

# Clinical Features of Hypoxia

## Symptoms:

- Hypoxia of gradual onset:
  - dyspnoea, drowsiness, headache, disorientation, anoxia
  - nausea, vomiting
- Severe hypoxia onset:
  - a sudden drop in inspired air of  $pO_2$
  - $< 20 \text{ mm Hg}$ : loss of consciousness in 10-20 sec & death in 4-5 min

## Signs

- Cyanosis [bluish discoloration of skin and mucous memb caused by  $> 5 \text{ g}$  of deoxy Hb / 100 ml of capillary blood].
- Tachycardia [as a result of peripheral chemoreceptor reflex response to low arterial  $O_2$  tension].
- Tachypnoea [rapid breathing] - reflex response to hypoxia that are activated by peripheral chemoreceptors.

## Effects of Hypoxia

### 1. Respiratory System

- all types of hypoxia except anaemic hypoxia stimulates peripheral chemoreceptors to increase respiration.

### 2. CNS

- drowsiness, impaired judgement
- depression or excitement
- headache
- talkativeness, emotional outburst of laughing, shouting or crying

### 3. CVS

- ~~inc~~  $\uparrow$  in Heart Rate
- $\uparrow$  in BP
- $\uparrow$  respiration

### 4. Nausea, vomiting, anorexia

### 5. On all: hypoxia metabolic factors (ATP)

## Treatment of Hypoxia

### Oxygen Therapy

- Arterial  $PO_2$  can be Fed either by:
  - a. Inhalation of 100% pure  $O_2$
  - b. Inhalation of 100% pure  $O_2$  at high barometric pressure, called hyperbaric  $O_2$  therapy.

### \* Methods

- 1) Oxygen Tent
- 2) Oxygen Mask
- 3) Mechanical ventilation
- 4) Arterial line

### $O_2$ therapy in different forms of Hypoxia

- In hypoxic hypoxia - Per arterial  $PO_2$
- In anemic hypoxia - Per quantity of dissolved  $O_2$  in plasma
- Obstructive hypoxia - not useful
- Stiffling hypoxia - not useful.

### Hyperbaric Oxygen Therapy

- Administration of 100% oxygen at high barometric pressure (3 atm P)
- Main aim: to ↑ the  $O_2$  dissolved in the dissolved form in the plasma.
- This is the most common cause for  $O_2$  toxicity
- Normal dissolved  $O_2$  in plasma is 0.003/100 ml/mm Hg  $PO_2$

### Indication

- CO poisoning (anemic hypoxia)
- Gas gangrene
- Decompression sickness
- Air embolism
- Stiffling hypoxia

# HYPOXIA

Definition :- Deficiency of  $O_2$  at tissue level.

• Tissue suffer from hypoxia :-

- 1) when adequate  $O_2$  supply ↓
- 2) failure of utilizing available  $O_2$

## Types of Hypoxia

- Hypoxic hypoxia
- Anemic hypoxia
- Stagnant hypoxia
- Histotoxic hypoxia

### 1) Hypoxic Hypoxia

- $PO_2$  of the arterial blood ↓.
- $O_2$  GC of blood is normal
- Low arterial  $O_2$  content
- Low arterial % saturation of Hb.
- Low A-V  $pO_2$  difference.

#### Causes

1. Low  $PO_2$  in inspired air

a. At high altitude

b. Breathing gas mixture at low  $pO_2$

2. Hypoventilation / Decreased pulmonary ventilation

a. Obstruction of resp. passages in bronchial asthma.

b. Paralysis of resp. muscles in - poliomyelitis

c. Respiratory centre depression - by drugs like morphine

d. Kyphosis and scoliosis.

3. Diffusion defects

a. Marked reduction in area of resp. membl.

b. Increased thickness of resp membl.

4. Venous arterial shunts

→ venous blood enters arterial blood without going into the lungs

eg:- cyanotic congenital heart disease

→ therefore, arterial  $pO_2$  decreases

### 3) STAGNANT / ISCHAEMIC HYPOXIA

• Blood flow to tissues is so low / slow that adequate oxygen is not delivered to them despite normal arterial  $pO_2$  & Hb.

- Normal arterial  $pO_2$
- Normal arterial  $O_2$  content
- Arterial Hb content - normal
- Normal arterial % saturation of Hb.
- A-V  $pO_2$  difference - more than normal.

#### Causes

##### 1) Generalised

- congestive cardiac failure
- shock

##### 2) Localised

- atherosclerosis
- thrombosis
- embolism

### 4) HISTOTOXIC HYPOXIA

- Due to ↓ed ability of tissues to utilize  $O_2$ .
- Arterial  $pO_2$  is normal.
- Arterial  $O_2$  content - normal
- Arterial Hb content - normal
- Normal arterial % saturation of Hb
- A-V  $pO_2$  difference - less than normal.

#### Causes

- 1) cyanide poisoning
- 2) sulphite poisoning
- 3) Vitamin B deficiency / Beri beri

#### Pathophysiology

cyanide poisoning & sulphite poisoning  
↓  
disrupt cellular oxidative enzymes of paralyse the  
cytochrome oxidative system  
↓  
affects tissue oxidation

- When CO combines with Hb.
  - (i) the amt of Hb to combine with  $O_2$  is reduced
  - (ii) Moreover, COHb releases CO slowly
- CO has 200 times more affinity than  $O_2$ .
- ODC shifts to left decreasing the amt of  $O_2$  to be released.

