutes per increment with maximum single dose 5 mg
 - a. Reduce by 50% for patients 69 years or older
 - 2. **Pediatric:** midazolam 0.1 mg/kg in 2 mg increments slow IV push over one to two minutes per increment with maximum single dose 4 mg or midazolam 0.2 mg/kg IN to maximum single dose of 10 mg
- vii. For seizures, treat per Seizure Guideline.
- j. Tricyclic Antidepressant (TCA)/Sodium Channel Blocker Overdose:
 - i. Consider early airway management
 - ii. If widened QRS (100 msec or greater) or a terminal R wave >3 mm in avR, consider sodium bicarbonate 1–2 mEq/kg IV, this can be repeated as needed to narrow QRS and improve blood pressure
 - iii. Immediate sodium bicarbonate administration for any dysrhythmias. Include sodium bicarb administration in ACLS for cardiac arrest.
 - iv. Consider fluid challenge (20 mL/kg) for hypotension
 - v. Consider vasopressors after adequate fluid resuscitation (1–2 liters of crystalloid) for the hypotensive patient.
 - vi. For agitation, consider midazolam.
 - 1. **Adult:** midazolam 0.1 mg/kg in 2 mg increments slow IV push over one to two minutes per increment with maximum single dose 5 mg
 - a. Reduce by 50% for patients 69 years or older
 - 2. **Pediatric:** midazolam 0.1 mg/kg in 2 mg increments slow IV push over one to two minutes per increment with maximum single dose 4 mg or midazolam 0.2 mg/kg IN to maximum single dose of 10 mg
 - vii. For seizures, treat per Seizures guideline
- k. If there is a risk of rapidly decreasing mental status, ingestion of a caustic substance, or for petroleum-based ingestions, do not administer oral agents
- 7. Consider administration of activated charcoal without sorbitol (1 g/kg) if within the first 1 hour after ingestion (including acetaminophen) *and* there will be prolonged transport to definitive care.
 - a. Patients who have ingested medications with extended release or delayed absorption may also be administered activated charcoal

Patient Safety Considerations

1. Scene/environmental safety for patient and clinician
 - a. Consider ambient air testing when available and indicated (e.g., CO, hydrogen sulfide)
2. Monitor patient airway, breathing, pulse oximetry, EtCO₂ for adequate ventilation as they may change over time
3. Repeat vital signs often
4. Frequent neuro checks to monitor level of consciousness
5. Monitor EKG with special attention to rate, rhythm, QRS and QT duration
6. Maintain or normalize patient temperature
7. The regional poison center should be engaged as early as reasonably possible to aid in appropriate therapy and to track patient outcomes to improve knowledge of toxic effects. **The national 24-hour toll-free telephone number to poison control centers is (800) 222-1222**, and it is a resource for free, confidential expert advice from anywhere in the United States
8. Unknown exposures in the industrial setting may need to involve serial communications between the scene and the ED, as testing done at either site may be needed to identify the responsible toxin. When there are criminal considerations (e.g., illicit methamphetamine synthesis), law enforcement may also be involved in those communications.
9. When the patient expires or is at risk of dying, communication pathways will need to include the medical examiner's office to protect individuals from secondary exposure when the body has been contaminated.

Notes/Education Pearls

Key Considerations

1. Each toxin or overdose has unique characteristics which must be considered in individual protocols
2. Activated charcoal (which does not bind to all medications or agents) is still a useful adjunct in the serious-agent, enterohepatic, or extended-release agent poisoning if the patient does not have the potential for rapid alteration of mental status or airway/aspiration risk. Precautions should be taken to avoid or reduce the risk of aspiration
3. Gastric decontamination may need to be performed in the obtunded patient after securing the airway. Not used in caustic ingestions.
4. Ipecac is not recommended for any poisoning or toxic ingestion — the manufacturer has stopped production of this medication
5. Flumazenil is **not** indicated in a suspected benzodiazepine overdose as it can precipitate refractory/intractable seizures if the patient is a benzodiazepine dependent patient

