Slide 1

Hypoxia and cyanosis

Objectives

- **By the end of this lecture you should be able to:**
- Define hypoxia and list its various physiological and pathological causes.
- Define hypo and hyper-ventilation in terms of arterial PCO2 and PO2.
- Define cyanosis and its clinical presentation
- Define ventilation/perfusion ($\dot{V}/Q$) ratio and its normal values.

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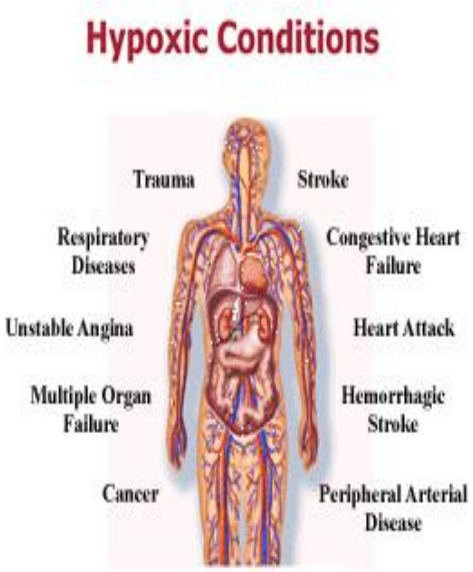
Hypoxia

Is defined as deficiency of oxygen in the tissue cells.

It can be classified into the following groups:-

- **Hypoxic or arterial hypoxia**
- **Anemic hypoxia**
- **Stagnant hypoxia**
- **Histiotoxic hypoxia**



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hypoxia;

The following is a descriptive classification of the causes of hypoxia:

Inadequate oxygenation of the blood in the lungs because of extrinsic reasons
- Deficiency of oxygen in the atmosphere
- Hypoventilation (neuromuscular disorders)

Pulmonary disease
- Hypoventilation caused by increased airway resistance or decreased pulmonary compliance
- Abnormal alveolar ventilation-perfusion ratio (including either increased physiologic dead space or increased physiologic shunt)
- Diminished respiratory membrane diffusion

Venous-to-arterial shunts (“right-to-left” cardiac shunts)

Inadequate oxygen transport to the tissues by the blood
- Anemia or abnormal hemoglobin
- General circulatory deficiency
- Localized circulatory deficiency (peripheral, cerebral, coronary vessels)

- Tissue edema

Inadequate tissue capability of using oxygen

- Poisoning of cellular oxidation enzymes
- Diminished cellular metabolic capacity for using oxygen, because of toxicity, vitamin deficiency, or other factors

This classification of the types of hypoxia is mainly self-evident from the discussions earlier in the chapter. Only one type of hypoxia in the classification needs further elaboration: the hypoxia caused by inadequate capability of the body's tissue cells to use oxygen.

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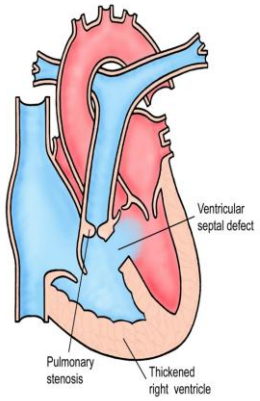
I-Hypoxic or arterial hypoxia

Reduced arterial PO2 it can be due to

- Alveolar hypoventilation
- Diffusion abnormalities
- Right to left shunt
- Ventilation-perfusion imbalance (including increased physiological dead space and physiological shunt).



Right to left shunt

A right-to-left shunt occurs when:
there is an opening or passage between the atria, ventricles, and/or great vessels; *and*,
right heart pressure is higher than left heart pressure and/or the shunt has a one-way valvular opening.

A **pulmonary shunt:** the blood entering the arterial system without passing through ventilated areas of lung causing the PO2 of arterial blood to be less than that of alveolar PO2.

is a physiological condition which results when the alveoli of the lungs are perfused with blood as normal, but ventilation (the supply of air) fails to supply the perfused region. In other words, the ventilation/perfusion ratio (the ratio of air reaching the alveoli to blood perfusing them) is zero. A pulmonary shunt often occurs when the alveoli fill with fluid, causing parts of the lung to be unventilated although they are still perfused
Abnormal alveolar ventilation-perfusion ratio (including either increased physiologic dead space or increased physiologic shunt)

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II-Anemic hypoxia

- It is caused by reduction in the oxygen carrying capacity of the blood, due to decreased amount of Hb or abnormal type of Hb which is unable to carry oxygen.
- The PO2 and % Hb-O2 is normal.

