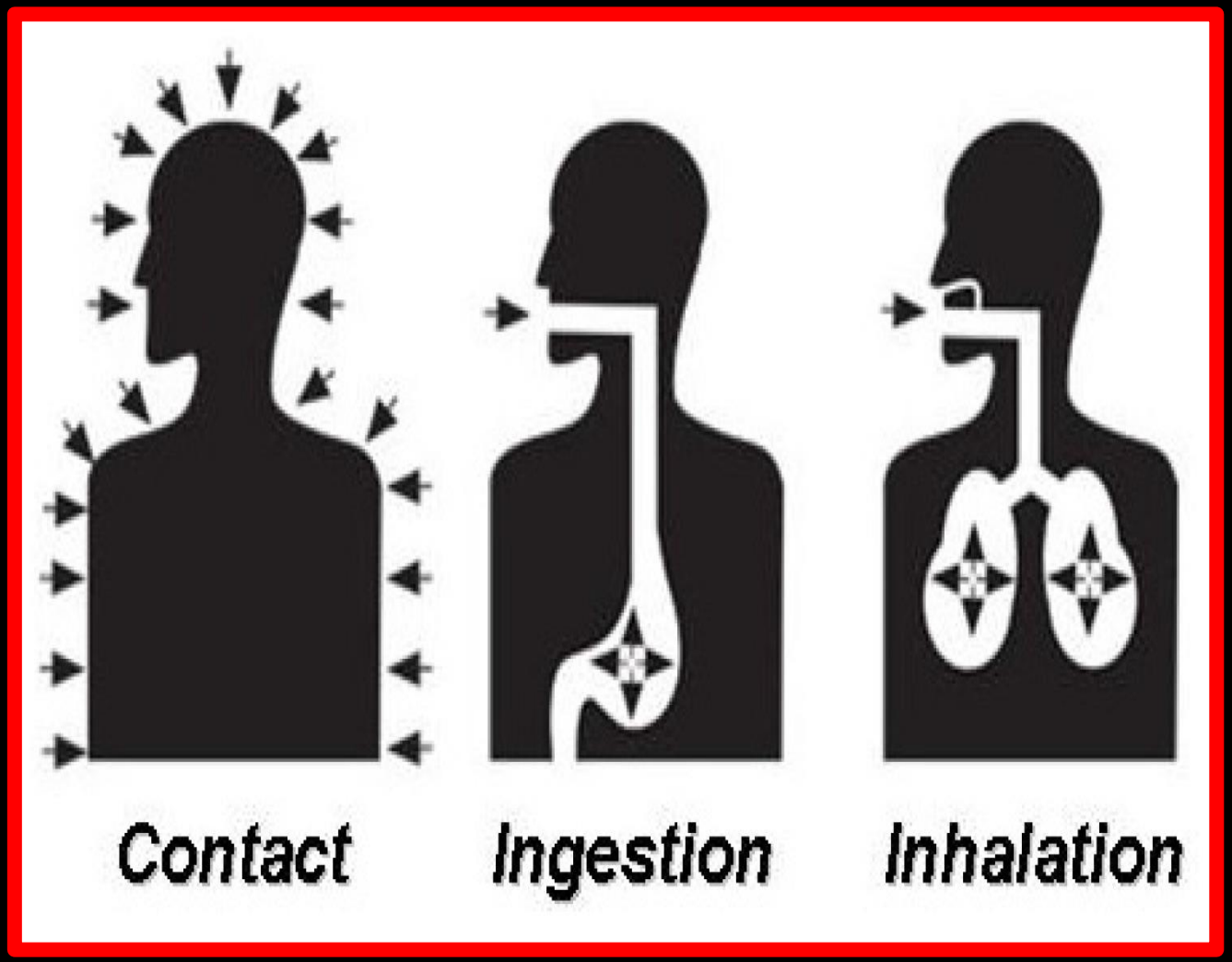
INHALation
toxicology

Dr. Tawfiq Almez

MBBS FRCPC (CCM


Contact
Ingestion
Inhalation

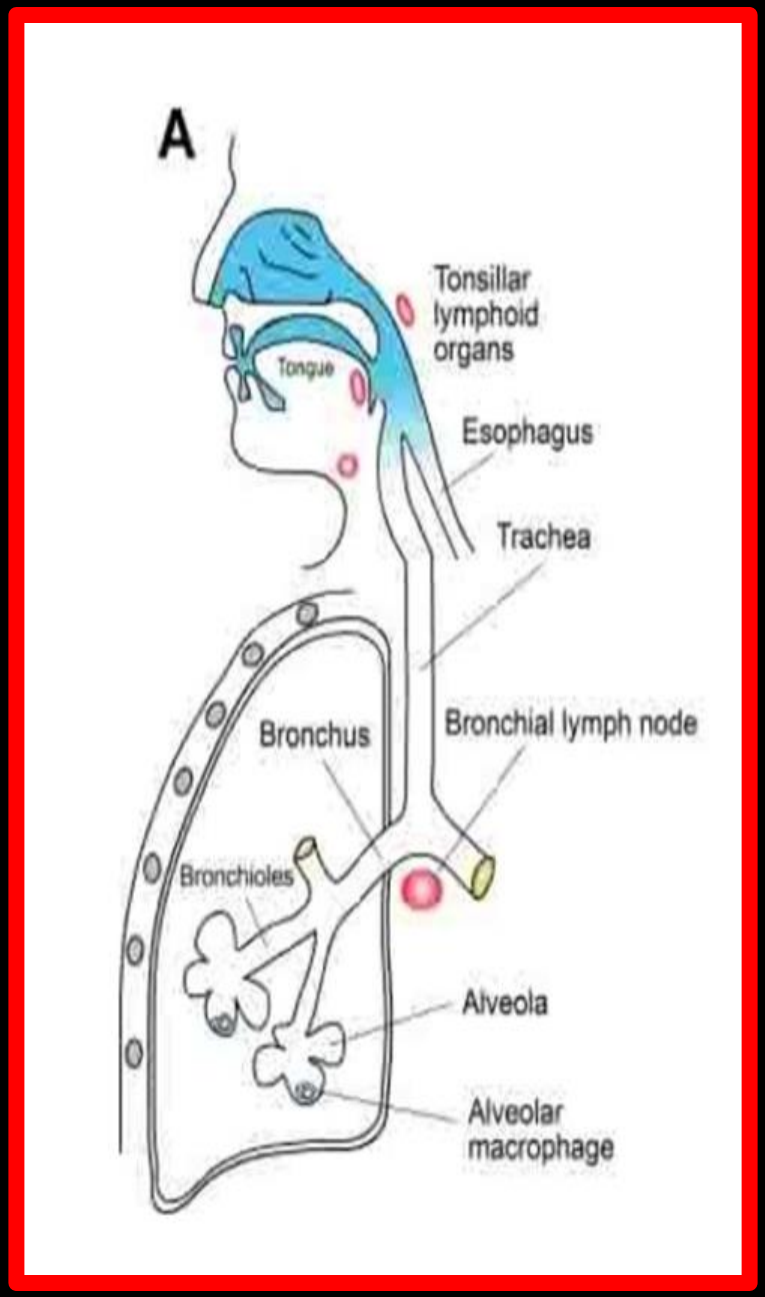
DANGER

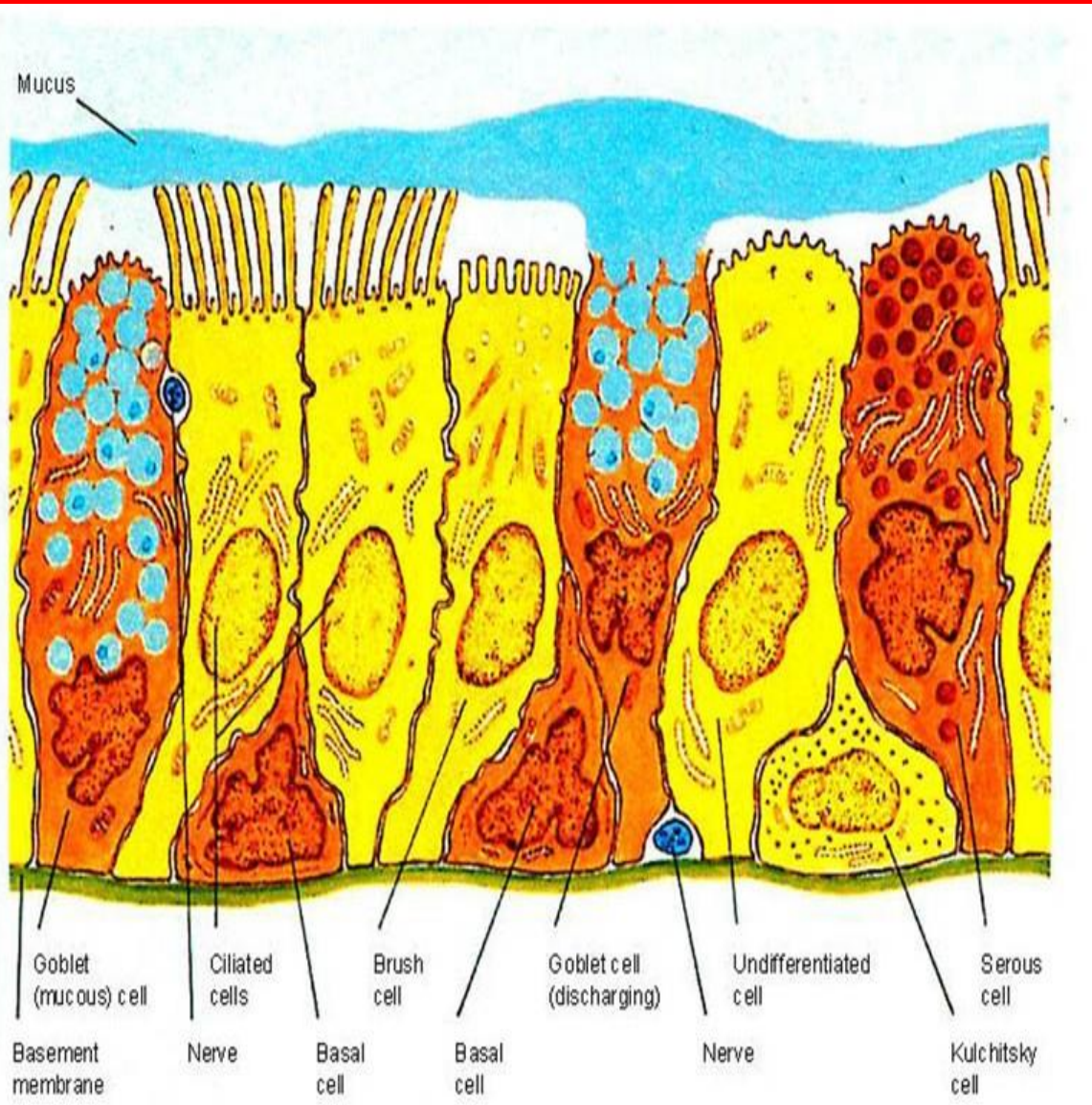
INHALATION HAZARD

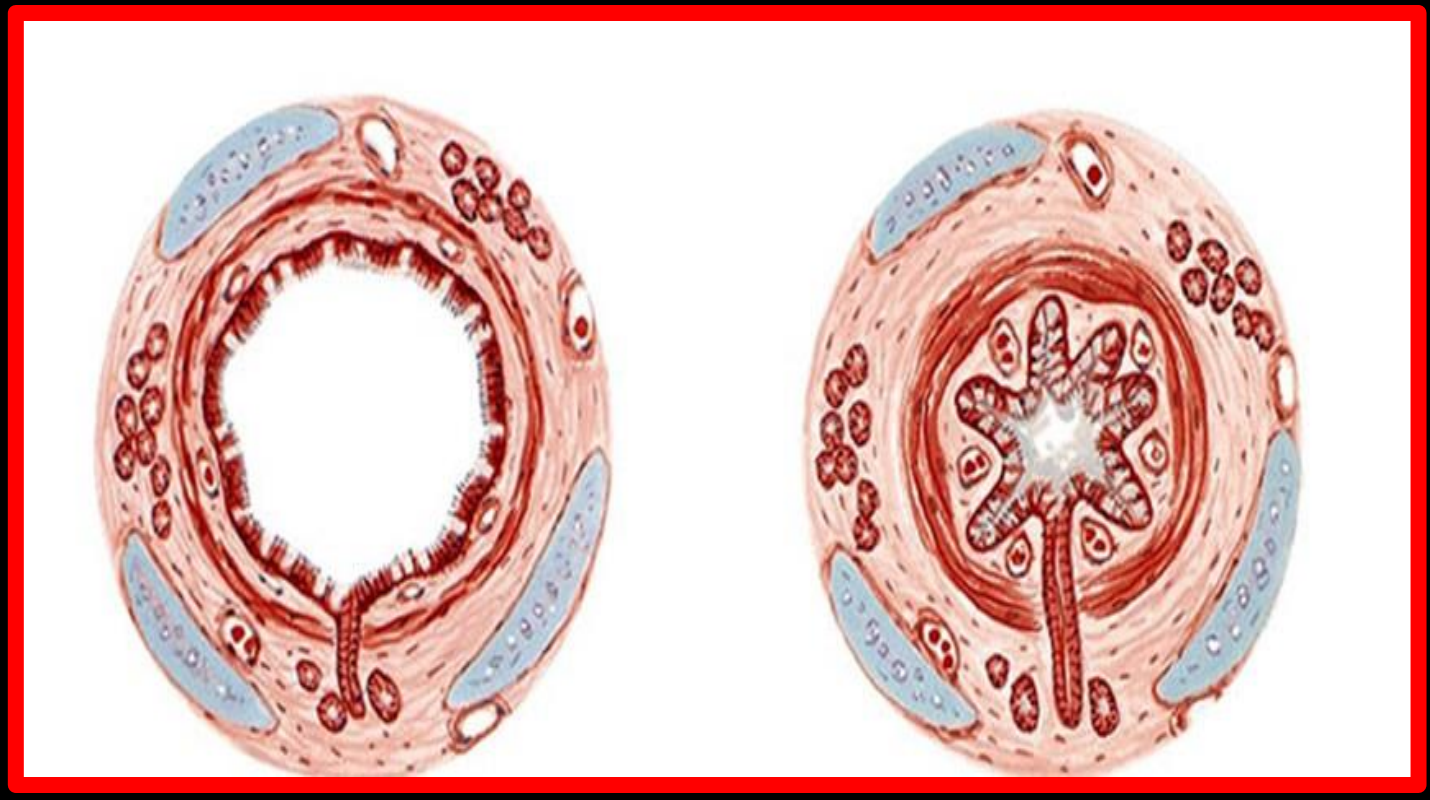
VAPORS ARE TOXIC

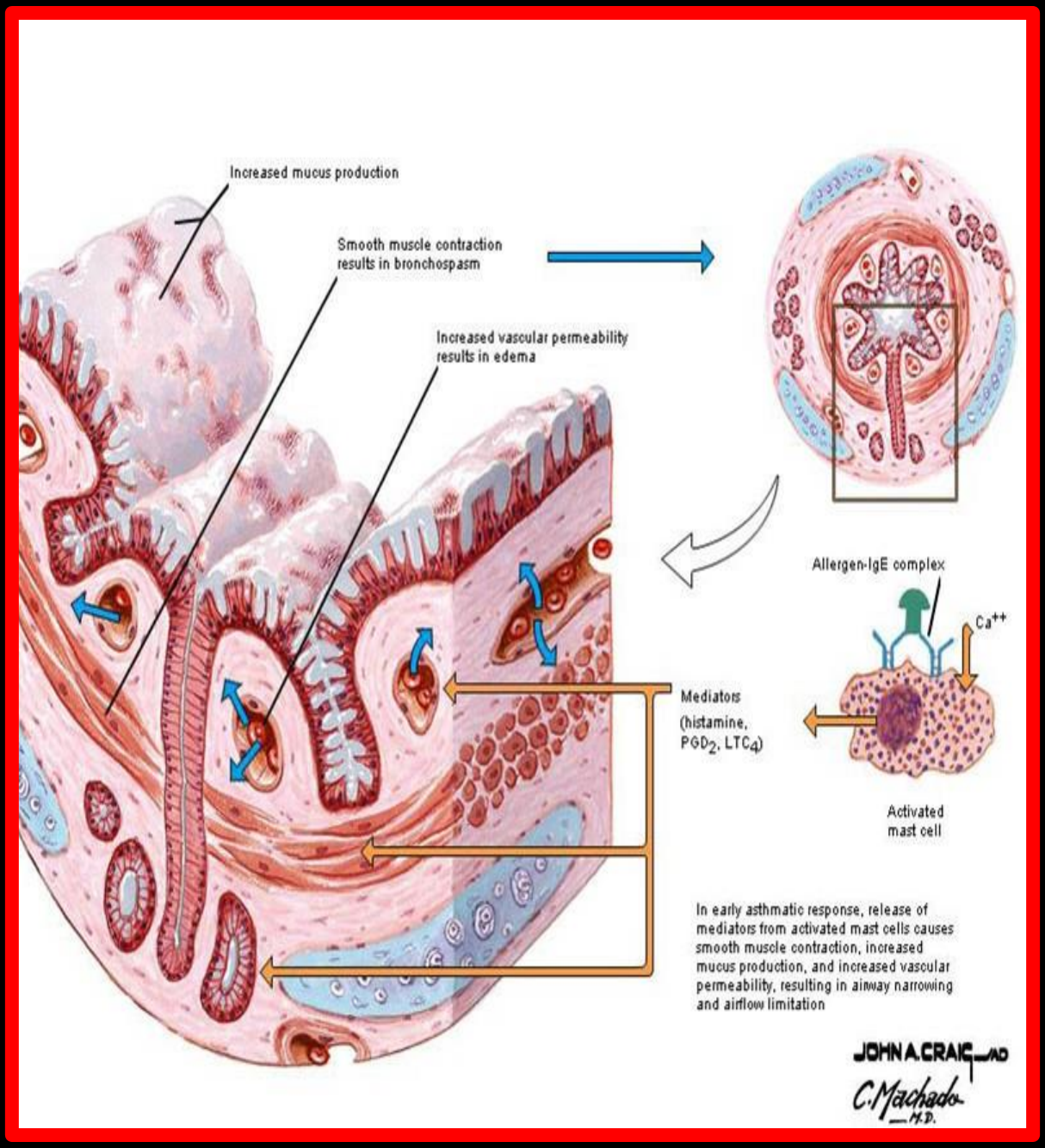
