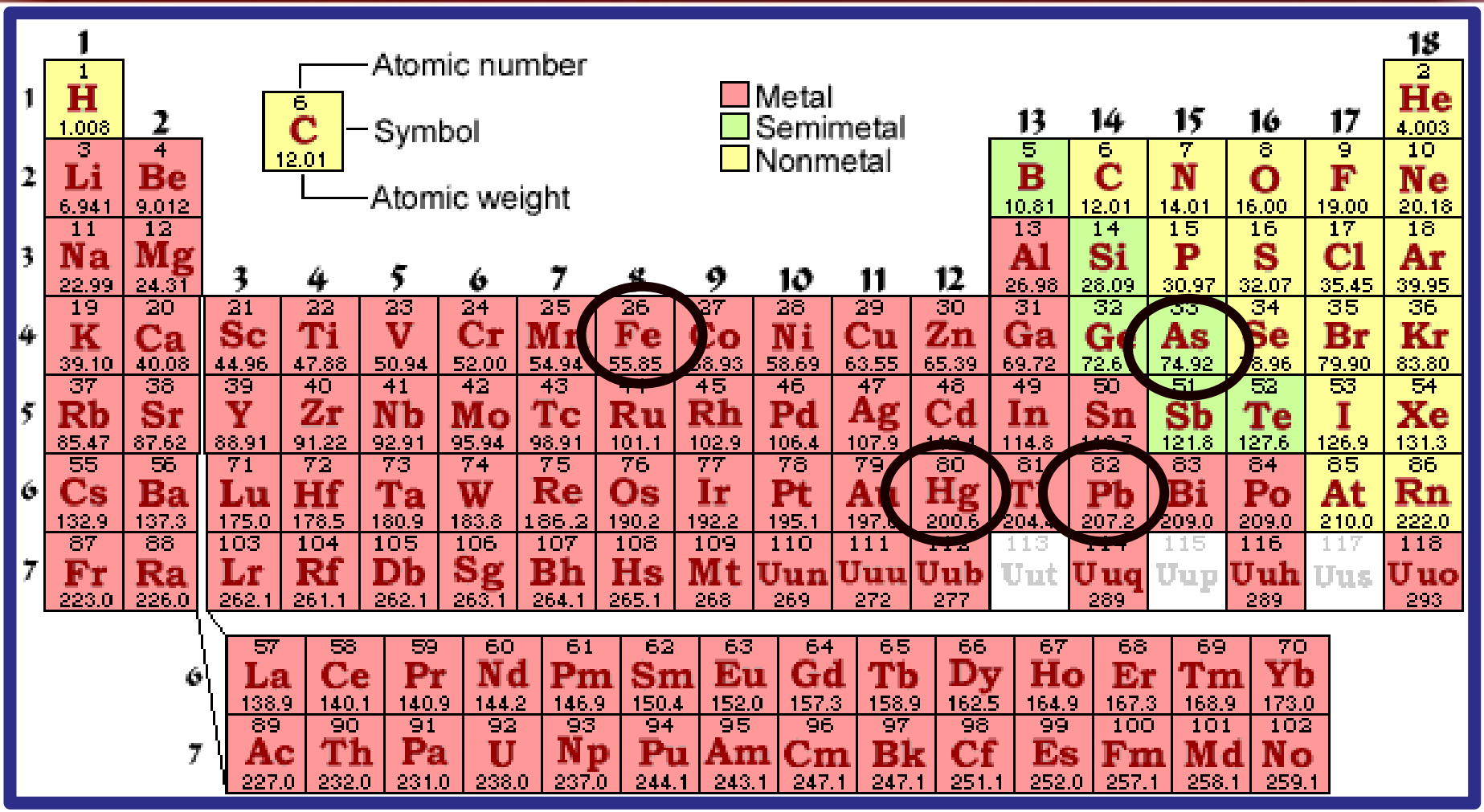
HEAVY METALS : Review



INHALED TOXINS

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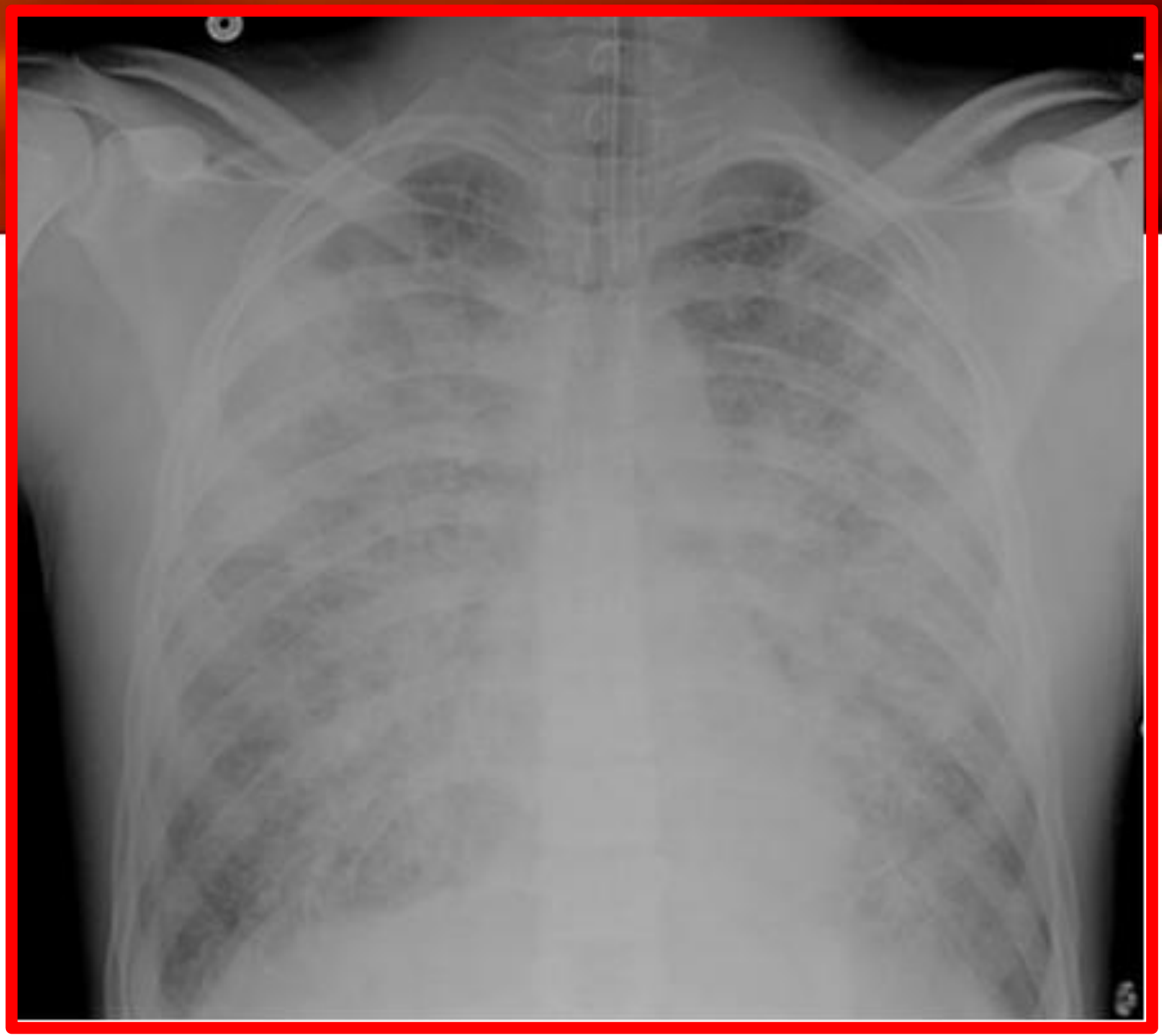
OF FIRE
DDLETOWN



Introduction

- Airborne toxins typically produce local noxious effects on the airways and lungs.
- Examples of Inhalational exposure:

Asbestos

Urban exposure to photochemical smog

Introduction

- There are wide spectrum of possible toxic inhalants

- identification of a specific inhalant is generally unnecessary
- Therapy is based on the clinical manifestations.

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

- 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”.

Principles of Disease

- Most simple asphyxiants are **inert** and produce toxicity only by displacing oxygen and lowering the fraction of inspired oxygen (FiO_2)
- Exposed patients remain asymptomatic if the Fio_2 is normal.

