Forensic & Toxicology Team

MED 433

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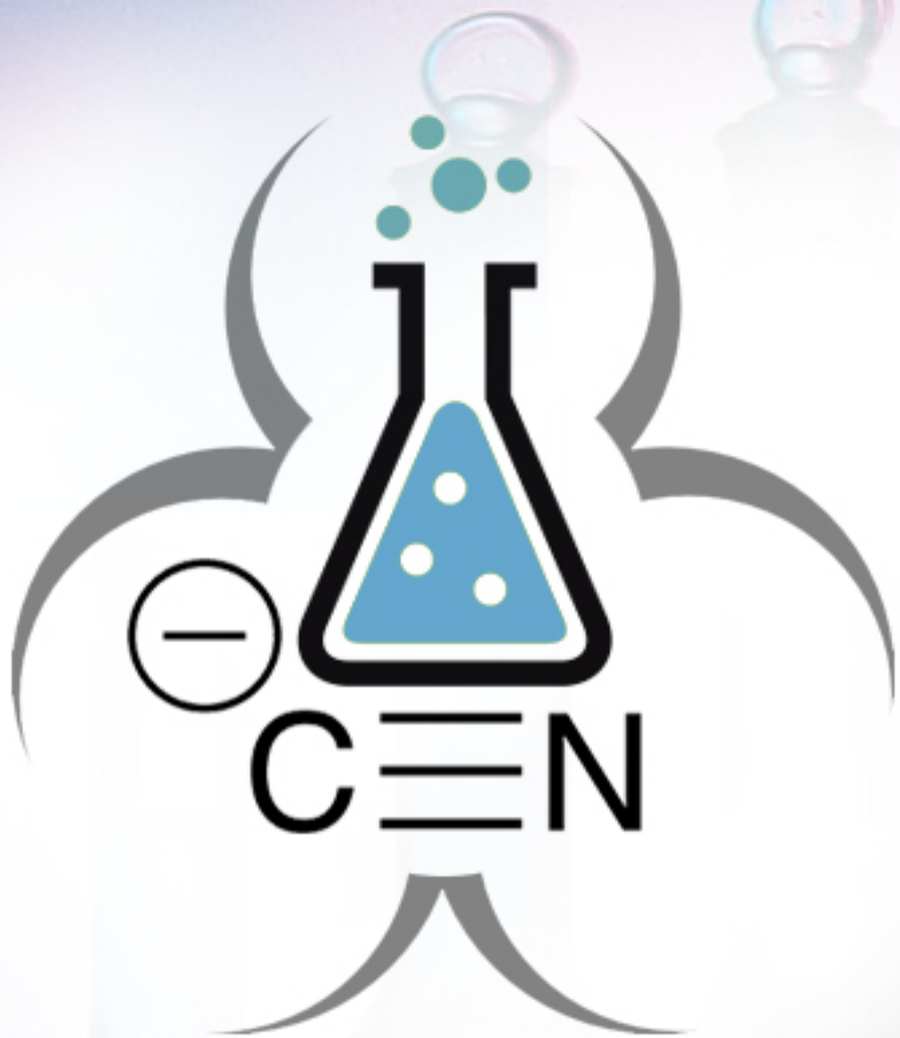


Lecture 8: **INHALED TOXINS**

Introduction

Airborne toxins typically produce local noxious effects on the airways and lungs.

#There are wide spectrum of possible toxic inhalants and identification of a specific inhalant is generally unnecessary because therapy is based on the clinical manifestations.

#Examples of Inhalational exposure:

Asbestos, Urban exposure to photochemical smog.

We don't admit asymptomatic or mild symptomatic patients.

