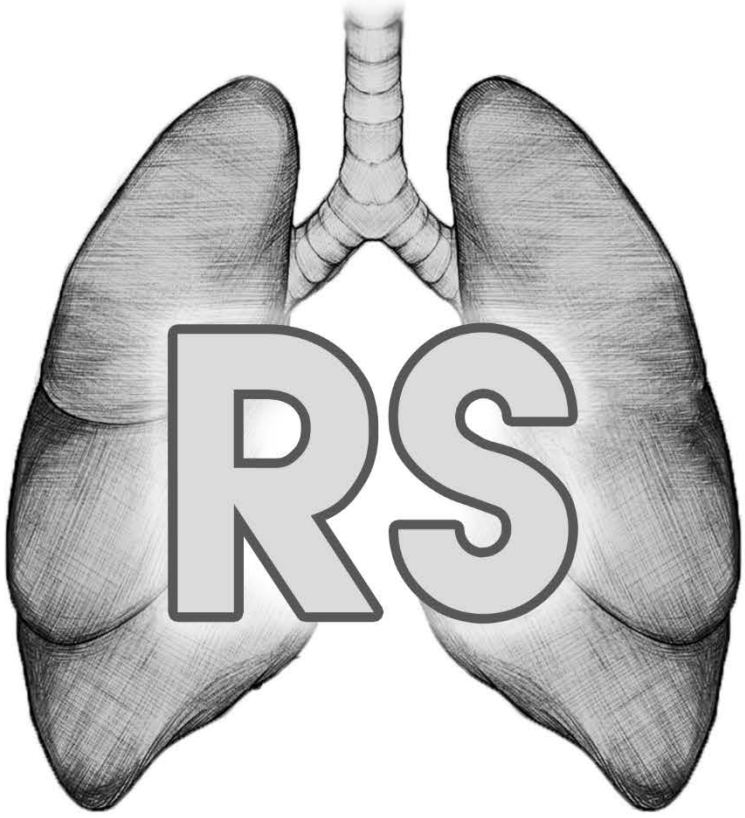

PBL

☑ Sheet ○ Slide ○ Handout

Number

3

Subject

Respiratory Failure

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Date: 00/00/2016

Price:

Before We Start:

- This sheet is written according to the recording of section 2.
- This lecture was discussion-based, but I tried to write the concepts rather than the discussion.
- Concepts are easy, read them quickly and everything would be fine.

- **Respiratory Failure:**
  - Introduction:
    - The main function of the respiratory system is to deliver oxygen to the tissues and to wash out carbon dioxide.
    - Parts of the respiratory system: airways (the conduction system) alveoli of the lungs and, CNS (the control system), respiratory muscles, and chest wall with its ribs and intercostal muscles (pump system).
    - Respiratory failure is the cause of death in many patients with respiratory, chest wall and neurological diseases. That's why we will talk about today.
    - Control of breathing:

      - Peripheral and central chemoreceptors send afferent information to the respiratory center so as to control the rate of breathing.
      - Information sent to the respiratory center are: Pa_{O2}, Pa_{co2}, pH.

An abnormality in any one of these components of the respiratory system may lead to respiratory failure.

Respiratory failure:

Respiratory failure is a syndrome in which the respiratory system fails in one or both of its gas exchange functions: oxygenation and carbon dioxide elimination. In practice, it may be classified as either **hypoxemic** or **hypercapnic**.

Type I Respiratory Failure: Hypoxemia respiratory failure (due to hypoxemia).

Type II Respiratory Failure: Hypercapnic respiratory failure (due to hypercapnia).

- **Hypoxemia**: is defined as a decrease in arterial P_{O2}. However, respiratory failure only occurs when partial pressure of oxygen is inadequate to supply tissues with oxygen.
- You should remember from your basics in physiology that the set-point of P_{O2} control is 60 mm Hg and this is based on the shape of oxygen-Hb dissociation curve (this was discussed thoroughly in previous lectures).
- So, only when oxygen drops below 60 mmHg, we may have respiratory failure.
- **Hypercapnia**: is defind as an increase in arterial P_{CO2}.

- Hypercapnic respiratory failure occurs when partial pressure of CO_2 is above 50 mm Hg.

- **Hypoxemic respiratory failure (type I):**

- Characterized by an arterial oxygen tension (PaO_2) lower than 60 mm Hg with a normal or low arterial carbon dioxide tension ($PaCO_2$).
- This is the most common form of respiratory failure, and it can be associated with virtually all acute diseases of the lungs.

We said that the respiratory system is composed of the lungs, the control system and the pump system.

- Here, we are talking about diseases of the lungs (ex: ARDS, pulmonary fibrosis).

- These diseases occur when oxygen is unable to reach the blood sufficiently.
- Hypoxemia respiratory failure due to ARDS is severe and refectory to oxygen.

- **Hypercapnic respiratory failure (type II):**

These occur when we are not able to get rid of CO_2 as much as needed. Respiratory pump diseases cause this (ex: myasthenia gravis, poliomyelitis, all motor neuron diseases, advanced COPD).

- Many patients with motor neuron diseases die of respiratory failure (ex: Amyotrophic lateral sclerosis, Duchenne muscular dystrophy).

Other causes:
- Hypokalemia

- Disturbances in the control system: morphine overdose.
Morphine is used as a pain-killer in cancer patients. It's also used for COPD in some countries (but not in Jordan) for palliative care and management of dyspnea.

Summary: Lung diseases cause hypoxemia respiratory failure, while pump system and control system diseases cause hypercapnic respiratory failure.

What are the mechanisms of hypoxemia?