- **CO_2 and N_2** :

constituents of air

produce narcosis at elevated levels

Clinical Features

Acute effects occur within minutes of onset of asphyxia:

manifestations of hypoxia

A fall in the FiO_2 from 21% to 15%

Autonomic stimulation

(e.g., tachycardia, tachypnea, and dyspnea)

Cerebral hypoxia

(e.g., ataxia, dizziness, incoordination, and confusion).

Stimulator: hypoxemia or hypercarbia ?

Clinical Features

If FiO_2 falls below 10% :

Cerebral Edema and Lethargy.

FiO_2 below 6%: is Fatal !

*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.

Diagnostic Strategies and Differential Considerations

Consistent history

Spectrum of complaints

Rapid resolution on removal from exposure

Minimally symptomatic or asymptomatic patients do not require any tests.

Scene investigation(CSI): will yield the causative agent ;by a trained and suitably outfitted team.

Diagnostic Strategies and Differential Considerations

Determination of the exact nature of the gas is of limited clinical value

It may have important public health implications.

The presenting complaints :nonspecific

(e.g., dizziness, syncope, and dyspnea)

The differential diagnosis is extensive.

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.

Management and Disposition

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.

Principles of Disease

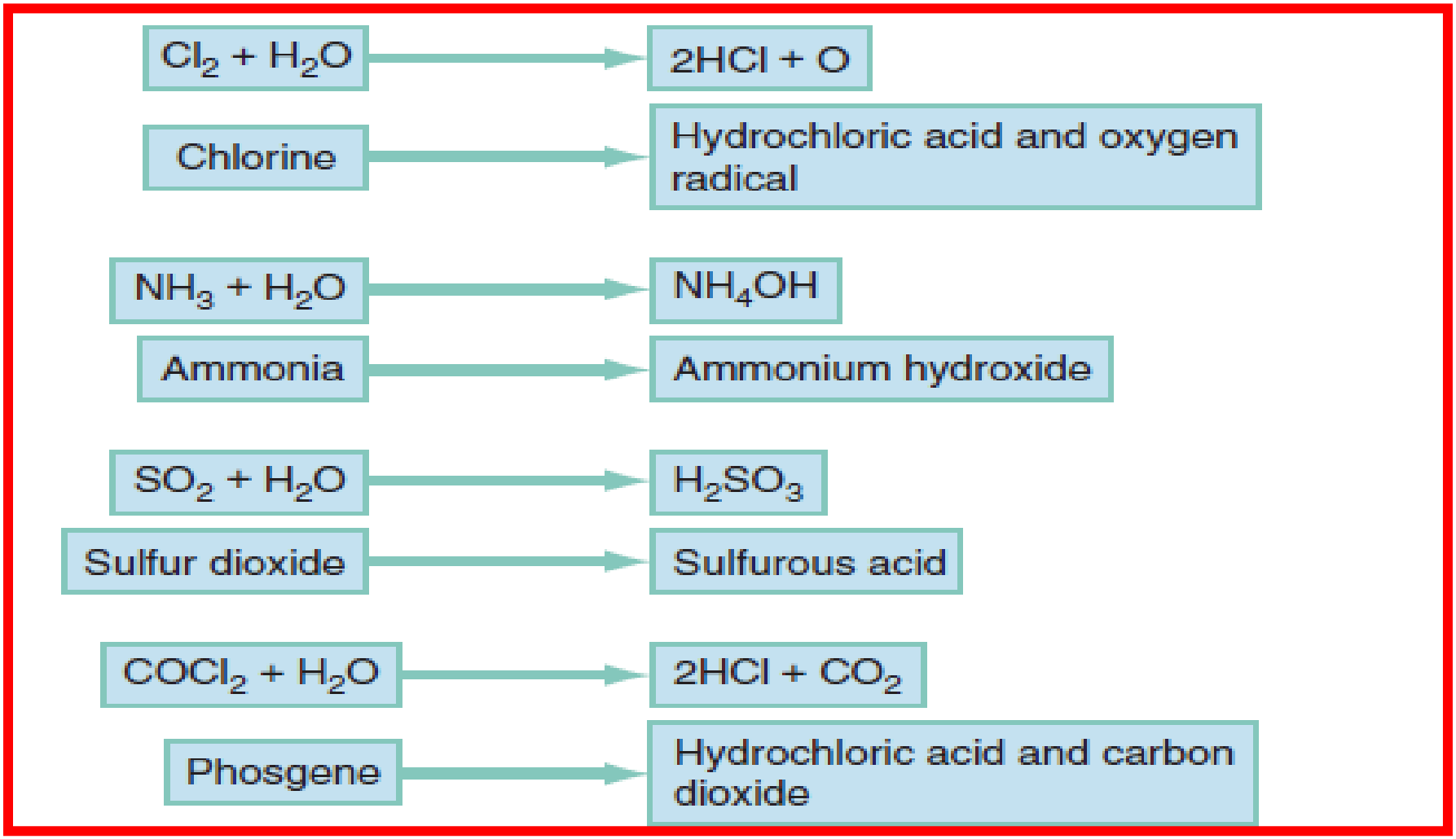
1)Irritant gases dissolve in the respiratory tract mucus.