INHALANT	SOURCE/USE	PREDOMINANT CLASS
Acrolein	Combustion	Irritant, highly soluble
Ammonia	Fertilizer, combustion	Irritant, highly soluble
Carbon dioxide	Fermentation, complete combustion, fire extinguisher	Simple asphyxiant; systemic effects
Carbon monoxide	Incomplete combustion, methylene chloride	Chemical asphyxiant
Chloramine	Mixed cleaning products (e.g., hypochlorite bleach and ammonia)	Irritant, highly soluble
Chlorine	Swimming pool disinfectant, cleaning products	Irritant, intermediate solubility
Chlorobenzylidenemalononitrile (CS)/chloroacetophenone (CN)	Tear gas (Mace)	Pharmacologic irritant
Hydrogen chloride	Tanning and electroplating industry	Irritant, highly soluble
Hydrogen cyanide	Combustion of plastics, acidification of cyanide salts	Chemical asphyxiant
Hydrogen fluoride	Hydrofluoric acid	Irritant, highly soluble; systemic effects
Hydrogen sulfide	Decaying organic matter, oil industry, mines, asphalt	Chemical asphyxiant; irritant, highly soluble
Methane	Natural gas, swamp gas	Simple asphyxiant
Methylbromide	Fumigant	Chemical asphyxiant
Nitrogen	Mines, scuba diving (nitrogen narcosis, decompression sickness)	Simple asphyxiant; systemic effects
Nitrous oxide	Inhalant of abuse, whipping cream, racing fuel booster	Simple asphyxiant
Noble gases (e.g., helium)	Industry, laboratories	Simple asphyxiant
Oxides of nitrogen	Silos, anesthetics, combustion	Irritant, intermediate solubility
Oxygen	Medical use, hyperbaric conditions	Irritant, free radical; systemic effects
Ozone	Electrostatic energy	Irritant, free radical
Phosgene	Combustion of chlorinated hydrocarbons	Irritant, poorly soluble
Phosphine	Hydration of aluminum or zinc phosphide (fumigants)	Chemical asphyxiant
Smoke (varying composition)	Combustion	Variable, but may include all classes
Sulfur dioxide	Photochemical smog (fossil fuels)	Irritant, highly soluble

FIRST : SIMPLE ASPHYXIANTS

#Most simple asphyxiates are inert and produce toxicity only by displacing oxygen and lowering the fraction of inspired oxygen **(FiO2).**

#Exposed patients remain asymptomatic if the Fio2is normal.

#Normal o2 concentration in the air is 21%.

CO2 and N2 : constituents of air produce narcosis at elevated levels.

#If the FiO2 fall to be from 21% to 15% patients will develop:

1- Autonomic stimulation: (e.g., tachycardia, tachypnea, and dyspnea)

2- Cerebral hypoxia: (e.g., ataxia, dizziness, incoordination, and confusion).

#If FiO2 falls below 10% :

patient will have Cerebral Edema and Lethargy.

#If FiO2 falls below 6%:

It is Fatal.

#The vast majority of simple asphyxiations are workplace related and usually occur :

- During the use of liquefied gas.
- While breathing through airline respirators .
- While working in confined spaces.

#Deaths from the inhalation of automotive exhaust result from simple asphyxiation "not carbon monoxide poisoning".

#asphyxiation is the most fatal type of inhaled toxins.

FIRST : SIMPLE ASPHYXIANTS

Diagnostic Strategies and Differential Considerations:

#Consistent history

#Scene investigation(CSI): will yield the causative agent ;by a trained and suitably outfitted team. #The presenting complaints :nonspecific (e.g., dizziness, syncope, and dyspnea)

#The differential diagnosis is extensive.

(Determination of the exact nature of the gas is of limited clinical value but It may have important public health implications).

Management and Disposition:

#Removal from exposure

#supportive care:

Administration of supplemental oxygen.

Neurologic injury or cardiorespiratory arrest should be managed with standard resuscitation protocols.

#Mildly-poisoning patients:

- recover after removal from the exposure
- observed briefly and discharged.

*Removal from exposure terminates the hypoxia and results in clinical improvement.

*Most patients present with resolving symptoms.

***Failure to improve** suggests complications of ischemia (e.g., seizures, coma, and cardiac arrest) That is poor prognosis.

Risk for complications of hypoxia:

Elderly # Pediatrics # Coma

Seizures # Cardiac # COPD

Prolonged exposure

Second: PULMONARY IRRITANTS

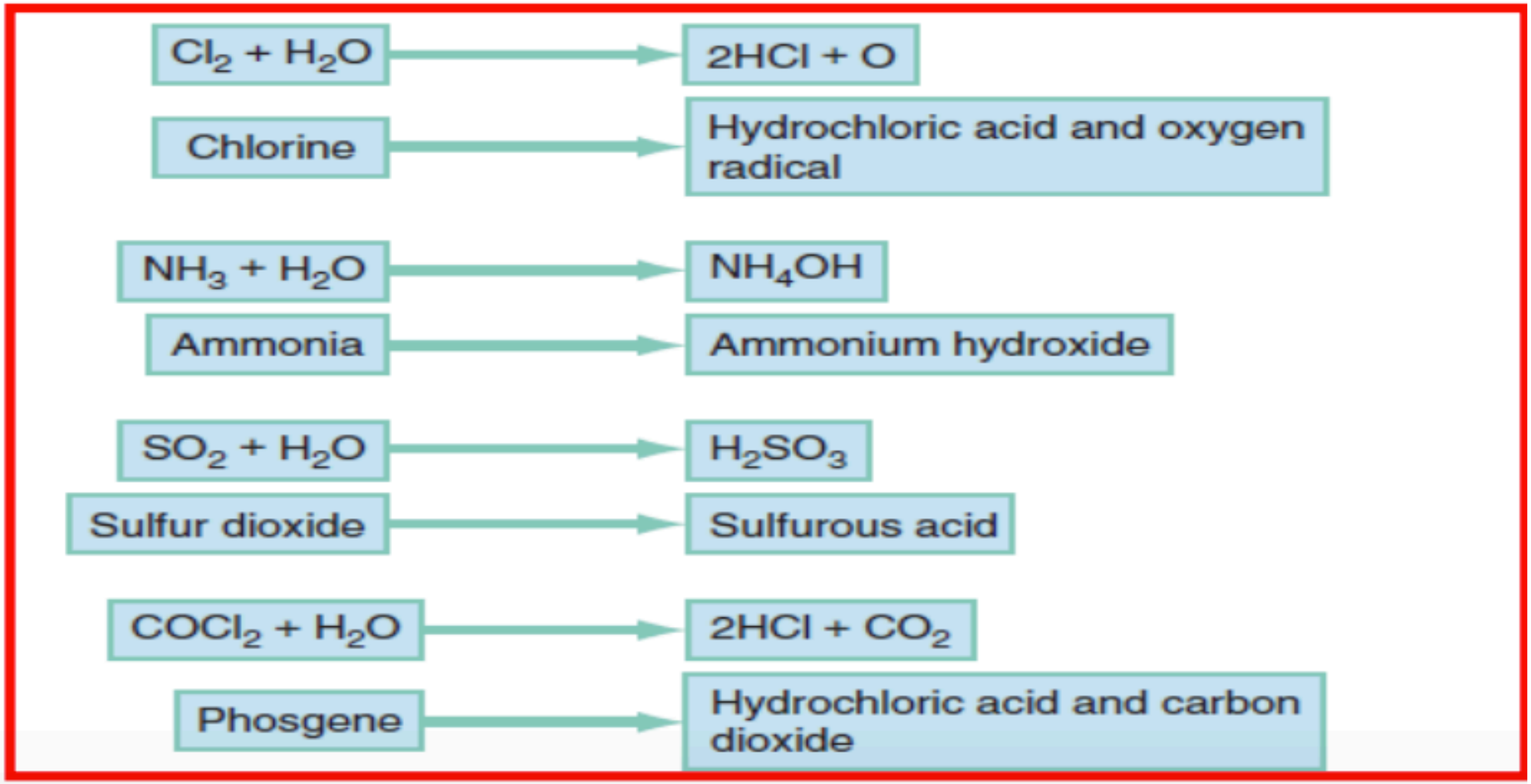
Pulmonary irritant gases:
large group of agents that produce a common toxicological syndrome when inhaled in moderate concentrations.