Causes:

1- True anemia

2-Abnormal Hb e.g met hemoglobin, Fe^{3+} (ferric)not the Fe^{2+} ,carboxyhemoglobin.

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Anemic hypoxia is oxygen deprivation in the tissues of the body caused by a decline in the blood's ability to carry oxygen. This can be caused by a number of factors including true anemia, where the number of red blood cells drops, making it harder for the blood to supply the body with the oxygen it needs. Patients with this condition may initially appear well, but start to develop fatigue, air hunger, and confusion as the limited oxygen supply persists and affects the brain

in which the iron in the heme group is in the Fe^{3+} (ferric) state, not the Fe^{2+} (ferrous) of normal hemoglobin

Carboxyhemoglobin: that has carbon monoxide instead of the normal oxygen bound to it. Carbon monoxide has a much stronger binding to hemoglobin than oxygen

III-Stagnant hypoxia:

- Caused by reduced blood flow through the tissues,

  so more and more oxygen is extracted from the blood, and due to slow circulation less oxygen is carried by the blood at the lung , leading to hypoxia.
- **Causes:**
- 1-General slowing of the circulation, as in heart failure and shock
- 2-Local slowing e.g vasoconstriction, cold, arterial wall spasm.

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IV- Histiotoxic hypoxia

- This is inability of the tissues to use oxygen due to inhibition of the oxidative enzyme activity
- An oxidative enzyme is an enzyme that catalyses oxidation reaction
- e.g cyanide poisoning causing blockade of the cytochrome oxidase activity

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An oxidative enzyme is an enzyme that catalyses oxidation reactio

Cyanide poisoning occurs when a living organism is exposed to a compound that produces cyanide ions (CN^-) when dissolved in water. Common poisonous cyanide compounds include hydrogen cyanide gas and the crystalline solids potassium cyanide and sodium cyanide The cyanide ion halts cellular respiration by inhibiting an enzyme in the mitochondria called cytochrome c oxidase.

Effects of hypoxia

- According to the degree of hypoxia it could lead to

  impairment of judgment,

  inability to perform complex calculations,

  headache, nausea, irritability, dyspnea, increased heart rate,

  reduction in muscle working capacity,

  even coma and death may result.

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Treatment of hypoxia

- Is by giving oxygen therapy in a tent or high oxygen tension mask.
- This is useful in hypoxic hypoxia, but of less value in other types of hypoxia .
- Histiotoxic hypoxia will not benefit from O2 therapy.

Oxygen Therapy in Different Types of Hypoxia

Oxygen can be administered by (1) placing the patient's head in a "tent" that contains air fortified with oxygen, (2) allowing the patient to breathe either pure oxygen or high concentrations of oxygen from a mask, or (3) administering oxygen through an intranasal tube. Recalling the basic physiologic principles of the different types of hypoxia, one can readily decide when oxygen therapy will be of value and, if so, how valuable.

In *atmospheric hypoxia*, oxygen therapy can completely correct the depressed oxygen level in the inspired gases and, therefore, provide 100 percent effective therapy.

In *hypoventilation hypoxia*, a person breathing 100 percent oxygen can move five times as much oxygen into the alveoli with each breath as when breathing normal air. Therefore, here again oxygen therapy can be extremely beneficial. (However, this provides no benefit for the excess blood carbon dioxide also caused by the hypoventilation.)

Figure 42-8 Absorption of oxygen into the pulmonary capillary blood in pulmonary edema with and without…In *hypoxia caused by impaired alveolar membrane diffusion*, essentially the same result occurs as in hypoventilation hypoxia because oxygen therapy can increase the PO_2 in the lung alveoli from the normal value of about 100 mm Hg to as high as 600 mm Hg. This raises the oxygen pressure gradient for diffusion of oxygen from the alveoli to the blood from the normal

value of 60 mm Hg to as high as 560 mm Hg, an increase of more than 800 percent. This highly beneficial effect of oxygen therapy in diffusion hypoxia is demonstrated in Figure 42-8, which shows that the pulmonary blood in this patient with pulmonary edema picks up oxygen three to four times as rapidly as would occur with no therapy.