2)Alter the air-lung interface by inflammatory response.

3)When dissolved, produce an acid or alkaline product.

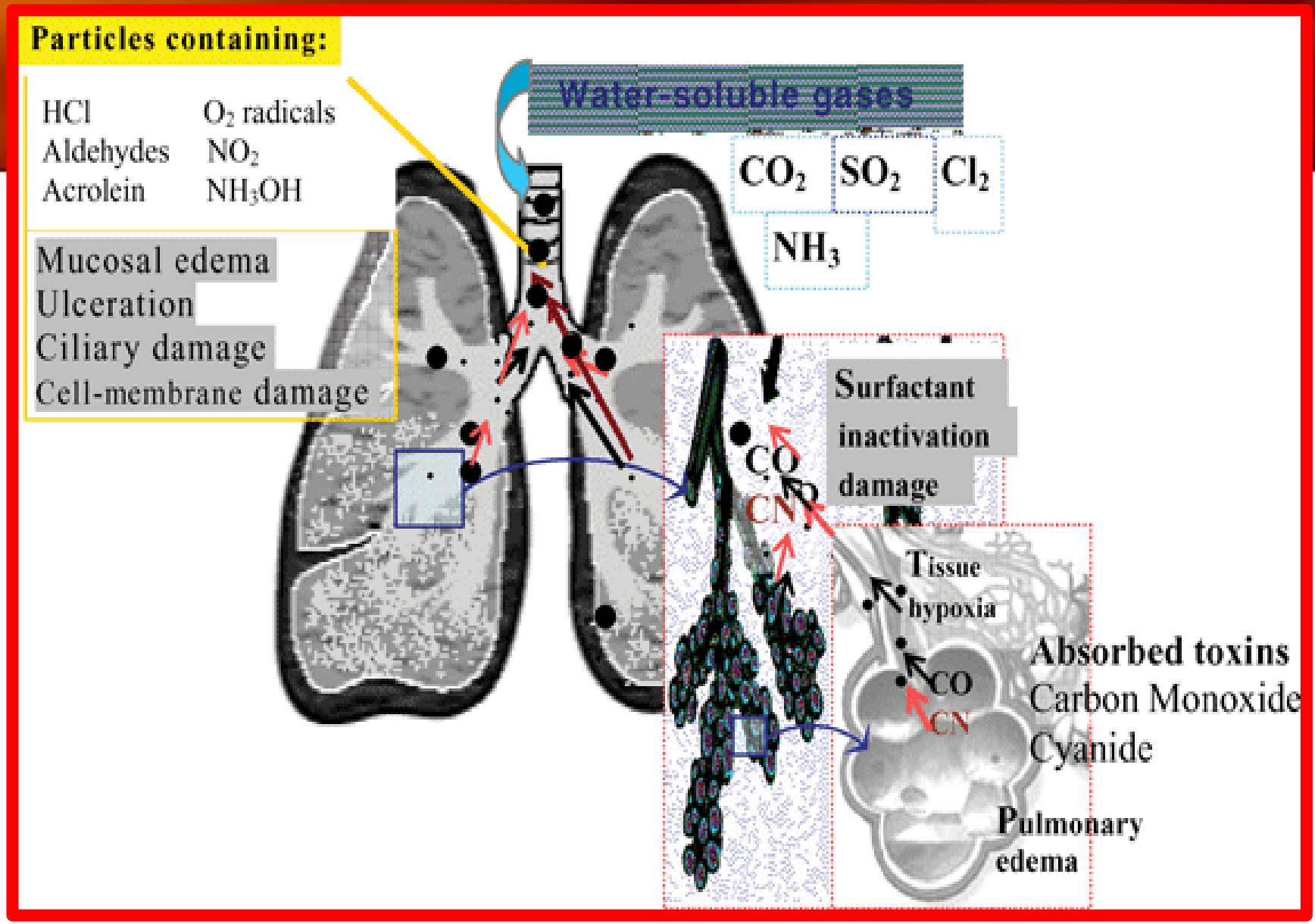
4)generate oxygen-derived free radicals that produce direct cellular toxicity.