These gases when they go to the lung and dissolve because they are water soluble

Principle of the disease

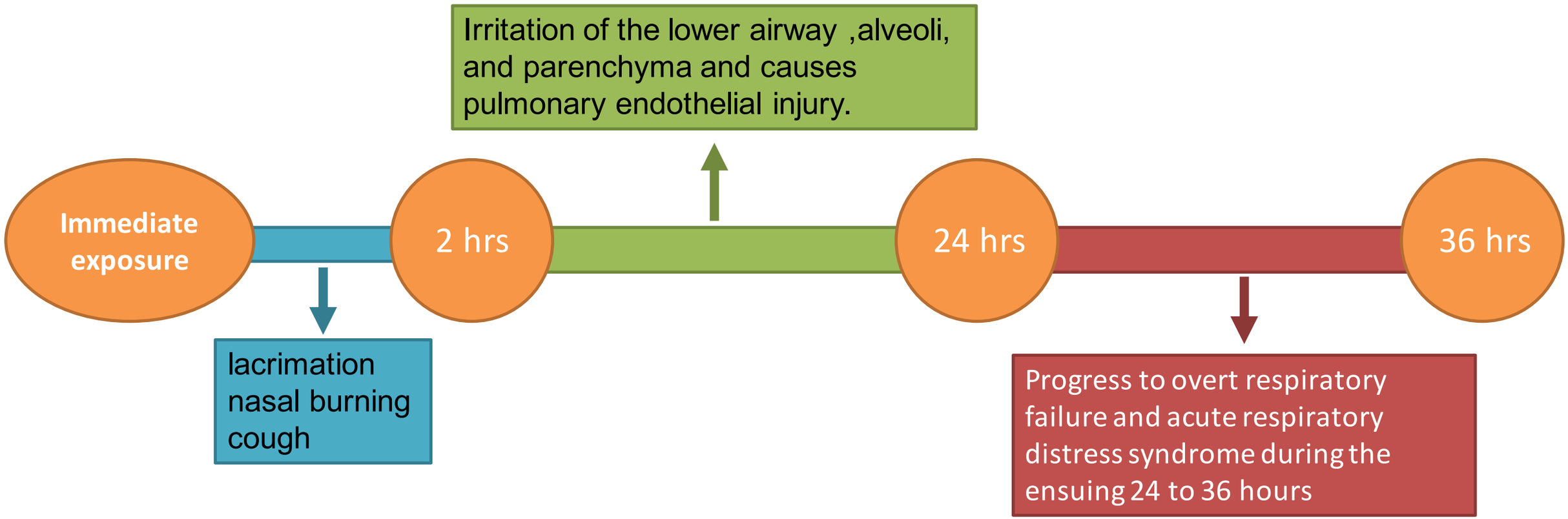
1. Irritant gases dissolve in the **respiratory tract mucus**
2. Alter the air-lung interface by **inflammatory response**.
3. When dissolved, produce an **acid or alkaline product**.
4. generate oxygen-derived free radicals that produce direct **cellular toxicity**

Simple reactions of pulmonary irritants reacting with water in the lung



*Each one combines with water to give a new toxic substance.

Clinical features



Massive or prolonged exposure:

*laryngeal edema

*Laryngospasm

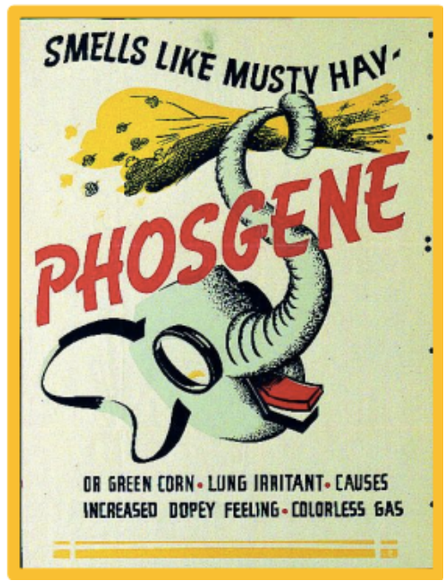
*bronchospasm

*Acute Lung Injury (ALI) "noncardiogenic pulmonary edema"