Pertinent Assessment Findings

Frequent reassessment is essential as patient deterioration can be rapid and catastrophic

Quality Improvement

Associated NEMSIS Protocol(s) (eProtocol .01)

9914135—General - Overdose/Poisoning/Toxic Ingestion

Key Documentation Elements

- Repeat evaluation and documentation of signs and symptoms as patient clinical conditions may deteriorate rapidly
- Identification of possible etiology of poisoning
- Initiating measures on scene to prevent exposure of bystanders when appropriate/indicated
- Time of symptoms onset and time of initiation of exposure-specific treatments

Performance Measures

- Early airway management in the rapidly deteriorating patient
- Accurate exposure history
 - Time of ingestion/exposure
 - Route of exposure
 - Quantity of medication or toxin taken (safely collect all possible medications or agents)
- Alcohol or another intoxicant taken
- Appropriate protocol selection and management
- Multiple frequent documented reassessments

References

1. Boyer EW, Shannon MS. The serotonin syndrome. *N Engl J Med*. 2005; 352:1112–20
2. Bruccoleri RE, Burns MM. A Literature Review of the Use of Sodium Bicarbonate for the Treatment of QRS Widening. *J Med Toxicol*. 2016 Mar;12(1):121-9. doi: 10.1007/s13181-015-0483-y. PMID: 26159649; PMCID: PMC4781799
3. Cushing TA. Selective Serotonin Reuptake Inhibitor Toxicity <https://emedicine.medscape.com/article/821737-overview>. Updated April 24, 2018. Accessed March 11, 2022
4. Gresham C. Benzodiazepine toxicity treatment and management. <http://emedicine.medscape.com/article/813255-treatment#d10>. Updated January 23, 2020. Accessed March 11, 2022
5. Hoffman RS, Howland MA, Lewin NA, Nelson LS, Goldfrank LR. *Goldfrank's Toxicologic Emergencies, 10th Edition*. China: McGraw -Hill Education;2015.<http://accessemergencymedicine.mhmedical.com/book.aspx?bookID=1163> Accessed March 11, 2022
6. Lemyze M, Masse J, Queva C, Huchette D. Cardiac effect of sodium bicarbonate in sodium- channel blocker poisoning. *Intensive Care Med*. 2016 Apr;42(4):588-590. doi: 10.1007/s00134-015-4122-5
7. Spiller H. A prospective evaluation of the effect of activated charcoal before N-Acetyl cysteine in acetaminophen overdose. *Ann of Emerg Med*. 1994;23(3):519 -23
8. Tsai V. Tricyclic Antidepressant Toxicity. <http://emedicine.medscape.com/article/819204-overview>. Updated May 19, 2020. Accessed March 11, 2022
9. Wolf S. Clinical policy: critical issues in the management of patients presenting to the emergency department with acetaminophen overdose. *Ann of Emerg Med*. 2007;50(3):292– 313

White Phosphorus

Introduction

Toxin Name Aliases

- White Phosphorus
- Yellow Phosphorus
- WP

All references in this document to “phosphorus” refer to elemental white phosphorus. Red phosphorus is a different compound (allotrope); although it is elemental phosphorus it does not burn as white phosphorus does nor does it have the same toxicity.

Patient Care Goals

- Rapid gross decontamination of contaminated and melted clothing while providing initial wound covering
- Limit further injury to the patient by preventing re-ignition of phosphorus
- Limit systemic absorption of phosphorus
- Prevent injury to bystanders and the health care providers from igniting and splattering phosphorus