Sample reactions of pulmonary irritants reacting with water in the lung

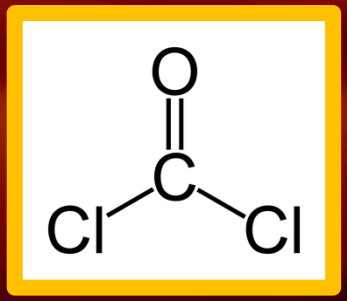


Clinical Features

- Exposure results in immediate irritation,
  - lacrimation
  - nasal burning
  - cough
- massive or prolonged exposure:
  - laryngeal edema
  - laryngospasm
  - bronchospasm
  - Acute Lung Injury (ALI)
    - “noncardiogenic pulmonary edema”


Particles containing:
HCl
Aldehydes
Acrolein
O_2 radicals
NO_2
NH_3OH
Mucosal edema
Ulceration
Ciliary damage
Cell-membrane damage
Water-soluble gases
CO_2
SO_2
Cl_2
NH_3
Surfactant inactivation damage
CO
CN
Tissue hypoxia
CO
CN
Absorbed toxins
Carbon Monoxide
Cyanide
Pulmonary edema

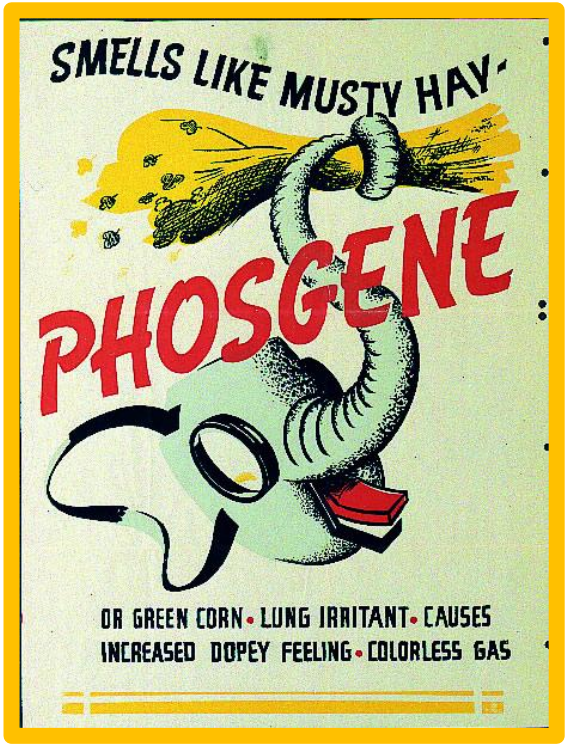
Clinical Features



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.



Weapons of War - Poison Gas (WMD) : WWI
Chlorine – Phosgene – Mustard gas

- The German army ended the war as the heaviest user of gas.
- It is suggested that German use reached 68,000 tons
- French utilised 36,000 tons
- British 25,000 tons

Clinical Features

- After 2-24 Hours:

Irritation of the lower airway, alveoli, and parenchyma and causes pulmonary endothelial injury.

- Initial symptoms consistent with ALI may be mild
- Progress to overt respiratory failure and acute respiratory distress syndrome during the ensuing 24 to 36 hours.

Diagnostic Strategies and Differential Consideration

- The evaluation of upper airway :

  physical examination

  laryngoscopy
- normal oropharyngeal or laryngeal evaluation may NOT exclude subsequent deterioration.
- Radiographic and laboratory studies: not useful.