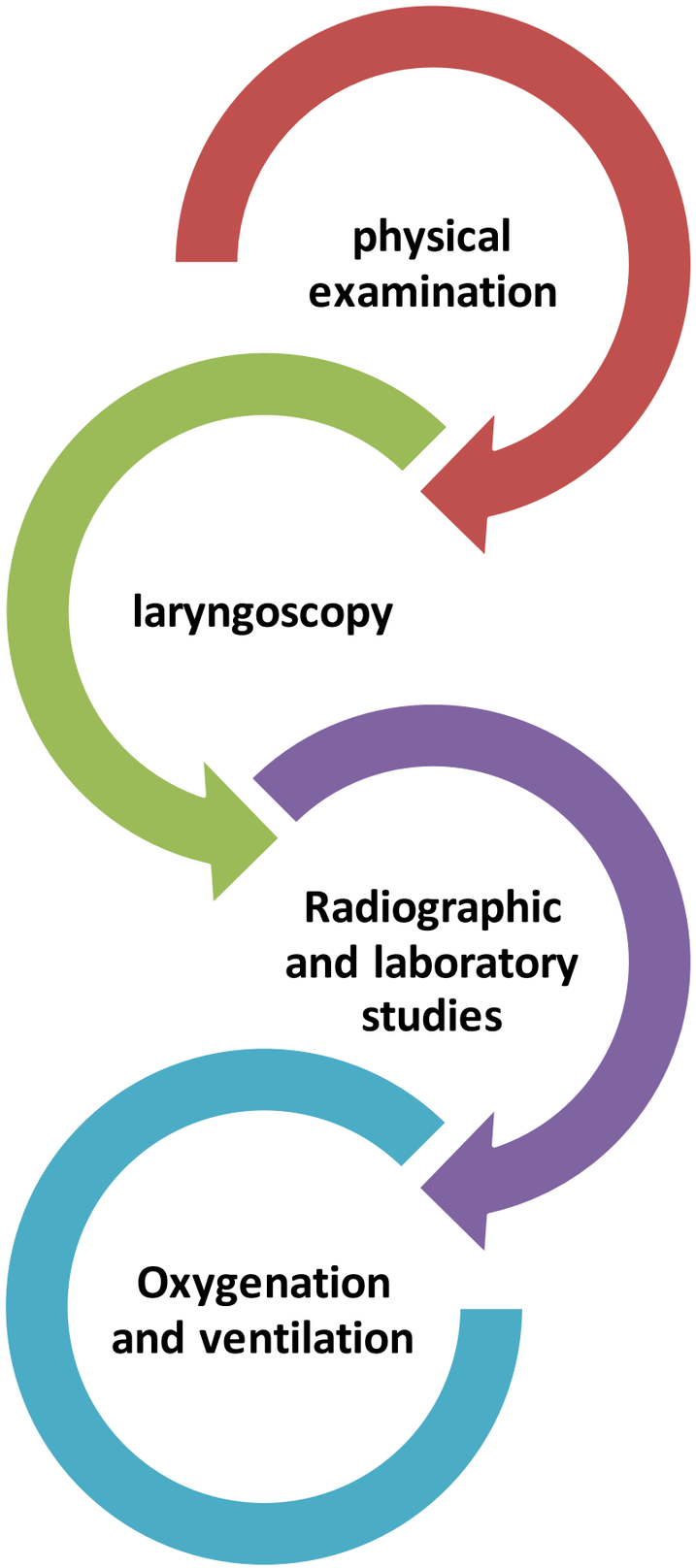
Poorly water-soluble gases

#do not readily irritate the mucous membranes !!
#some have pleasant odors (e.g., phosgene's odor is similar to that of hay).

#Since there are no immediate symptoms, prolonged breathing in the toxic environment allows the gas to reach the alveoli.



Diagnostic Strategies and Differential Consideration



normal oropharyngeal or laryngeal evaluation may **NOT** exclude subsequent deterioration.

NOT useful

Assessed by:

Serial chest auscultation, pulse oximetry ,chest radiography ,ABGs

***No clinical tests can identify the specific irritant**

identification is not generally necessary

Management

Presentation	Management	Notes
Signs of upper airway dysfunction (e.g., hoarseness and stridor)	Visualization of the larynx and immediate airway stabilization	potential rapidity of airway deterioration)
Bronchospasm	Inhaled **beta2-adrenergic agonists** (Salbutamol) **Ipratropium bromide** (Atrovent).	No clearindication for corticosteroids
Chlorine or hydrogen chloride gas	Nebulized 2% sodium bicarbonate solution symptomatic relief	Patients require extensive discharge instructions for signs and symptoms of pulmonary irritation.
Diagnosis of ALI (ARDS)	Aggressive **supportive care** • Advanced ventilation modes in ICU. • Exogenous surfactant • Nitric Oxide (NO) ventilation	ARDS = (Acute respiratory distress syndrome)

Third: **Smoke inhalation**

1)Irritant injury: heated particulate matter and adsorbed toxins injure normal mucosa, similar to other irritant gases.

2)Carbon monoxide and Cyanide are systemic toxins.

- **At Temp° : 350° C and 500° C**,

Air has a low heat capacity. rarely produces lower airway damage.

Heat capacities of steam : (approximately 4000 times that of air) plus heated soot suspended in air (i.e., smoke) can transfer heat and cause injury **deep within the respiratory tract.**

The nature of the fuel determines the composition of its smoke.

- **Irritant toxins** produced by the fire are adsorbed onto **carbonaceous particles** that deposit in the airways.
- The irritant substances **damage the mucosa** through mechanisms similar to those of the irritant gases, including **generation of acids and free radical formation**.