Patient Presentation

- White phosphorus is primarily found in military munitions used as an incendiary and to create smokescreens.
- White phosphorus has a consistency similar to candle wax; at low temperatures it is solid. Once it reaches an approximate temperature above 34°C it will spontaneously ignite and melt in a strongly energetic reaction and reach temperatures over 800°C causing a combination of thermal and chemical injury.
- If water is applied directly to burning phosphorus it splatters and will not extinguish, similar to a hydrocarbon fire.
- White phosphorus reacts with oxygen to produce phosphorus pentoxide (P_4O_{10}), a compound that causes inhalation and mucosal injury.
- White phosphorus and wounds contaminated by white phosphorus may appear to smolder and produce white “smoke”. In this case phosphorus is not fully ignited but is reacting with oxygen in the air producing phosphorus pentoxide.
- While phosphorus producing the white “smoke” has not ignited, it is still oxidizing, producing heat and acid while consuming water.
- Residual white phosphorus may remain either at the scene near the patient, on articles of clothing, or embedded in the patient’s burns.
- Removal of embedded white phosphorus should not be attempted in the field; instead the focus is on external decontamination and prevention of re-ignition.

Inclusion Criteria

- Includes patients with dermal burns following confirmed or potential exposure to burning white phosphorus.
- Includes patients suffering from burns most likely due to white phosphorus even if there is not believed to be any white phosphorus remaining.
- Includes patients with coverings (cloth, sand) over body areas where there was history suggestive of exposure to phosphorus
- Includes patients with inhalational exposure (and/or ocular irritation) *and* burns, recognizing that the acute inhalation exposure to burning phosphorus is due to its combustion product which is an ocular and pulmonary irritant of much less health concern; it is not direct exposure to elemental white phosphorus.

Exclusion Criteria

- Inhalation only (or ocular only) exposures to phosphorus smoke without any dermal component.
 - Inhalation exposures following burning white phosphorus (i.e., phosphorus pentoxide) are to the oxides of phosphorus, and not to the elemental phosphorus form
 - These exposures cause upper airway and mucus membrane irritation which are typically not severe; pulmonary injury would be very rare.
- Ingestion of white phosphorus

Signs & Symptoms

- Single or multiple patients suffering from dermal burns
- Burns can be any thickness but usually at least 2nd degree.
- Burns often appear in a splash or punctate pattern because the white phosphorus is burning and in a “liquid” state when it impacts the victim, appearing somewhat like they were splattered with paint.
 - It does not usually run or produce linear flow patterns that are typical of burns from hot liquids.
 - It also generally does not have a gradient pattern sometimes typical of chemical burns
 - These burns will usually be at least 2nd degree
- Clothing and/or wounds that continue to smolder especially if the “smoke” is white
- Potential for mild mucus membrane, ocular, and airway irritation from exposure to phosphorus pentoxide (oxide of phosphorus).

Patient Management

- Residual white phosphorus poses an ongoing threat to patients and health care providers as it can continue to cause injury to tissues, generate heat, and smolder or pop as well as reignite.
- Providers making initial patient contact and performing gross decontamination should be wearing clothing and personal protective equipment (PPE) that provides thermal protection and eye/face protection until gross decontamination has been completed due to risk of residual phosphorus.

- Eye protection should be worn through all stages of care; a face shield should be worn for close, prolonged work.
- Gross patient decontamination should occur before patient transport.
- Perform gross patient decontamination by removal of clothing and covering the wounds and burn areas with soaking wet clean linens or gauze.
 - Removed clothing and materials should be soaked in cool water and stored that way to reduce risk of residual phosphorus reacting with air and re-igniting
 - If sand was used to extinguish and cover an area, it should be wetted and kept moist and covered with wet linens or gauze until more definitive wound covering is applied.
- Phosphorus pentoxide (the white “smoke”) may cause ocular and respiratory irritation but does not cause inhalation burns or systemic illness.
- There is no requirement for health care personnel to wear a self-contained breathing apparatus (SCBA) or elastomeric respirator with chemical cartridge when managing a patient after gross decontamination has occurred.
- Discuss presentation and management recommendations with Poison Center by calling 800-222-1222 (will connect to local Poison Control Center).

