Hypoxia

Fb/Nurse-Info

Hypoxia:

- **Is defined as oxygen deficiency at tissue level.**
- Traditionally divided into four categories:

1. Hypoxic hypoxia
2. Anaemic hypoxia
3. Stagnant hypoxia
4. Histotoxic hypoxia

Hypoxia:

1. **Hypoxic hypoxia-** PO_2 of arterial blood is reduced

2. **Anaemic hypoxia** - the arterial PO_2 is normal but the amount of Hb available to carry O_2 (oxygen carrying capacity) is reduced

- 3. Stagnant hypoxia - blood flow to a tissue is so low that adequate O_2 is not delivered to the tissues in spite of normal PO_2

- 4. Histotoxic hypoxia - the amount of O_2 delivered to the tissues is adequate but, because of the action of a toxin, the tissue cells cannot use the O_2 supplied to them.

Effects on Cells

- Hypoxia causes the production of transcription factors (hypoxia-inducible factors; HIFs).
- These are made up of α and β subunits.
- In normally oxygenated tissues, the subunits are rapidly destroyed.
- However, in hypoxic cells, the factors dimerize with the β
- the dimers activate genes that produce angiogenic factors and erythropoietin.

Effects of hypoxia:

- Sudden drop of inspired PO_2 to 20 mm Hg:
- 1. Loss of consciousness in 10-20 sec.
- 2. Death in 4-5 minutes.

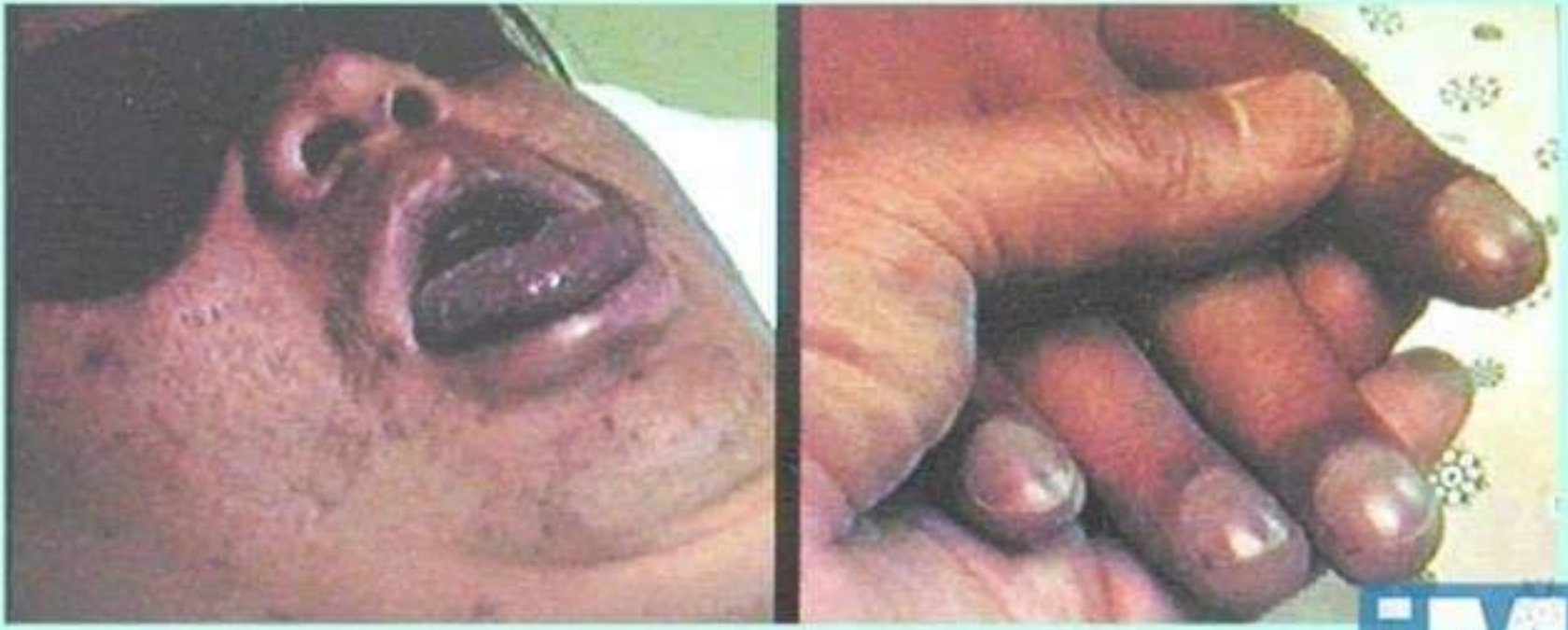
Less severe hypoxia:

1. Impaired judgement
2. Drowsiness
3. Dulled pain sensibility
4. Excitement
5. Disorientation
7. Anorexia, nausea, vomiting
8. Hypertension
9. Increased rate of ventilation

Respiratory stimulation

- Dyspnea: Difficult or laboured breathing in which the subject is conscious of shortness of breath.
- Hyperpnea: Increase in the rate and depth of breathing regardless of the patient's subjective sensation.
- Tachypnea: Rapid shallow breathing.