AVOID EXPOSURE TO VAPORS

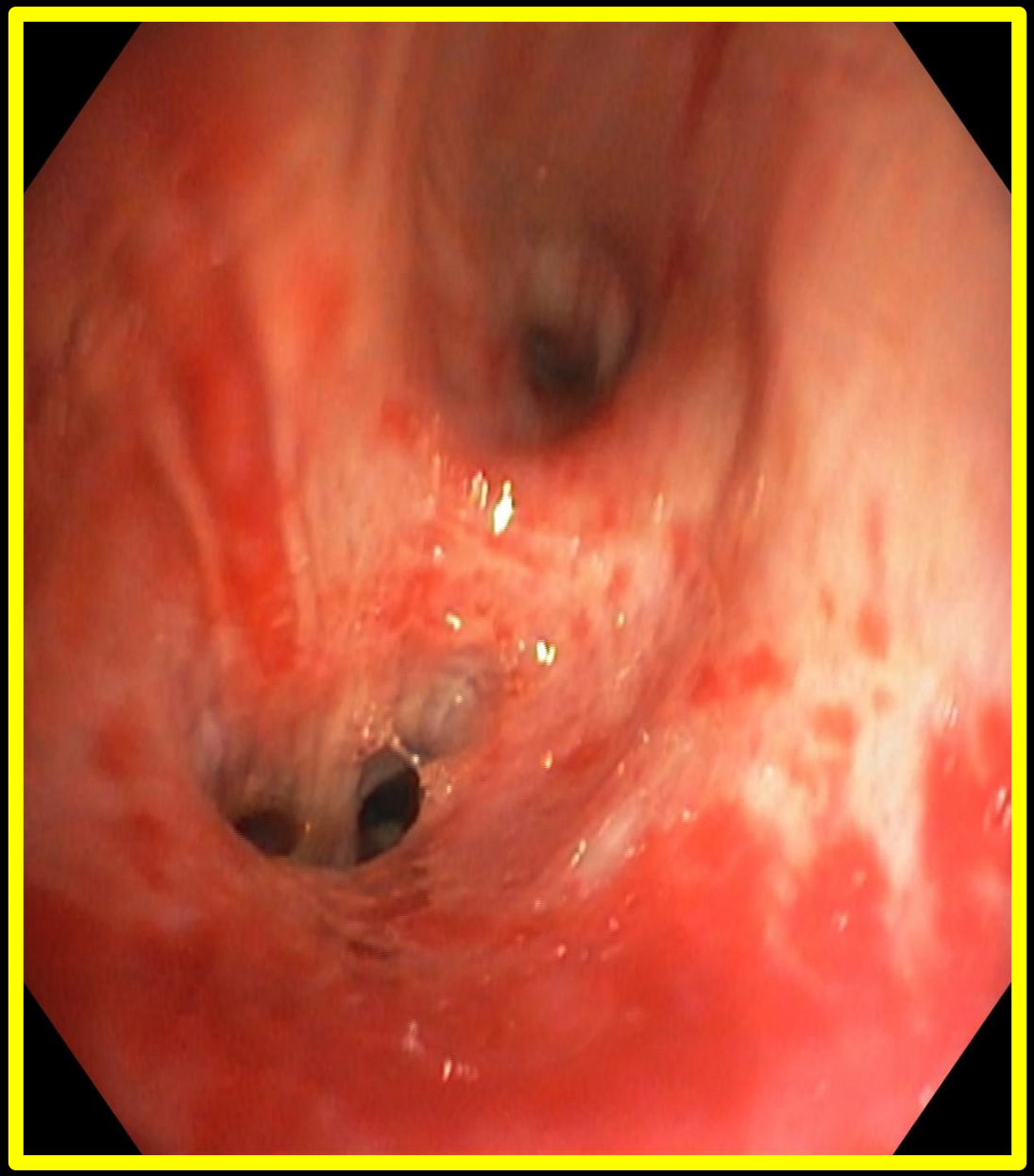
WEAR PROPER PERSONAL PROTECTION EQUIPMENT

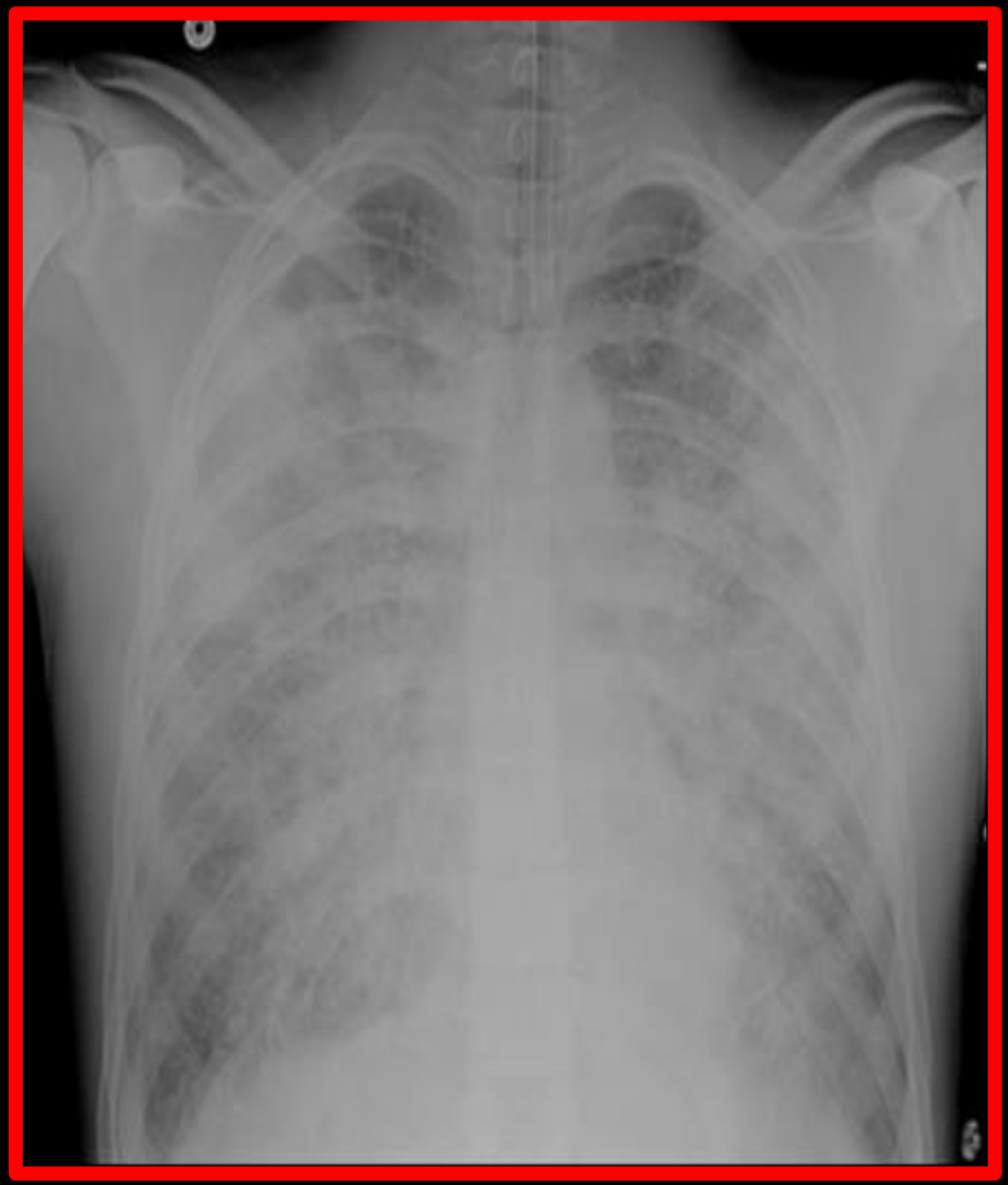

A
Tonsillar lymphoid organs
Tongue
Esophagus
Trachea
Bronchus
Bronchial lymph node
Bronchioles
Alveola
Alveolar macrophage


Mucus
Goblet (mucous) cell
Ciliated cells
Brush cell
Goblet cell (discharging)
Undifferentiated cell
Serous cell
Basement membrane
Nerve
Basal cell
Basal cell
Nerve
Kulchitsky cell




Increased mucus production
Smooth muscle contraction results in bronchospasm
Increased vascular permeability results in edema
Allergen-IgE complex
Ca^{++}
Mediators (histamine, PGD_2, LTC_4)
Activated mast cell
In early asthmatic response, release of mediators from activated mast cells causes smooth muscle contraction, increased mucus production, and increased vascular permeability, resulting in airway narrowing and airflow limitation
JOHN A. CRAIG, MD
C. Machado, M.D.