Clinical Features

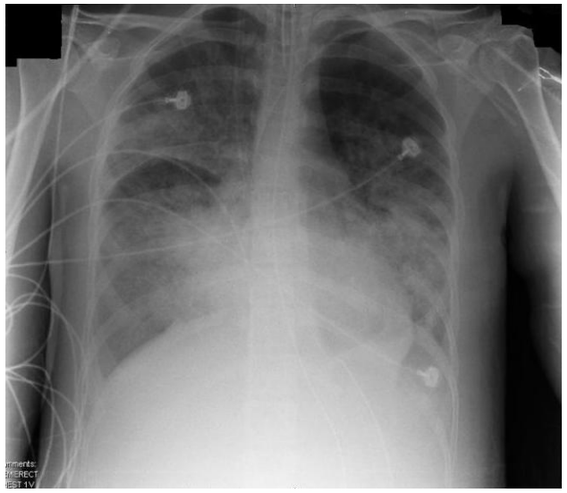
Morbidity and mortality : respiratory tract damage. Thermal and irritant-induced laryngeal injury

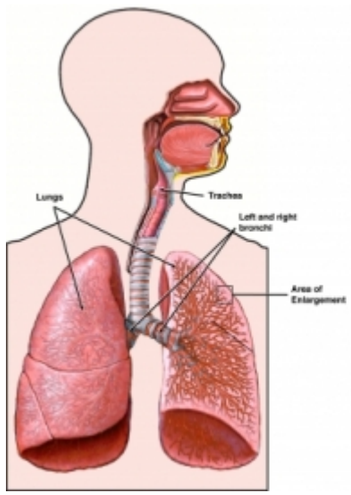
***Cough, Stridor, Bronchospasm**

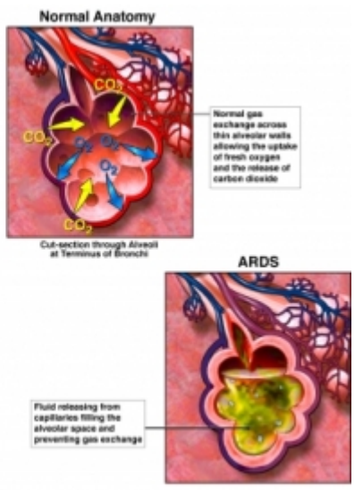
Then : Adult Respiratory Distress Syndrome (**Maybe delayed).**

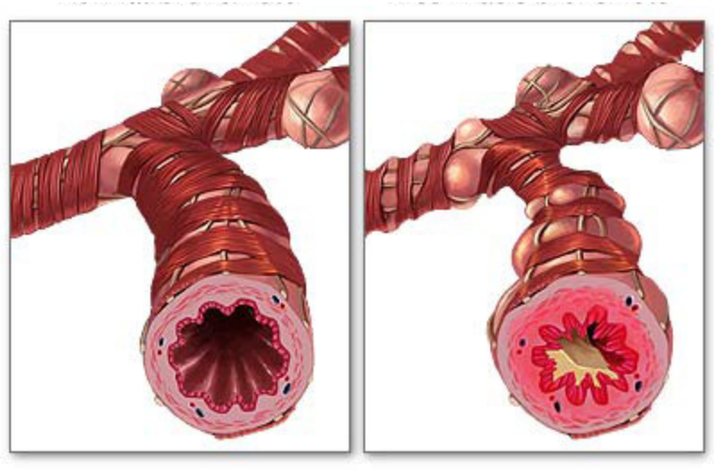
Latency period: variable and dependent on the degree and nature of the exposure.

• Singed nasal hairs and soot in the sputum suggest substantial exposure but are neither sensitive nor specific









Carbon monoxide (CO) and Cyanide

Must be routinely considered in these patients.

Exposure to filtered or distant smoke

(e.g., in a different room)

or (e.g., engine exhaust)

inhale predominantly CO, cyanide, and metabolic poisons and do not sustain smoke exposure.

Diagnostic Strategies and Differential Considerations

- Early death result from :

1.asphyxia

2.airway compromise

3.metabolic poisoning (e.g., CO).

Management

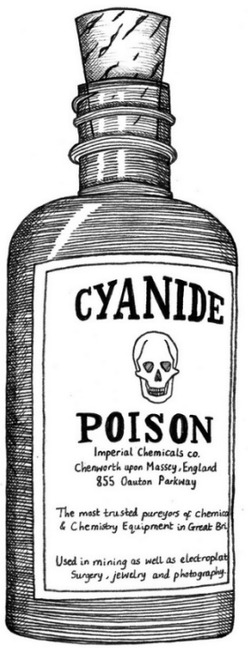
Cyanide :

Must suspect : when? **Metabolic acidosis**! (with **serum lactate level greater than 10 mmol/L**),
suggests concomitant cyanide poisoning.
Oxygenation should be assessed by co-oximetry because ABG analysis and pulse oximetry may be inaccurate in CO-poisoned patients.