Cyanosis – bluish discoloration of mucous membranes & nails



Cyanosis: Appears when reduced Hb in the capillaries is more than 5 g /dL.

- Depends on

1. Total amount of Hb - easily seen in polycythaemia
2. Degree of Hb unsaturation
3. State of the capillary circulation

- Easily seen in: nail beds, mucous membranes, ear lobes, lips and fingers.
- Cyanosis does not occur in:

a. anaemic hypoxia - low Hb

b. CO poisonimg - CarbonmonoxyHb is cherry-red.

c. Histotoxic hypoxia - Blood gas content is normal

- High circulating levels of methaemoglobin also shows a bluish discoloration similar to cyanosis.

Causes of hypoxia:

- Hypoxic hypoxia:

1. Inadequate oxygenation of blood in the lungs due to extrinsic causes.

a. *Deficiency of O_2 in the atmosphere* -
Altitude; Mines

b. *Hypoventilation* –
Neuromuscular disorders;
Fatigue
Depression of RC

2. Lung disease

a. *Failure of gas exchange* - V-A shunts

b. *Failure of respiratory pump* -

- Pulmonary fibrosis
- Ventilation-perfusion imbalance
- Collapse of lung
- Pneumothorax
- Asthma
- Emphysema

- Anaemic hypoxia:

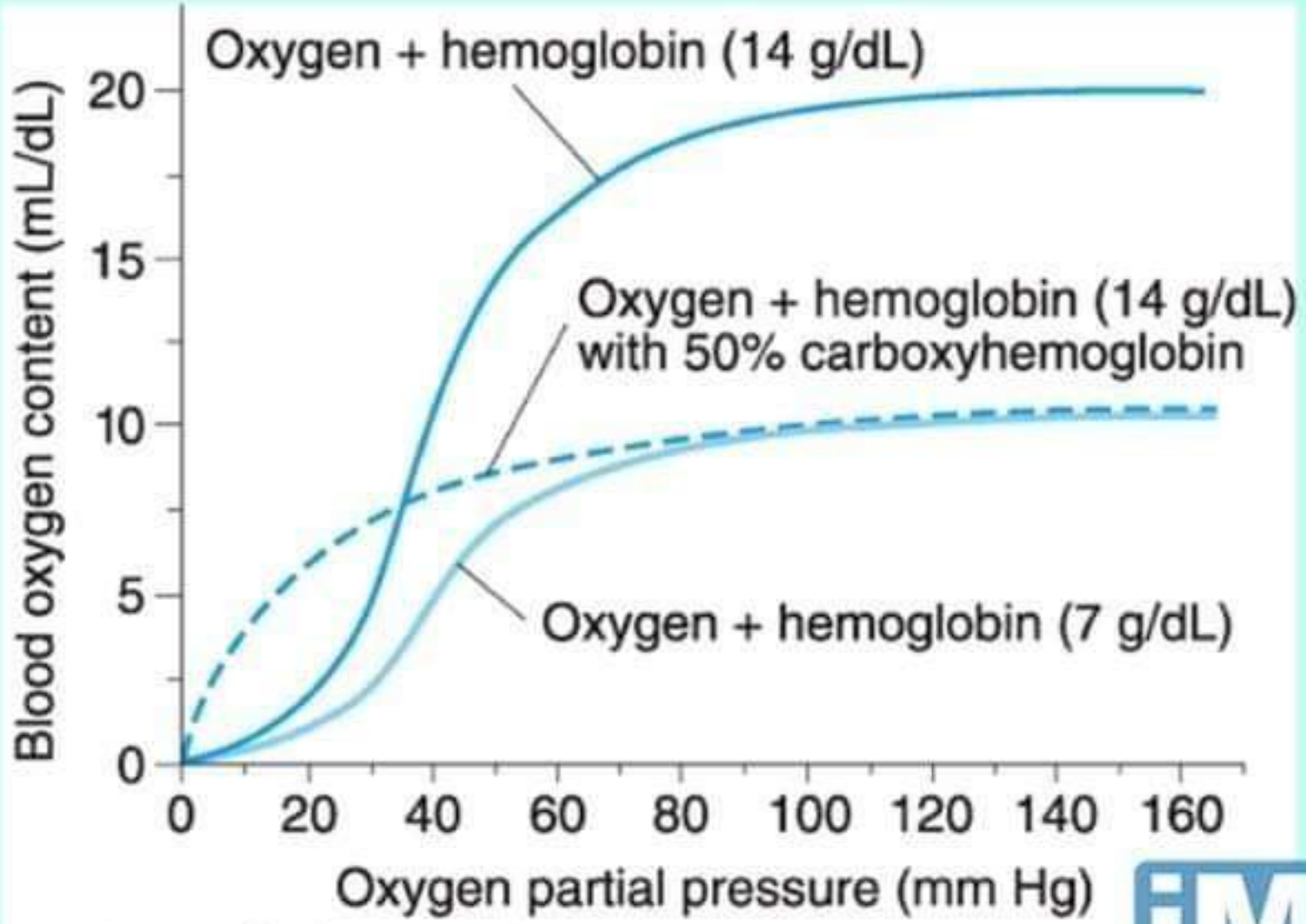
a. *Anaemia;*

b. *Carbonmonoxyhaemoglobin*

Anemic Hypoxia

- Hypoxia due to anemia is not severe at rest unless the hemoglobin deficiency is marked, because red blood cell 2,3-BPG increases.

- However, anemic patients may have considerable difficulty during exercise because of limited ability to increase O_2 delivery to the active tissues



Carbon Monoxide Poisoning

- CO is toxic
- it reacts with hemoglobin to form carbonmonoxyhemoglobin (carboxyhemoglobin, COHb)
- COHb cannot take up O2.

- The affinity of hemoglobin for CO is 210 times its affinity for O2, and COHb liberates CO very slowly.

- when COHb is present the dissociation curve of the remaining HbO2 shifts to the left, decreasing the amount of O2 released.

- This is why an anemic individual who has 50% of the normal amount of HbO2 may be able to perform moderate work,

- an individual whose HbO2 is reduced to 50% because of the formation of COHb is seriously incapacitated.

- The symptoms of CO poisoning are those of any type of hypoxia, headache and nausea,
- No stimulation of respiration, since in the arterial blood, PO_2 remains normal and the carotid and aortic chemoreceptors are not stimulated
- The cherry-red color of COHb is visible in the skin, nail beds, and mucous membranes.
- Death results when about 70–80% of the circulating hemoglobin is converted to COHb.
- The symptoms produced by chronic exposure to sublethal concentrations of CO are those of progressive brain damage, including mental changes and, sometimes, a parkinsonism-like state

Treatment of CO poisoning

- immediate termination of the exposure and adequate ventilation, by artificial respiration if necessary.

- Ventilation with O_2 is preferable to ventilation with fresh air, since O_2 hastens the dissociation of COHb.

- Hyperbaric oxygenation (see below) is useful in this condition

- Stagnant hypoxia:

a. General or local circulatory deficiency

- Histotoxic hypoxia:

b. Inhibition of tissue oxidative processes

a. cyanide

b. vitamin deficiency

OXYGEN TREATMENT

- **Value**
- Administration of oxygen-rich gas mixtures is of very limited value in stagnant, anemic, and histotoxic hypoxia because all that can happen is an increase in the amount of dissolved O_2 in the arterial blood.

- This is also true in hypoxic hypoxia when it is due to shunting of unoxygenated venous blood past the lungs..

- In other forms of hypoxic hypoxia, O_2 is of great benefit.
- Treatment regimes that deliver less than 100% O_2 are of value both acutely and chronically,

- In hypercapnic patients in severe pulmonary failure, the CO_2 level may be so high that it depresses rather than stimulates respiration.
- Some of these patients keep breathing only because the carotid and aortic chemoreceptors drive the respiratory center.
- If the hypoxic drive is withdrawn by administering O_2, breathing may stop.
- During the resultant apnea, the arterial PO_2 drops but breathing may not start again, because the increase in PCO_2 further depresses the respiratory center.
- Therefore, O_2 therapy in this situation must be started with care

Oxygen Toxicity

- The toxicity seems to be due to the production of the superoxide anion (O_2^-), which is a free radical, and H_2O_2.

- When 80–100% O_2 is administered to humans for periods of 8 hours or more, the respiratory passages become irritated, causing substernal distress, nasal congestion, sore throat, and coughing.

- Some infants treated with O_2 for respiratory distress syndrome develop a chronic condition characterized by lung cysts and densities **(bronchopulmonary dysplasia).**
- This syndrome may be a manifestation of O_2 toxicity.
- Another complication in these infants is **retinopathy of prematurity (retrolental fibroplasia),** the formation of opaque vascular tissue in the eyes, which can lead to serious visual defects