In *hypoxia caused by anemia, abnormal hemoglobin transport of oxygen*, *circulatory deficiency*, *or physiologic shunt*, oxygen therapy is of much less value because normal oxygen is already available in the alveoli. The problem instead is that one or more of the mechanisms for transporting oxygen from the lungs to the tissues are deficient. Even so, a small amount of extra oxygen, between 7 and 30 percent, can be *transported in the dissolved state* in the blood when alveolar oxygen is increased to maximum even though the amount transported by the hemoglobin is hardly altered. This small amount of extra oxygen may be the difference between life and death.

In the different types of *hypoxia caused by inadequate tissue use of oxygen*, there is abnormality neither of oxygen pickup by the lungs nor of transport to the tissues. Instead, the tissue metabolic enzyme system is simply incapable of using the oxygen that is delivered. Therefore, oxygen therapy provides no measurable benefit.

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Hypercapnea

Excess of CO2 in body fluids, it usually occurs with hypoxia, PCO2 increases above 52 mmHg, it decreases the PH

Features of hypercapnea

- Peripheral vasodilatation
- Sweating
- Warm extremities and bounding pulse
- Muscle twitching
- Headache, drowsiness and com
- Papilledema (swelling of optic disc)

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The **optic disc** or **optic nerve head** is the point of exit for ganglion cell axons leaving the eye.

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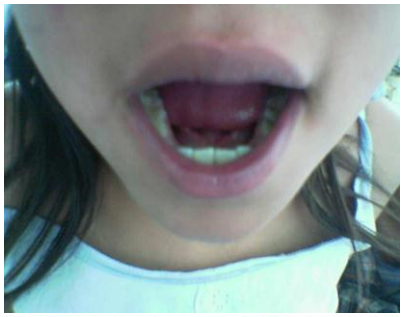
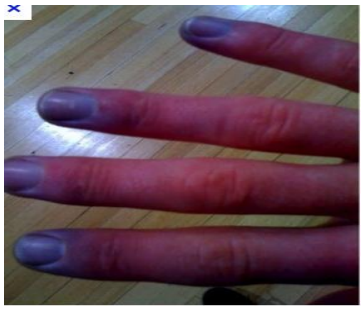
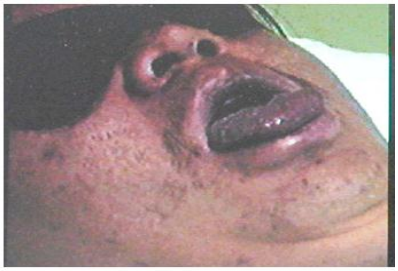
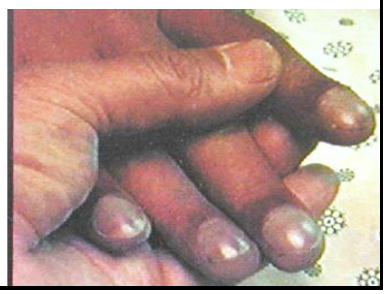
Cyanosis









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Cyanosis

- Blue discoloration of the skin and mucus membrane due to more than 5 g/dl of reduced (deoxygenated) hemoglobin in blood.
- A person with anemia almost never develop cyanosis due to low amount of Hb for 5 grams to be deoxygenated /100ml blood.

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Cyanosis

The term *cyanosis* means blueness of the skin, and its cause is excessive amounts of deoxygenated hemoglobin in the skin blood vessels, especially in the capillaries. This deoxygenated hemoglobin has an intense dark blue–purple color that is transmitted through the skin.

In general, definite cyanosis appears whenever the *arterial blood* contains more than 5 grams of deoxygenated hemoglobin in each 100 milliliters of blood. A person with *anemia* almost never becomes cyanotic because there is not enough hemoglobin for 5 grams to be deoxygenated in 100 milliliters of arterial blood. Conversely, in a person with excess red blood cells, as occurs in *polycythemia vera*, the great excess of available hemoglobin that can become deoxygenated leads frequently to cyanosis, even under otherwise normal conditions.

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Ventilation –perfusion ratio (V/Q)

- It is the ratio of alveolar ventilation to pulmonary blood flow per minute.