When you should admit the patient?

- Worrisome clinical findings (e.g., **hoarseness** and respiratory distress) and those with
- Identifiers of **substantial exposure** (e.g., closed-space exposure)
- Carbonaceous sputum

- **Critical Care Unit or Burn Center**

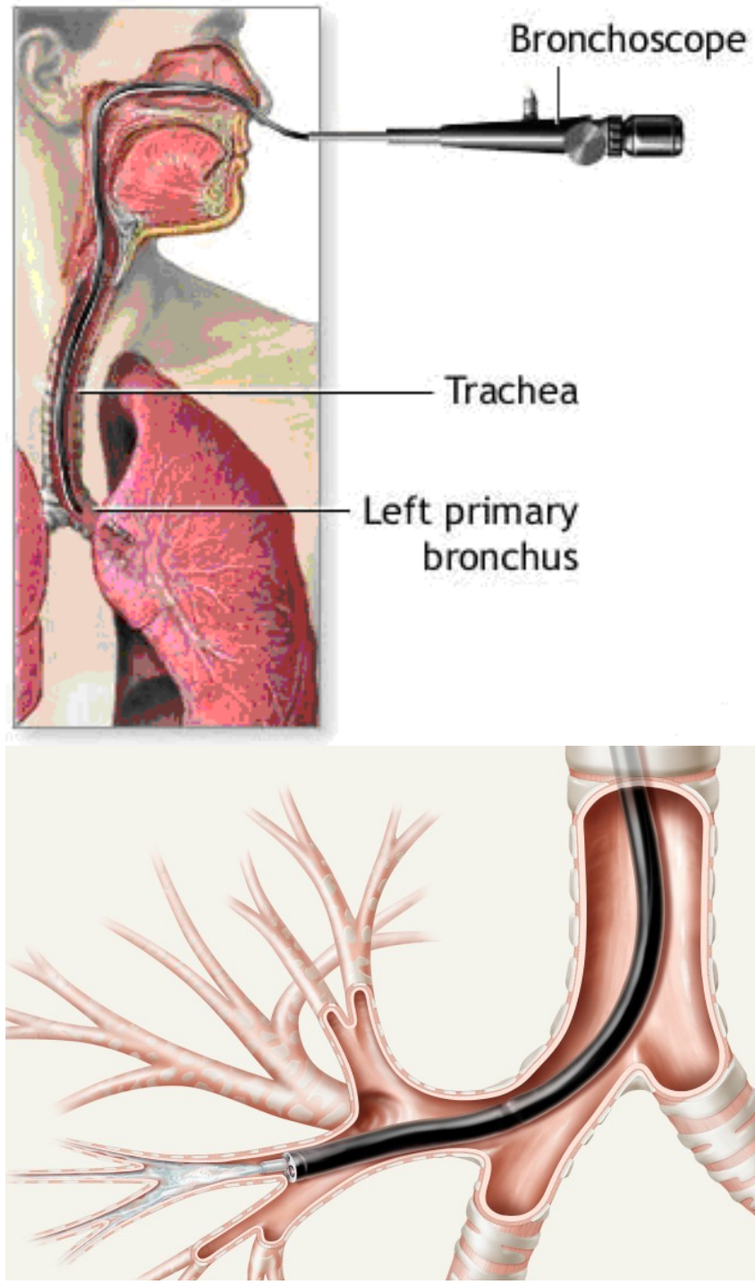


Management : Supportive

Bronchoscopy with bronchoalveolar lavage is frequently recommended to clear debris and toxins from the distal airways.
Corticosteroids whether inhaled or systemic, are not indicated and are **potentially harmful**
Antibiotics should be used only in patients with **suspected infection**.

Management of victims of smoke inhalation and irritant inhalational injuries : **similar.**

- **Rapid assessment of the airway and early intubation is critical!**
- **Inhaled beta-adrenergic agonists**
- **Optimal supportive care**
- **maintenance of adequate oxygenation (e.g., suctioning and pulmonary toilet)** "**most important aspects of care**"



Q1: Therapy of toxic inhalant depend on which of the following ?

A- How much the person inhale.
B- Clinical manifestation.
C- Chronic illnesses.
D- None of the above.

Q2: patient came to the ER complaining of tachycardia, tachypnea, dyspnea, cerebral hypoxia and cerebral edema FiO2 considered as ?