Diagnostic Strategies and Differential Consideration

Oxygenation and ventilation are assessed by

- serial chest auscultation
- pulse oximetry
- chest radiography
- ABGs

No clinical tests can identify the specific irritant

identification is not generally necessary

Management

- Signs of upper airway dysfunction

(e.g., hoarseness and stridor) :

visualization of the larynx and

immediate airway stabilization

(potential rapidity of airway deterioration)

Management

- Bronchospasm generally responds to

_Inhaled beta2-adrenergic agonists(Salbutmol)

_Ipratropium bromide(Atrovent).

No clearindication for corticosteroids

Management

Nebulized 2% sodium bicarbonate solution

chlorine or hydrogen chloride gas

symptomatic relief

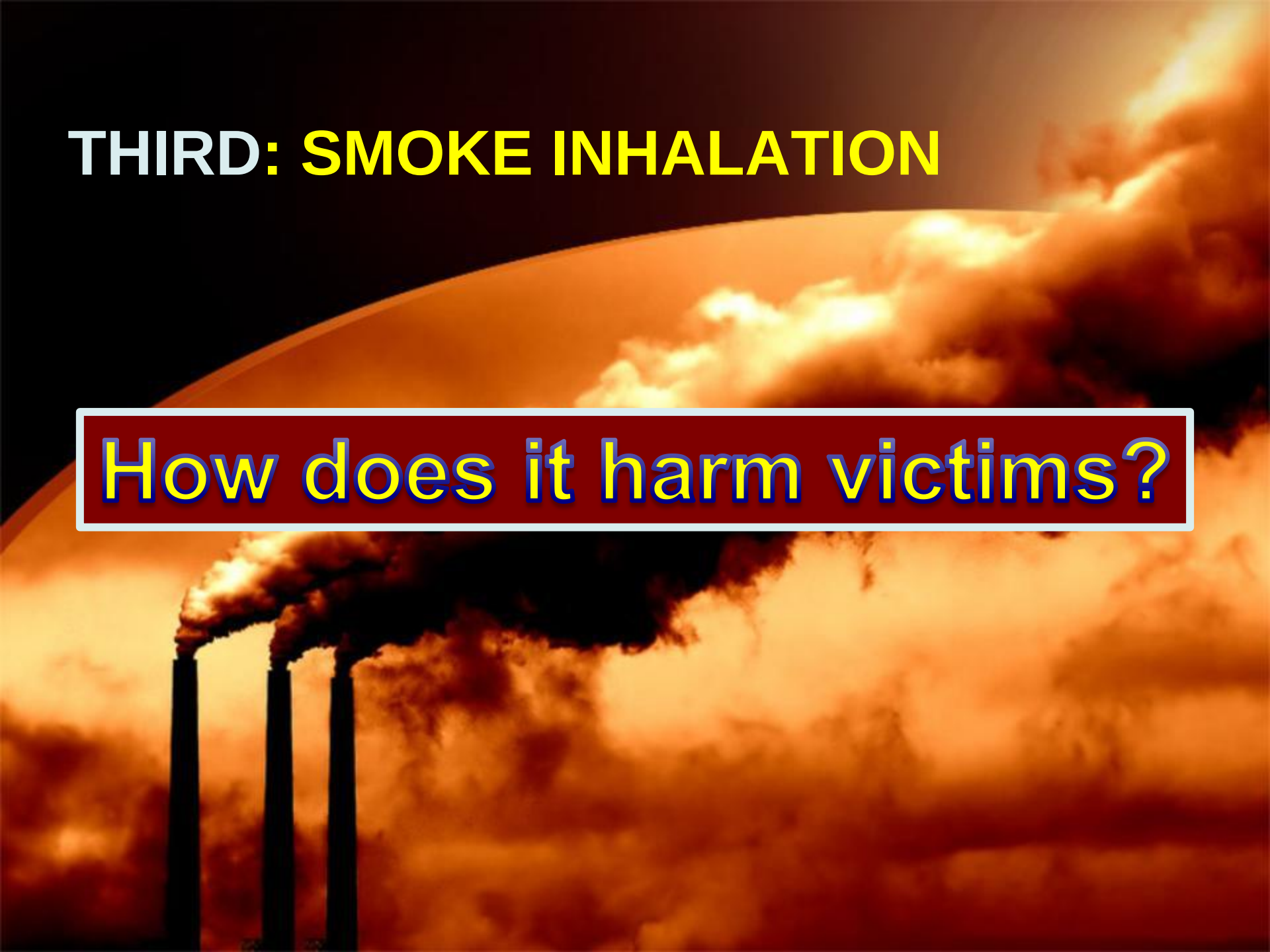
Patients require extensive discharge instructions for signs and symptoms of pulmonary irritation.