The alveolar ventilation at rest (4.2 L/min)

- The pulmonary blood flow is equal to right ventricular output per minute (5L/min)

SO $\quad$ V/Q ratio = $\frac{4.2}{5}$ = 0.84

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a highly quantitative concept has been developed to help us understand respiratory exchange when there is imbalance between alveolar ventilation and alveolar blood flow. This concept is called the *ventilation-perfusion ratio*.

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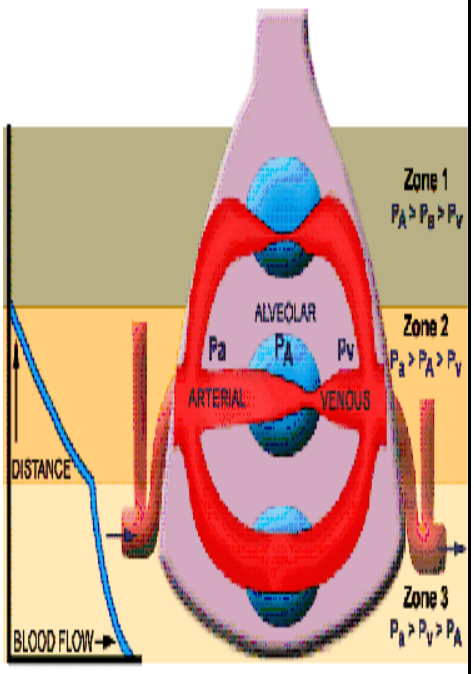
Cont…V/Q ratio

- Average V/Q ratio across the lung is 0.8.
- At the apex V/Q ratio = 3
- At the base V/Q ratio=0.6

  So the apex is more ventilated than perfused and the base is more perfused than ventilated.
- *During exercise the V/Q ratio becomes more homogenous among different parts of the lung*



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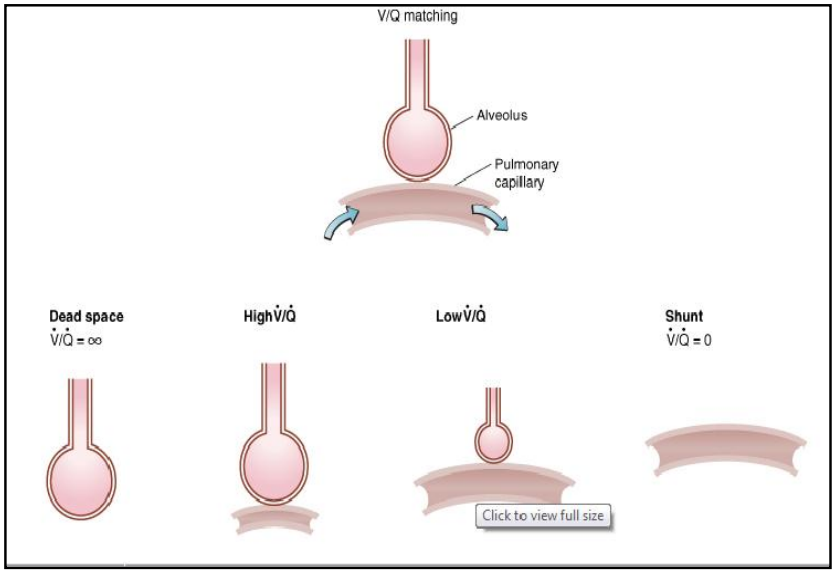
Ventilation/perfusion abnormalities



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Cont… V/Q ratio

- The main function of this ratio is to determine the state of oxygenation in the body.
- Any mismatch in the ratio can result in hypoxia.
- When the V/Q ratio is less than normal this is called physiologic shunt (a certain fraction of the venous blood is passing through the pulmonary capillaries without being oxygenated i.e shunted blood).
- When V/Q is more than normal this is called Physiologic dead space (when the ventilation of some of the alveoli is great but the alveolar blood flow is low, ventilation of these alveoli is wasted).

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Concept of "Physiologic Shunt" (When A/ Is Below Normal)

Whenever A/ is below normal, there is inadequate ventilation to provide the oxygen needed to fully oxygenate the blood flowing through the alveolar capillaries. Therefore, a certain fraction of the venous blood passing through the pulmonary capillaries does not become oxygenated. This fraction is called *shunted blood*. Also, some additional blood flows through bronchial vessels rather than through alveolar capillaries, normally about 2 percent of the cardiac output; this, too, is unoxygenated, shunted blood.