Assessment

- Initial assessment of airway, breathing and circulation should be as early as possible and must be accompanied by
 - Efforts to prevent re-ignition of the phosphorus.
 - Efforts to keep the responders safe
- Evaluate anatomic areas burned and extent of burns using the burn assessment tool utilized by EMS System.
- Evaluate for any respiratory irritation or respiratory distress.
- Evaluate for other traumatic injuries

Treatments and Interventions

- If the patient is actively burning, extinguish the source by covering it.
 - The ideal tool for extinguishing is a clean wet cloth, however anything which will limit oxygen flow will initially work. Flammable items or those that will melt should be avoided (i.e. nylon).
 - Sand will work to extinguish and cover an area; if this is done it should be wetted as soon as possible to suppress the combustion.
- All wounds potentially caused by white phosphorus should be covered with new wet dressings even if they are not “smoking”; this will limit the damage and allow them to be assessed in a controlled environment.
- Before bringing a patient into an ED, all clothing and initial burn dressings should be switched out to clean cold wet gauze (and ideally any possibly contaminated clothes and old dressings should be submerged in water).
- If sand was initially used as a wound dressing, a source of running water will work to rinse it away as long as the area is carefully monitored. Once most of the sand (not

all - this is not a debridement step) of the sand has been washed away the area should be covered with clean cold wet gauze for transport

- When area is properly covered and there is no evidence of further combustion, the patient can be transported.
- Additional water source and wet linens should be available to layer on any areas during transport that develop a white fume; these areas should be pointed out during handoff
- Do not apply any creams or ointments
- Perform patient monitoring following EMS System protocols for a burn victim; there are no specific cardiac effects expected from phosphorus this rapidly after exposure.
- Provide pain control following EMS System protocols.
- Provide IV fluid bolus following EMS System protocols for burn and trauma.

Patient Safety Considerations

- Eye protection should be worn when handling a patient with white phosphorus external contamination. A face shield should be used if there is going to be prolonged close contact with a patient
- Do not pour water directly on burning phosphorus as it will instantly boil and splatter liquid phosphorus without the occurrence of extinguishing.
- Wounds contaminated with white phosphorus are known to “spark” without warning causing ejection of burning fragment that have caused harm to health care providers
- Be very cautious when uncovering wounds to examine the area; this should only be done under controlled environments when soaking wet cold clean gauze (or something similar) is immediately available and with face protection

Notes/Education Pearls

Key Considerations

- White phosphorus has a consistency similar to candle wax; at low temperatures it is solid. Once it gets above about 34°C it will spontaneously ignite, melt and reach temperatures over 800 °C causing both thermal and chemical burns.
- If water is applied directly to burning phosphorus it splatters and will not extinguish
- Provider safety with appropriate PPE is critical, however the white smoke fume only has minimal irritant toxicity; it does not have any white phosphorus in it
- Apply wet dressings to all wounds to halt thermal injury and prevent phosphorus from continuing to react with air.
- Formal debridement and wound cleaning will require time and a safe environment requiring sufficient lighting, assessment of fluorescence and total darkness to find trace phosphorescence.

Pertinent Assessment Findings

- Patient has dermal burns following exposure to burning white phosphorus.
- These burns often appear in a splash or punctate pattern
- Extinguished areas that are still contaminated may have a white smoke emanating from them which must be immediately addressed

Quality Improvement

Associated NEMIS Protocol(s) (eProtocol .01)

- 9914085 - Injury-Burns-Thermal
- 9914213 - Injury-Topical Chemical Burn
- 9914071 - General-Pain Control

Key Documentation Elements

- Decontamination efforts
- Body surface burn
- Respiratory symptoms
- Performance Measures