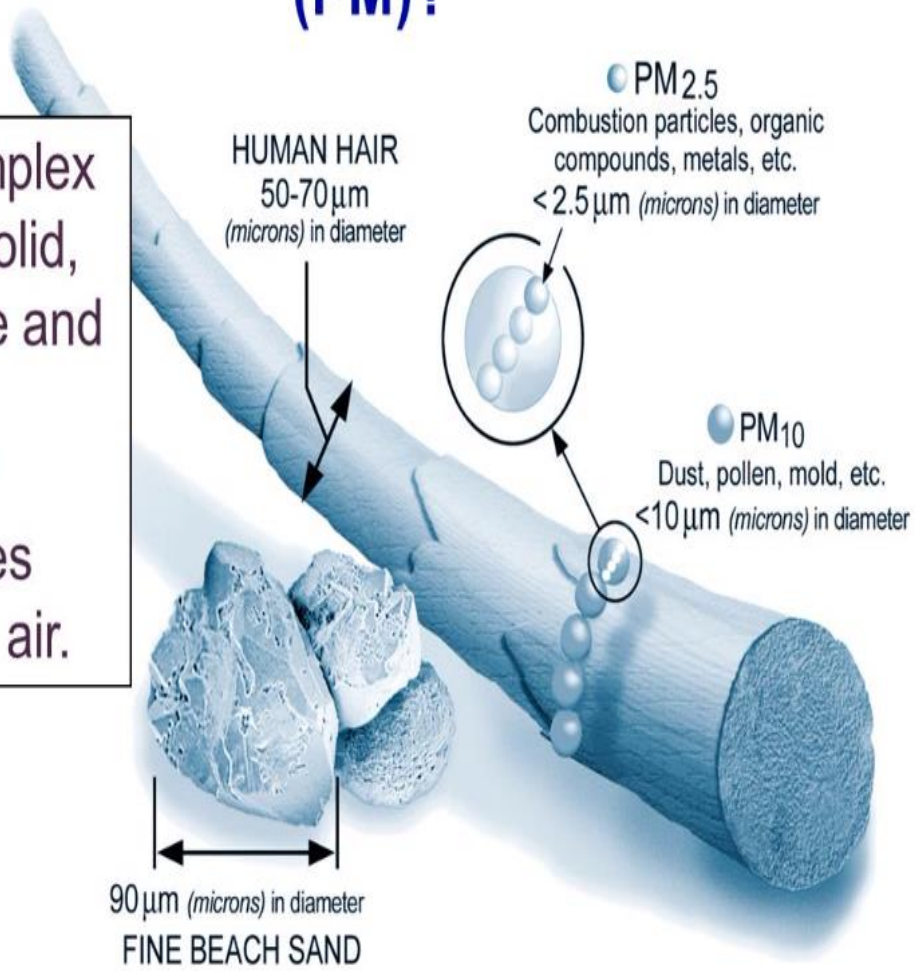


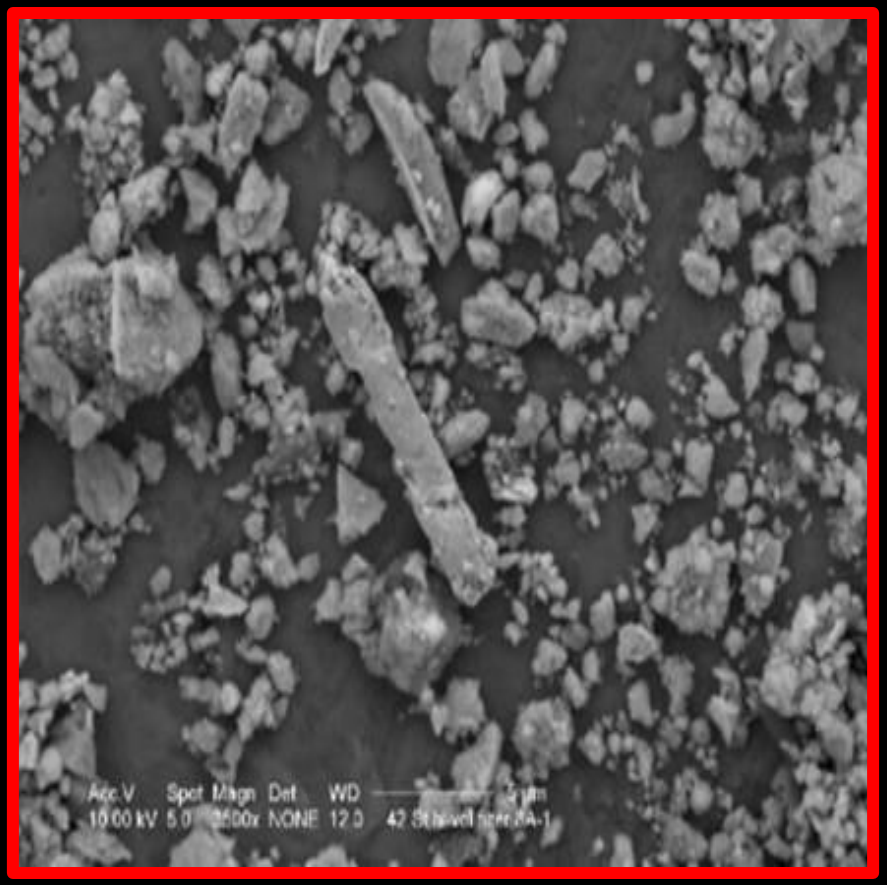


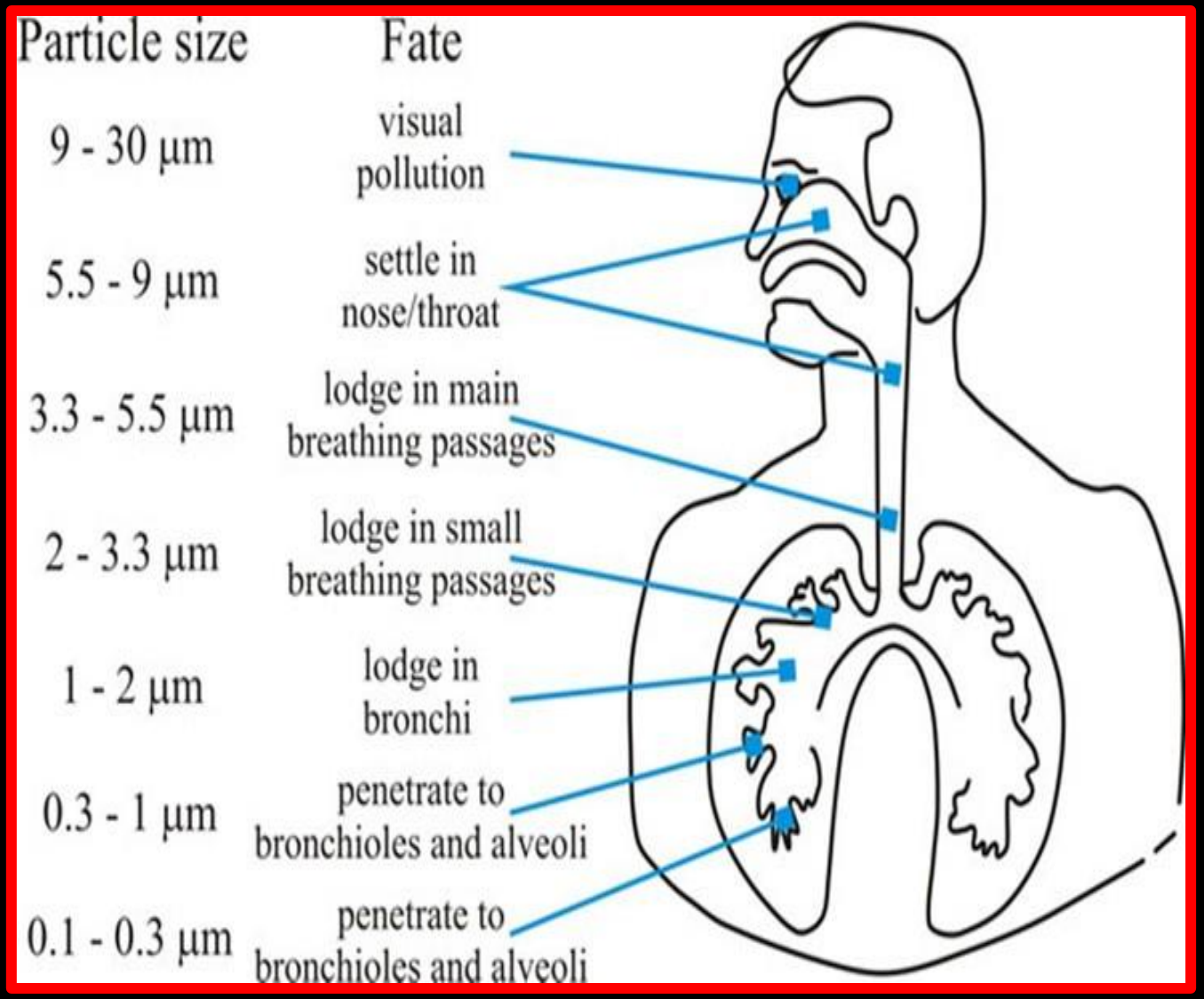
- Aerosol solid or liquid particles (fine drops or droplet) that are suspended in the air from a mixture.
- Dust consist of particles in the solid phase.
- Fumes- chemical change in a compound during processes such as welding.
- Smokes- consist of particles of both solid and some times liquid phase and the associated gases that result from combustion.
- Smog- Smokes and Fog
- Mists (small droplets of water suspended in air condensing on other particles)

