REGION 11 CHICAGO EMS SYSTEM PROTOCOL	Title: Chemical Airway Respiratory Irritants – BLS/ALS
	Section: Toxins and Environmental
	Approved: EMS Medical Directors Consortium
	Effective: August 15, 2024

CHEMICAL AIRWAY RESPIRATORY IRRITANTS – BLS/ALS

I. PATIENT CARE GOALS

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

2. Activate HAZMAT response to evaluate any potential chemical exposure.

II. PATIENT PRESENTATION

Airway respiratory irritants may include airway injuries, chemical respiratory injuries, respiratory injuries, respiratory irritants, and/or toxic inhalation.

A. Inclusion Criteria

1. Inhalation of a variety of gases, mists, fumes, aerosols, or dusts may cause irritation or injury to the airways, pharynx, lung, asphyxiation, or other systemic effects.

2. Inhaled airway respiratory irritants will interact with the mucous membranes and upper and lower airways based on solubility, concentration, particle size, and duration of exposure.

3. The less soluble and smaller the particle size of the inhaled airway respiratory irritant, the deeper it will travel into the airway and respiratory systems before reacting with adjoining tissues, thus causing a greater delay in symptom onset.

B. Exclusion Criteria

None

C. Signs and Symptoms

1. Many airway respiratory irritants have "warning properties" such as an identifiable or unpleasant smell or cause irritation to eyes or airways. Some agents do not have clear warning properties and will often have delayed onset of signs or symptoms.

2. As the type, severity and rapidity of signs and symptom onset depend on the agent, water solubility, concentration, particle size, and duration of exposure, the below signs and symptoms are often overlapping and escalating in severity:

    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
n. Change in voice
o. Airway obstructions including laryngospasm and laryngeal edema
p. Pulmonary edema (non-cardiogenic)
q. Seizures
r. Cardiac arrest

3. High water solubility/highly irritating airway respiratory irritants (oral/nasal and pharynx, particle size greater than 10 micrometers) include:

   a. Acrolein
   b. Ammonia
   c. Chloramine
   d. Ethylene oxide
   e. Formaldehyde
   f. Hydrogen chloride
   g. Methyl bromide
   h. Sodium azide
   i. Sulfur dioxide

4. Intermediate water solubility airway respiratory irritants (bronchus and bronchiole, particle size 5–10 micrometers) include:

   a. Chlorine

5. Low water solubility/less irritating airway respiratory irritants (alveolar, particle size less than 5 micrometers) include:

   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. Ozone

h. Phosgene

6. Asphyxia agents (two categories) include:

   a. Oxygen deprivation below 19.5% oxygen atmosphere ("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)

   b. Chemical interfering with oxygen delivery or utilization ("chemical asphyxiants")
      i. Carbon monoxide (see Carbon Monoxide/Smoke Inhalation – BLS/ALS Protocol)
      ii. Cyanide (see Cyanide Exposure – ALS Protocol)
      iii. Hydrogen sulfide

7. Inhalants of abuse include:

   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 inhalants of abuse 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 include:
      i. Volatile Solvents: Paint remover, degreasers, dry-cleaning fluids, gasoline, lighter fluid, correction fluid, felt tip markers, or glue.
      ii. Cosmetics/Paints/Sprays: Deodorant spray, vegetable oil spray, fabric protector spray, or spray paint.
      iii. Propellants/Asphyxiants/Nitrous Oxide: Propane gas, balloon tanks (helium), computer keyboard cleaner, ether, halothane, chloroform, butane, propane, or whipped cream dispensers.

8. Riot control agents (see Riot Control Agent – BLS/ALS Protocol)

9. A prototype agent is identified with each region of the affected airway respiratory track 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

track and the slower the rate of symptom onset, the less water soluble the airway respiratory irritant.