Management

- Diagnosis of ALI (ARDS)

(acute respiratory distress syndrome)

- aggressive supportive care
- Advanced ventilation modes in ICU.
- Exogenous surfactant
- nitric oxide (NO) ventilation


THIRD: SMOKE INHALATION
How does it harm victims?

SMOKE INHALATION
Principals :

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

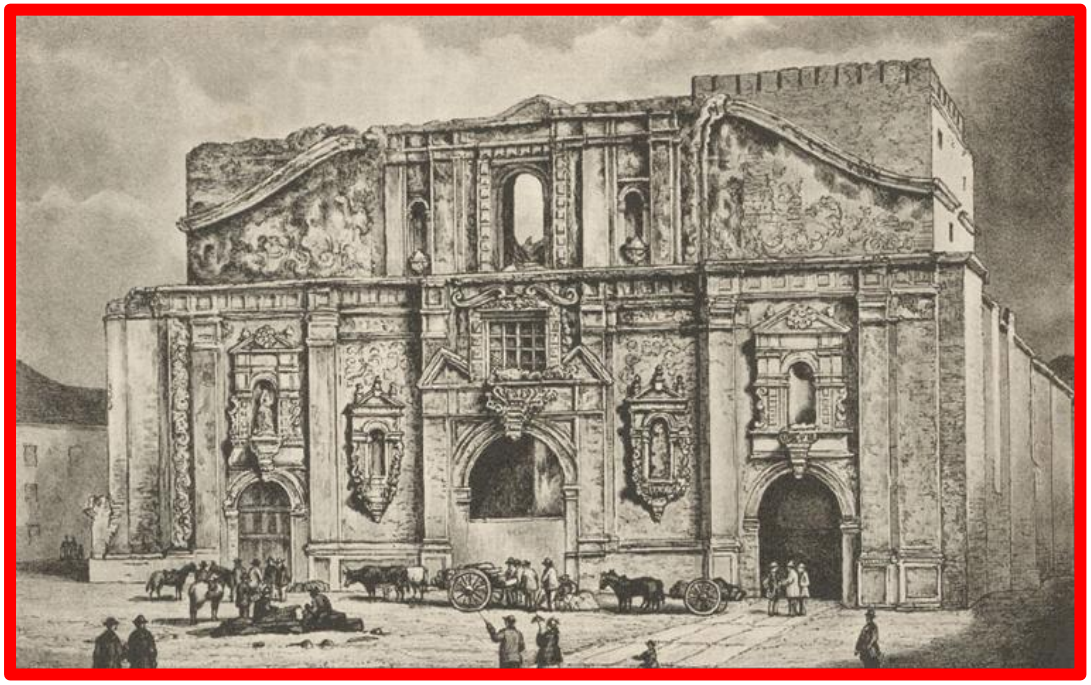
2)Carbon monoxide and Cyanide are systemic toxins.

Worst Fire Accidents !

Deaths ⬍	Incident
2000+	Church of the Company Fire (Santiago, Chile, 8 December 1863)
1995	Theater fire (Kamli, China, 1893)[92]
1670	Theater fire (Canton, China, 25 May 1845)
900	Theater fire (Shanghai, China, June 1871)
800	Lehman Theater fire (St. Petersburg, Russia, 1836)
667	Cinema fire (Xinjiang, China, 1977)
658	Antoung Movie Theater fire (China, 13 February 1937)
602	Iroquois Theater Fire (Chicago, Illinois, 30 December 1903)
600	Theater fire (Tientsin, China, 20 May 1872)
540	Dabwali tent fire (Haryana, India, 23 December 1995)

Church of the Company Fire (1863)

- Santiago , Chile
- More than 2000 :Died



Principles of Disease

- **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.

Principles of Disease

- 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 , Bronhospasm

Then : ARDS

***Maybe delayed !**

Principles of Disease

- 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

Principles of Disease

- **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.

Management : Admission

- 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

Burn Intensive Care Unit

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 : Supportive

- 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))

THANKS

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