Concept of ""Physiologic Dead Space" (When A/ Is Greater thanNormal)

When ventilation of some of the alveoli is great but alveolar blood flow is low, there is far more available oxygen in the alveoli than can be transported away from the alveoli by the flowing blood. Thus, the ventilation of these alveoli is said to be *wasted*. The ventilation of the anatomical dead space areas of the respiratory passageways is also wasted. The sum of these two types of wasted ventilation is called the *physiologic dead space*.

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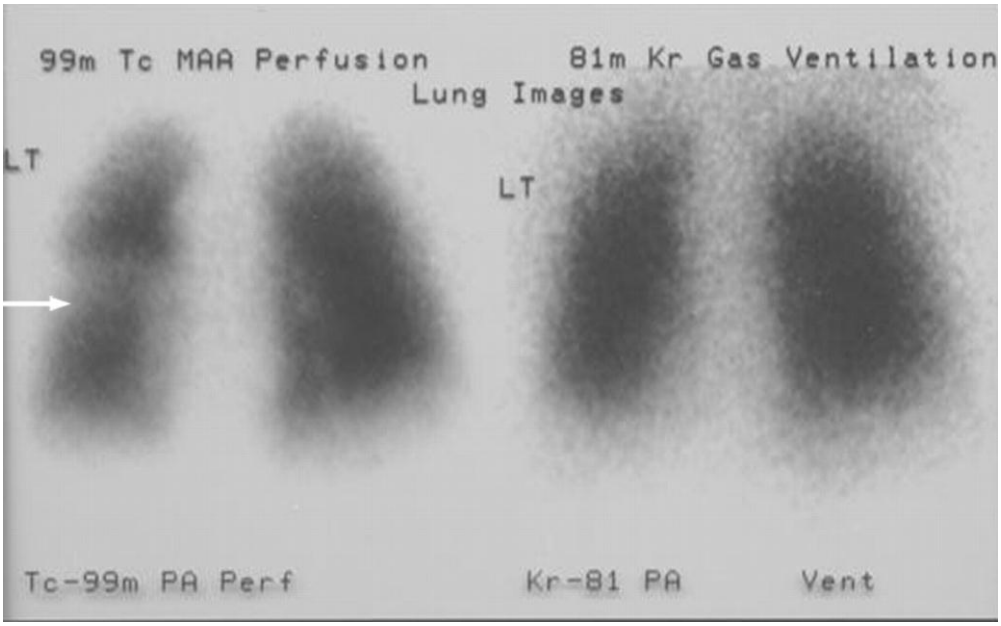
Ventilation- Perfusion Lung Scan



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Abnormalities of the V/Q ratio

- In the Upper and Lower normal lung
  apex V/Q ratio = 3 (moderate degree of physiologic dead space)
  base V/Q ratio= 0.6 (represent a physiologic shunt).
- In Chronic Obstructive Lung disease COPD.
  because of bronchial obstruction in some areas and destruction of the alveolar septa in other areas with patent alveoli those people has some areas of the lung exhibit serious physiologic shunt and other areas serious physiologic dead space.

  COPD is the most prevalent cause of pulmonary disability today, lung effectiveness as a gas exchange organ may decrease

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Abnormal A/ in Chronic Obstructive Lung Disease.

Most people who smoke for many years develop various degrees of bronchial obstruction; in a large share of these persons, this condition eventually becomes so severe that they develop serious alveolar air trapping and resultant emphysema. The emphysema in turn causes many of the alveolar walls to be destroyed. Thus, two abnormalities occur in smokers to cause abnormal A/ First, because many of the small bronchioles are obstructed, the alveoli beyond the obstructions are unventilated, causing a A/ that approaches zero. Second, in those areas of the lung where the alveolar walls have been mainly destroyed but there is still alveolar ventilation, most of the ventilation is wasted because of inadequate blood flow to transport the blood gases.

Thus, in chronic obstructive lung disease, some areas of the lung exhibitserious physiologic shunt, and other areas exhibit serious physiologic dead space. Both conditions tremendously decrease the effectiveness of the lungs as gas exchange organs, sometimes reducing their effectiveness to as little as one-tenth normal. In fact, this is the most prevalent cause of pulmonary disability today.