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 – BLS/ALS Protocol)
h. Anticholinesterase inhibitors (see Acetylcholinesterase Inhibitors – BLS/ALS Protocol)

10. 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 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

11. Chlorine

a. All of the above (see 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

12. Phosgene

a. Often have none of the above symptoms for the 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 wheezing
        C. Hypoxia
        D. Severe non-cardiac pulmonary edema
        E. Cardiopulmonary arrest

13. Hydrogen sulfide is 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. Cough
    c. Shortness of breath
    d. Rapid alternations in cognition or consciousness
    e. Bronchiole and lung hemorrhage/hemoptysis
    f. Non-cardiac pulmonary edema
    g. Hydrogen sulfide is known as the "knock down" gas because of near immediate and sudden loss of consciousness with high concentrations
    h. Asphyxia
    i. Cardiac arrest

14. 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 may cause:
        i. Altered mental status including agitation
        ii. Cyanosis
        iii. Vomiting
        iv. Dizziness
        v. Loss of consciousness

vi. Cardiopulmonary arrest

15. 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, unconsciousness)
    d. Loss of consciousness
    e. Cardiac dysrhythmias
    f. Cardiopulmonary arrest

III. PATIENT MANAGEMENT

A. 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 it's "knock down" effect of the initial unprotected responder and subsequence casualties associated with unprotected rescuers attempting to save the first downed responder.

2. Don appropriate PPE. Respiratory protection is critical.

3. Remove patient from the toxic environment.

4. Remove the patient's clothing, which may retain gases or decontaminate if liquid or solid contamination.

5. Decontaminate.

6. Flush irrigated effected/burned areas.

7. Assess ABCD and, if indicated, expose the patient and then cover the patient to assure retention of body heat.

8. Rapidly assess the patient's respiratory status, mental status, and oxygenation.

9. Assess patient vital signs (pulse, blood pressure, respiratory rate, neurologic status assessment), including temperature.

10. Establish intravenous access, if possible.

11. Place cardiac monitor and examine rhythm strip for arrhythmia potentials (consider 12-lead ECG).

12. Check blood glucose level.

13. Monitor pulse oximetry and ETCO2 for respiratory decompensation.

14. Perform carbon monoxide assessment with co-oximetry, if available.

15. Identify specific suspected agent, if possible.

16. Pertinent cardiovascular history or other prescribed medications for underlying disease.

17. Patient pertinent history.

18. Patient physical examination.
19. Continuous and ongoing patient reassessment is critical.

B. Treatment and Interventions

1. Assure a patent airway.

2. Administer oxygen and support breathing as indicated.

    a. Maintain the airway and assess for airway burns, stridor, or airway edema and, if indicated, perform airway management.
    b. Non-invasive ventilation techniques:
        i. Use CPAP for severe respiratory distress.
        ii. Use bag-valve-mask (BVM) ventilation in the setting of hypoventilation or respiratory failure.

3. Administer albuterol 2.5 mg nebulized for wheezing This can be repeated at this dose with for ongoing symptoms.

4. lpratropium 0.5 mg nebulized should be given in conjunction with albuterol.

5. Initiate IV access for infusion of normal saline and obtain blood glucose.

6. Fluid bolus (20 mL/kg) if evidence of hypoperfusion.

7. If the patient is experiencing significant pain, administer IV/IO analgesics:

    a. Fentanyl 1 mcg/kg IV or IO

8. Eye irrigation early.

9. Treat topical chemical burns (see Topical Chemical Burn Protocol).

10. In cases of severe respiratory irritation, in particular hydrogen sulfide, with altered mental status and no improvement with removal from the toxic environment, administer oxygen as appropriate with a target of achieving 94–98% saturation.

C. Patient Safety Considerations

1. Patients with exposure to highly soluble airway/respiratory irritants will self-extricate due to irritant warning properties such as smell, rapidity of onset of irritation, and other symptoms.

2. 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.

IV. NOTES/EDUCATIONAL PEARLS

A. 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. As patients may be off-gassing (particularly hydrogen sulfide and hydrogen cyanide) in the back of the transport vehicle, it is important to have adequate ventilation of the patient compartment.

3. Removal from the toxic environment, oxygen, general supportive therapy, bronchodilators, respiratory support, and rapid transport are core elements of care as there are no specific antidotes for any of these airway respiratory irritants except for heavy metals, which 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 very short exposure, will shut down and the exposed individual will not perceive the smell, yet the individual 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 move to a well-ventilated area and don a 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 very 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 a single use of inhalant of abuse.

    a. Some inhalants can cause cardiac arrest due to dysrhythmias from irritated myocardium.
    b. This syndrome most often is associated with abuse of butane, propane, and effects of the chemicals in the aerosols.

B. 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