References

1. Aviv U, et al: The burning issue of white phosphorus: a case report and review of the literature. *Disaster Mil Med* 2017;3:6.
2. Barillo DJ, Cancio LC, Goodwin CW. Treatment of white phosphorus and other chemical burn injuries at one burn center over a 51 year period. *Burns* 2004; 30: 448-452.
3. Barqouni L, et al: Interventions for treating phosphorus burns: Cochrane Database of Systematic Reviews, John Wiley & Sons, Ltd, 2012, pp.
4. Ben-Hur N, et al: Phosphorus burns--a pathophysiological study. *Br J Plast Surg* 1972;25:238-244.
5. Bowen TE, et al: Sudden death after phosphorus burns: experimental observations of hypocalcemia, hyperphosphatemia and electrocardiographic abnormalities following production of a standard white phosphorus burn. *Ann Surg* 1971;174:779-784.
6. Brown BJ et al. The Acute effects of single exposures to white phosphorus smoke in rats and guinea pigs. September 1980.
7. Chou T et al. The management of white phosphorus burns. *Burns*. 2001; 27: 492-497.
8. Conner JC, et al: Images in clinical medicine. White phosphorus dermal burns. *N Engl J Med* 2007;357:1530.
9. Curreri PW, et al: The treatment of chemical burns: specialized diagnostic, therapeutic, and prognostic considerations. *J Trauma* 1970;10:634-642.
10. Eldad A, et al: The phosphorous burn - a preliminary comparative experimental study of various forms of treatment. *Burns* 1991;17:198-200.
11. Eldad A, et al: Phosphorous burns: evaluation of various modalities for primary treatment. *J Burn Care Rehabil* 1995;16:49-55.

12. Frank M et al. Not all that glistens is gold: civilian white phosphorus burn injuries. *American Journal of Emergency Medicine* 2008; 26:974.e3-974.e5.
13. Kaufman T, Ullmann Y, Har-Shai YH. Phosphorus Burns: A practical approach to local treatment. *JBCR* 1988;9(5): 474-475.
14. Konjoyan TR: White phosphorus burns: case report and literature review. *Mil Med* 1983;148:881-884.
15. Mazingo DW, et al: Chemical burns. *J Trauma* 1988;28:642-647.
16. Okazaki A et al. Chemical pneumonitis caused by inhalation of white phosphorus fumes. *Am J Respiratory and Critical Care Medicine* 2020; 201(4):e12.
17. Saracoglu KT, et al: Delayed diagnosis of white phosphorus burn. *Burns* 2013;39:825-826.
18. Song ZY, et al: Treatment of yellow phosphorus skin burns with silver nitrate instead of copper sulfate. *Scand J Work Environ Health* 1985;11 Suppl 4:33.
19. Summerlin WT, et al: White phosphorus burns and massive hemolysis. *J Trauma* 1967;7:476-484.
20. US Department of Justice Drug Enforcement Administration. Red Phosphorus, White Phosphorus (also known as Yellow Phosphorus), and Hypophosphorous Acid are used to Manufacture Methamphetamine (Accessed June 21, 2023, at https://www.deadiversion.usdoj.gov/chem_prog/advisories/phosphorus.htm)
21. Walker J, Jr., et al. Quantitative analysis of phosphorus-containing compounds formed in WP burns. Washington DC: Environmental Protection Research Division U.S. Army Medical Research and Development Command; 1969.
22. Willers-Russo LJ: Three Fatalities Involving Phosphine Gas, Produced as a Result of Methamphetamine Manufacturing. *Journal of Forensic Sciences* 1999;44(3):647.
23. Witkowski W, et al: Experimental Comparison of Efficiency of First Aid Dressings in Burning White Phosphorus on Bacon Model. *Med Sci Monit* 2015;21:2361-2366.
24. Kao DS, Hijjawi J. Cold and Chemical Injury to the Upper Extremity. Section III Burns/Surgery. In: Neligan PC, ed. *Plastic Surgery*. 3rd ed, Vol 4. New York: Saunders; 2013:456-67.
25. Germann G, Hrabowski M. Burned Hand in Green's operative hand surgery. 7th edition. Elsevier, Philadelphia, USA, 2017.