A- Below 10%
B- From 21% to 15%
C- Normal
D- Below 6%

Q3: when a patient came to hospital with laryngeal edema , bronchospasm and ALI known to have ?

A- Immediate exposure
B- Passive exposure
C- Massive exposure
D- Cardiogenic exposure

Q4: Which of the following is diagnostic of cyanide toxicity:

A- Odor of sweet almonds.
B- Positive history +profound hypocalcaemia
c- Positive history +profound metabolic acidosis
D- None of the above.

Q5: Which of the following is chemical asphyxiant?

A- Acrolein
B- Hydrogen fluoride
C- Hydrogen sulfide
D-Phosgene

Q6:Which of the following react with water in the lung to form hydrochloric acid and O2 radicals?

A- Phosgene
B- Chlorine
C- Ammonia
D- Sulfur dioxide

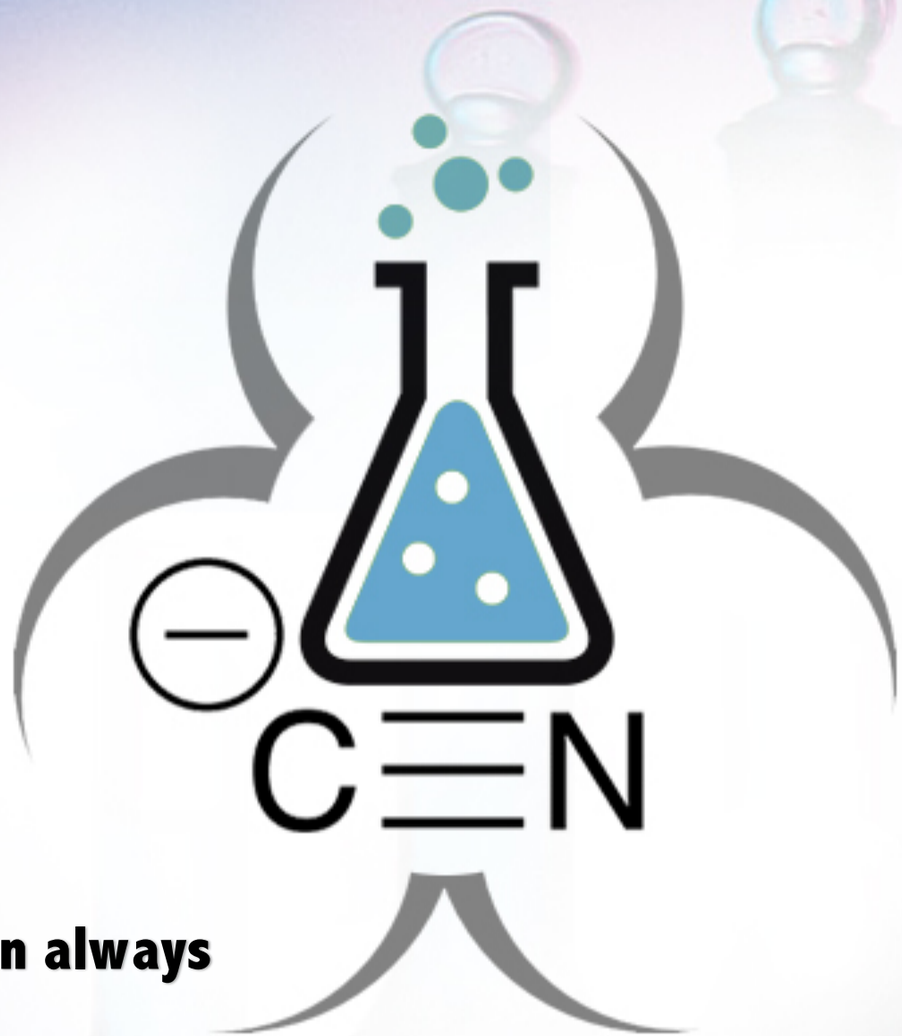
Q7: Which of the following gas poisons was used as a war weapon?

A- Carbon monoxide
B- Carbon dioxide
C- Mustard gas
D- Hydrofluoric acid

Q8: How simple asphyxiants produce toxicity?

A- Interfering with the electron transport chain in the mitochondria
B- Displacing O2 and lowering FiO2
C- Lowering Po2 and increasing Pco2
D- Production of free radicals

Answers: 1.B 2. A 3. C 4. C
5. C 6. B 7. C 8. B



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