What is a Particulate Mater (PM)?

PM is a complex mixture of solid, semi-volatile and aqueous materials of various sizes found in the air.




Acc.V Spot Magn Det WD
10.00 kV 5.0 3500x NONE 12.0


Particle size
Fate
9 - 30 µm
visual pollution
5.5 - 9 µm
settle in nose/throat
3.3 - 5.5 µm
lodge in main breathing passages
2 - 3.3 µm
lodge in small breathing passages
1 - 2 µm
lodge in bronchi
0.3 - 1 µm
penetrate to bronchioles and alveoli
0.1 - 0.3 µm
penetrate to bronchioles and alveoli

Introduction

Urban exposure to photochemical smog

Introduction

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

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

Principles of Disease

Clinical Features

manifestations of hypoxia

Autonomic stimulation

Cerebral hypoxia

Stimulator: hypoxemia or hypercarbia ?

Clinical Features

If FiO_2 falls below 10% :

FiO_2 below 6%: is Fatal !

Diagnostic Strategies and Differential Considerations

Minimally symptomatic or asymptomatic patients do not require any tests.

Diagnostic Strategies and Differential Considerations

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

The presenting complaints :nonspecific

Management and Disposition

Removal from exposure

- Mildly-poisoning patients

Management and Disposition

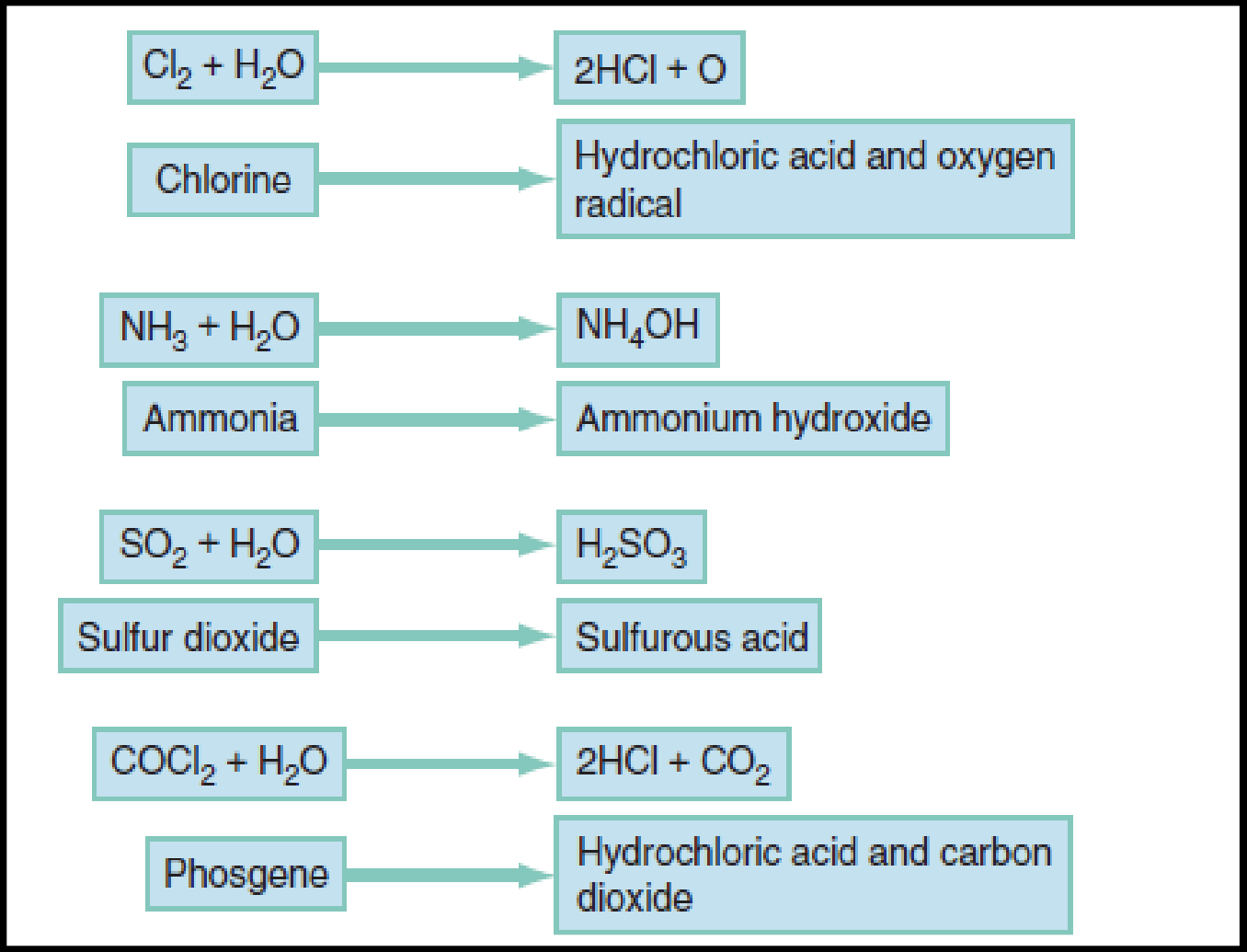
Risk for complications of hypoxia

SECOND : PULMONARY IRRITANTS

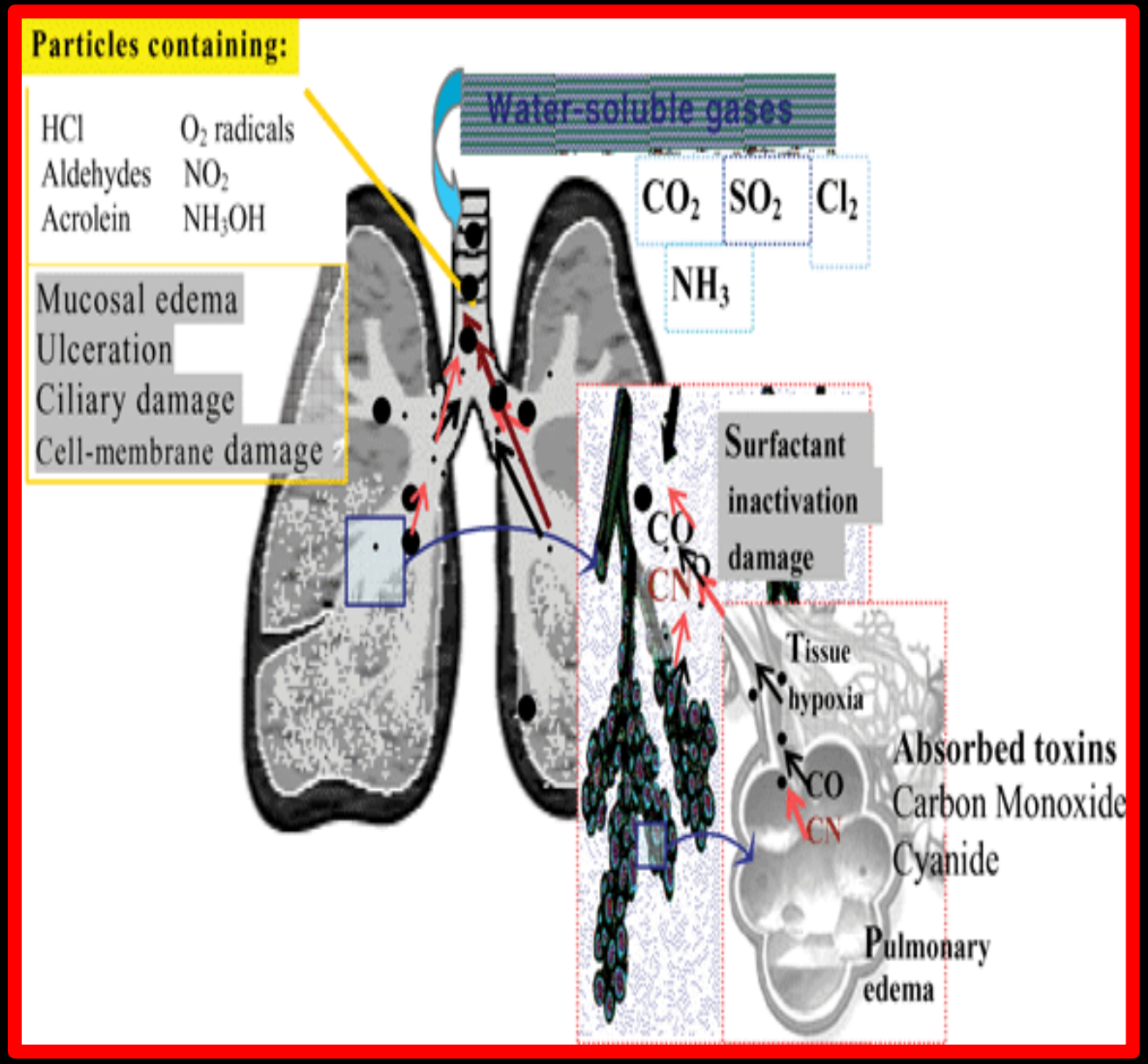
Pulmonary irritant gases

Principles of Disease

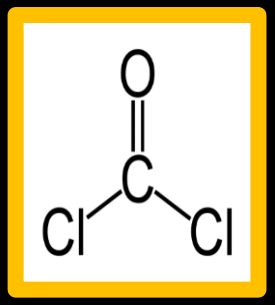
Sample reactions of pulmonary irritants reacting with water in the lung

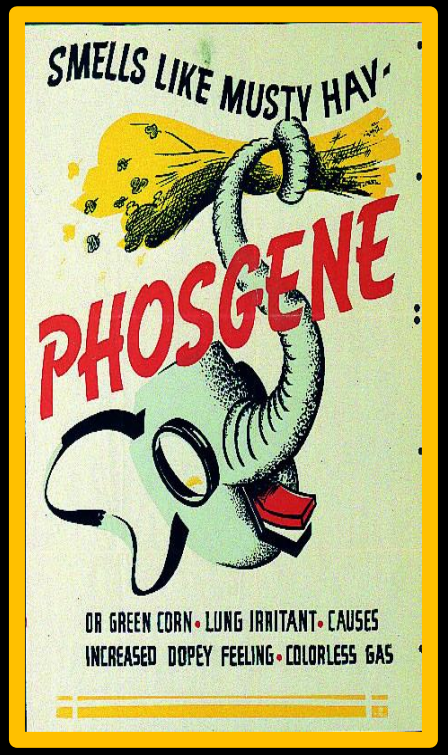


Clinical Features


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





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

68,000

36,000

25,000

Clinical Features

Diagnostic Strategies and Differential Consideration

physical examination

laryngoscopy

Diagnostic Strategies and Differential Consideration

No clinical tests can identify the specific irritant

Management

visualization of the larynx and immediate airway stabilization

(potential rapidity of airway deterioration)