### Signs and Symptoms of CO poisoning

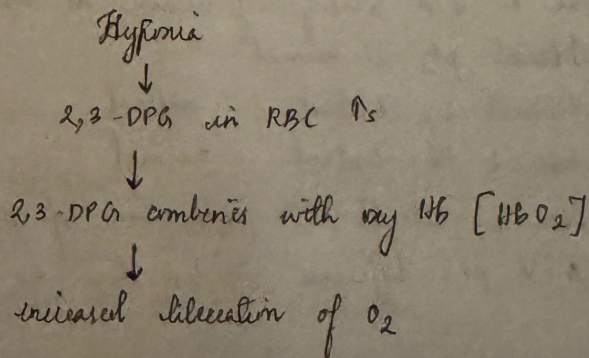
- Headache, nausea, loss of consciousness
- Cherry red discoloration of skin, mucous membrane and nail bed.
- Progressive brain damage, mental changes

### Treatment of CO poisoning

- Hyperbaric oxygenation : increases the dissociation of Hb

### Pathophysiology of Anemic Hypoxia

⇒ In anemic hypoxia, arterial hypoxia is not severe



⇒ In anemic hypoxia, in exercise

There is ↑  $O_2$  demand by tissues

↓

so ↑  $O_2$  consumption

↓

tissue demand is not fully met

↓

severe hypoxia

## Effect of $100\% \text{ O}_2$ therapy: oxygen toxicity

1. For  $> 8$  hrs, it stimulates nitric oxide synthesis in the resp system

- Normal oxygen, not blood, swelling, oxygen, tachypnea will burn/amblyopia

2. For  $> 24$  hrs = pneumothorax

- decrease in surfactant synthesis

- inhibitory ability of lung macrophages to kill bacteria

• Neonates infants are very sensitive to  $\text{O}_2$  toxicity

• They should never be given  $> 20\% \text{ O}_2$  inhalation as it can lead to:

a. Retrolental fibroplasia (retrolental fibroplasia) - formation of fibrous tissue

b. Formation of lung cyst & emphysema (surfactant deficiency)

3. CNS effects

• Nausea, irritability, dizziness, disorientation, muscle twitching, convulsions, coma and even death.

• Irritability, loss of eye, blurred vision etc.

• Oxidative damage can occur in any cell in the body.

### Mechanism of $\text{O}_2$ toxicity

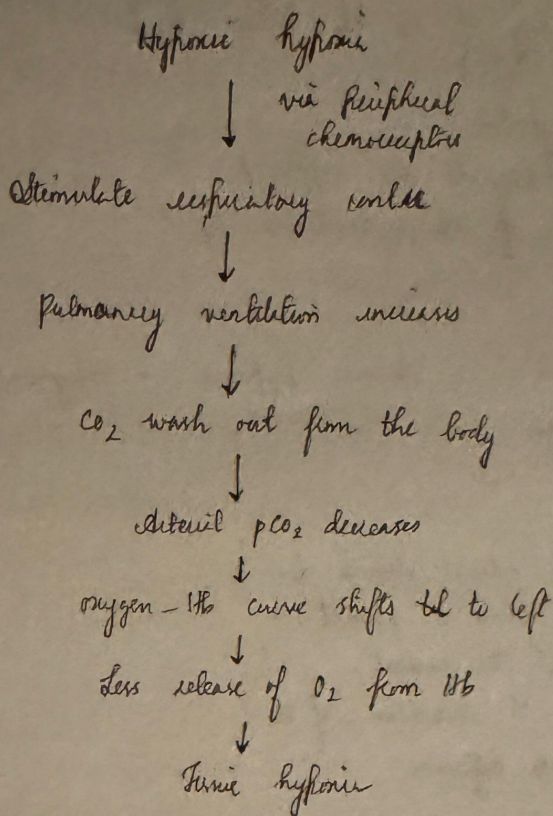
Inhalation of  $100\% \text{ O}_2$

↓  
• Conversion of molecular  $\text{O}_2$  to oxidizing free radicals like superoxide anion [ $\text{O}_2^-$ ] and hydrogen peroxide ( $\text{H}_2\text{O}_2$ )

↓  
They oxidise DNA (essential components of all animals) and destroy cellular enzymes (damaging cellular metabolic systems).

↓  
Tissue effects

# Pathophysiology of Hypoxic Hypoxia



## 2) ANEMIC HYPOXIA

- arterial pO<sub>2</sub> is normal but amt of Hb available to carry oxygen is reduced.
- Due to decreased Cc of blood
- pO<sub>2</sub> → normal
- arterial O<sub>2</sub> content → ~~normal~~ low
- arterial % of H saturation of Hb → low
- A-V pO<sub>2</sub> difference → normal

### Causes

- Anemia
- Altered Hb
  - (i) met Hb [Fe<sup>3+</sup> instead of Fe<sup>2+</sup>]
  - (ii) carboxy Hb [as CO poisoning]

### CO poisoning

- CO is toxic and it combines with Hb to form carboxymyoglobin as carboxy Hb [COHb]