Management

Inhaled beta2-adrenergic

Ipratropium bromide

No clearindication for corticosteroids

Management

Nebulized 2% sodium bicarbonate solution

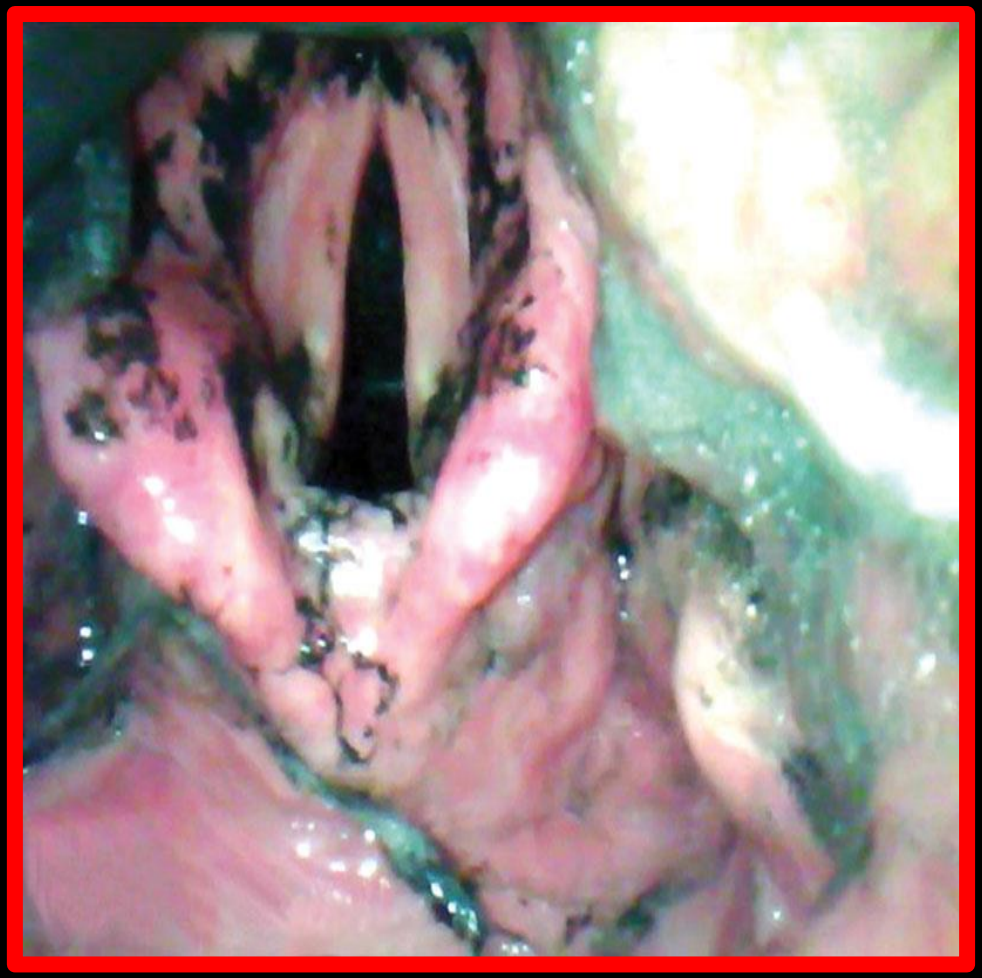
chlorine or hydrogen chloride gas

Management

- Diagnosis of ALI (ARDS)

THIRD: SMOKE INHALATION

How does it harm victims?




A
B
C

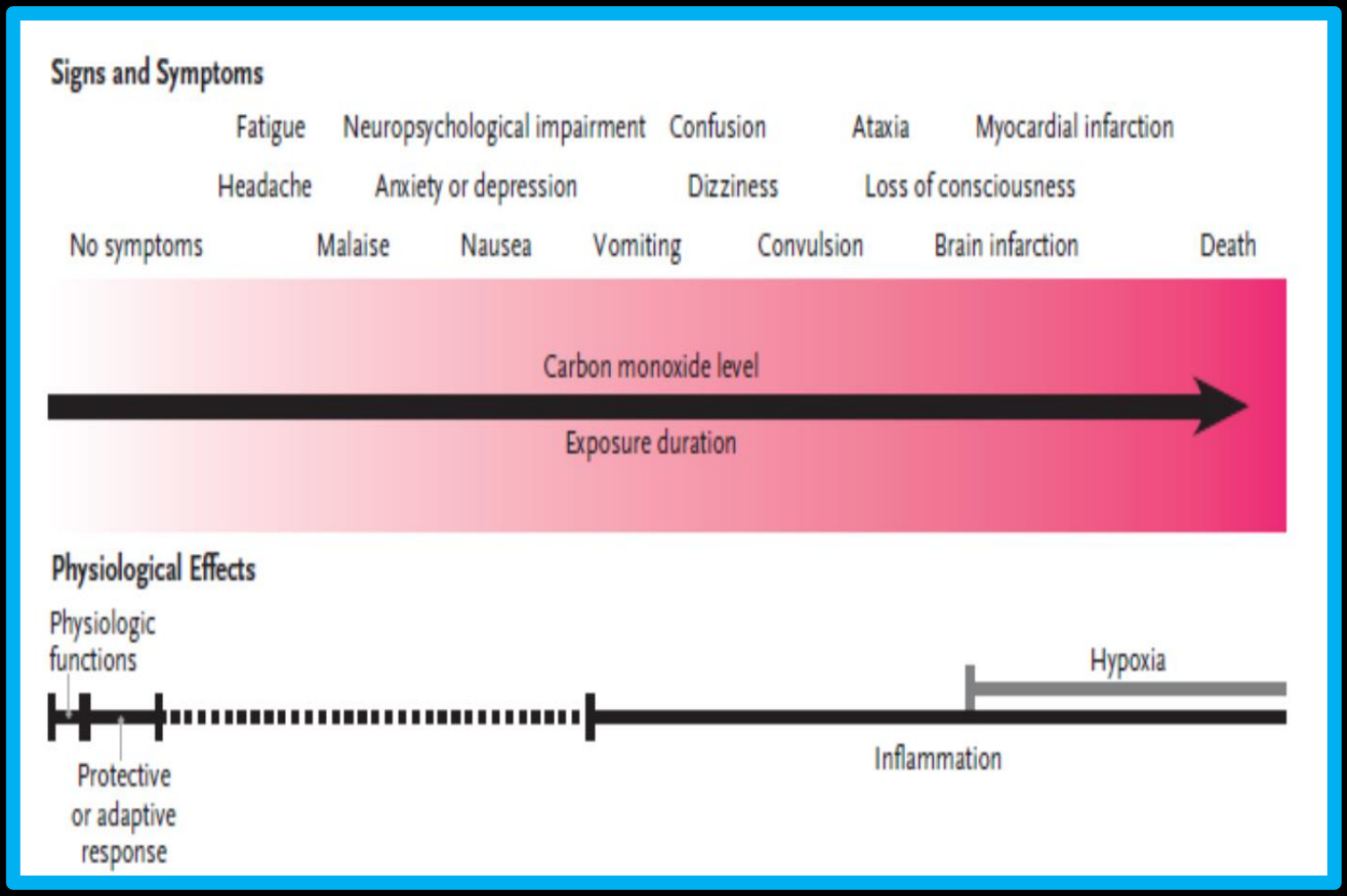
SMOKE INHALATION
Principals :

Principles of Disease

- **At Temp° : 350° C and 500° C**

- **Heat capacities of steam**

(approximately 4000 times that of air)


Signs and Symptoms
Fatigue
Neuropsychological impairment
Confusion
Ataxia
Myocardial infarction
Headache
Anxiety or depression
Dizziness
Loss of consciousness
No symptoms
Malaise
Nausea
Vomiting
Convulsion
Brain infarction
Death
Carbon monoxide level
Exposure duration
Physiological Effects
Physiologic functions
Hypoxia
Protective or adaptive response
Inflammation

COHb %	Symptoms
0-5	Normal
15-20	Headache, confusion, fatigue
20-40	Hallucination, vision Δ's
40-60	Combative, coma
60 +	Cardiopulmonary arrest

Clinical Features

morbidity and mortality : respiratory tract damage.

***Maybe delayed !**

Principles of Disease

- Latency period: variable

Principles of Disease

- **Carbon monoxide (CO) and Cyanide**

in a different room

engine exhaust

Diagnostic Strategies and Differential Considerations

1.asphyxia

2.airway compromise

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

Management

Cyanide :

suggests concomitant cyanide poisoning

Management : Admission

Critical Care Unit or Burn Center

Burn Intensive Care Unit

Management : Supportive

Bronchoscopy with bronchoalveolar lavage

Corticosteroids

Antibiotics

Management : Supportive

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

- Rapid assessment of the airway and early intubation is critical !

((most important aspects of care))

THANKS