1- Hypoventilation:

Alveolar ventilation = $(V_T - V_D)$ x RR

Hypoventilation is either a decrease in tidal volume or RR. Any disease that decreases the tidal volume or the RR will cause hypoventilation. This occurs in diseases of the control system and the pump system (neural diseases that we talked about, morphine overdose).

2- V/Q mismatch

V/Q mismatch is the most common cause of hypoxemia. Alveolar units may vary from low-V/Q to high-V/Q in the presence of a disease process. The low-V/Q units contribute to hypoxemia and hypercapnia, whereas the high-V/Q units waste ventilation but do not affect gas exchange unless the abnormality is quite severe.

The low V/Q ratio may occur either from a decrease in ventilation secondary to airway or interstitial lung disease or from overperfusion in the presence of normal ventilation. The overperfusion may occur in case of pulmonary embolism, where the blood is diverted to normally ventilated units from regions of lungs that have blood flow obstruction secondary to embolism.

Administration of 100% oxygen eliminates all of the low-V/Q units, thus leading to correction of hypoxemia. Hypoxemia increases minute ventilation by chemoreceptor stimulation, but the PaCO2 generally is not affected.

- The most common cause of hypoxemia in patients with COPD is V/Q mismatch.

3- Shunt

Shunt is defined as the persistence of hypoxemia despite 100% oxygen inhalation. The deoxygenated blood (mixed venous blood) bypasses the ventilated alveoli and mixes with oxygenated blood that has flowed through the ventilated alveoli, consequently leading to a reduction in arterial blood content.

- This is seen in patients with ARDS (here, the blood-air barrier is destructed, so even with full ventilation, there's no gases exchange). In this case ABGs are shifted toward their values in mixed venous blood.
- That's why patients with ARDS are refractory to oxygen.

4- Diffusion-limitation:
This is not a major cause of hypoxemia. We usually see it in pulmonary fibrosis.

5- Low FiO_2.

Causes: High Altitude

- When do we say that the patient has hypoventilation?

The patient would have low CO_2.

- If a patient has hypercapnia, this means that he has hypoventilation. But, how can we know if he has one of the above-mentioned abnormalities or not?

By knowing the A-a difference.
- Pa_{O2} is taken from ABGs.
- P_{AO2} is calculated from the following calculation:

$P_{AO2} = P_{IO2} - (P_{ACO2} / R)$

- A-a difference tells you whether alveolar oxygen equilibrates with arterial oxygen or not.

In the following two pages, there's explanation of hypoxemia from Costanzo. I've added them to help you understand the concepts if you didn't get it all. If things are okay, skip them.

This sheet contains each testable point mentioned in the lecture.

Table 5–5 Causes of Hypoxemia

Cause	Pa_{O_2}	A – a Gradient	Supplemental O_2 Helpful?
High altitude (↓ P_B; ↓ PI_{O_2})	Decreased	Normal	Yes
Hypoventilation (↓ PA_{O_2})	Decreased	Normal	Yes
Diffusion defect (e.g., fibrosis)	Decreased	Increased	Yes
$\dot{V}/\dot{Q}$ defect	Decreased	Increased	Yes
Right-to-left shunt	Decreased	Increased	Limited

Acute Altitude Sickness

The initial phase of ascent to high altitude is associated with a constellation of complaints including headache, fatigue, dizziness, nausea, palpitations, and insomnia. The symptoms are attributable to the initial hypoxia and respiratory alkalosis, which abate when the adaptive responses are established.

HYPOXEMIA AND HYPOXIA

Hypoxemia is defined as a decrease in arterial P_{O_2}. Hypoxia is defined as a decrease in O_2 delivery to, or utilization by, the tissues. Hypoxemia is one cause of tissue hypoxia, although it is not the only cause.

Hypoxemia

Hypoxemia, a **decrease in arterial P_{O_2}**, has multiple causes, which are summarized in Table 5-5.

One useful tool for comparing the various causes of hypoxemia is the A – a gradient, or A – a difference. The **A – a gradient** is the difference between the P_{O_2} of alveolar gas (PA_{O_2}) and the P_{O_2} of systemic arterial blood (Pa_{O_2}). As explained earlier in this chapter, in this context, "A" stands for alveolar P_{O_2} and "a" stands for systemic arterial P_{O_2}.

$$\text{A – a gradient} = PA_{O_2} - Pa_{O_2}$$

PA_{O_2} is calculated with the alveolar gas equation and substituted as follows:

$$\text{A – a gradient} = \left(PI_{O_2} - \frac{PA_{CO_2}}{R}\right) - Pa_{O_2}$$

Briefly, the A – a gradient describes whether there has been equilibration of O_2 between alveolar gas and pulmonary capillary blood (which becomes systemic arterial blood). Normally, O_2 equilibrates across the alveolar-pulmonary capillary barrier and the A – a gradient is close to zero. In some but not all causes of hypoxemia, the A – a gradient is increased, or widened, signifying a defect in O_2 equilibration.

- **High altitude** causes hypoxemia because barometric pressure (P_B) is decreased, which decreases the P_{O_2} of inspired air (PI_{O_2}) and of alveolar air (PA_{O_2}). Equilibration of O_2 across the alveolar/pulmonary capillary barrier is normal, and systemic arterial blood achieves the same (lower) P_{O_2} as alveolar air. Because PA_{O_2} and Pa_{O_2} are nearly equal, the A – a gradient is normal. At high altitude, breathing supplemental O_2 raises arterial P_{O_2} by raising inspired and alveolar P_{O_2}.
- **Hypoventilation** causes hypoxemia by decreasing alveolar P_{O_2} (less fresh inspired air is brought into alveoli). Equilibration of O_2 is normal, and systemic arterial blood achieves the same (lower) P_{O_2} as alveolar air. PA_{O_2} and Pa_{O_2} are nearly equal, and the A – a gradient is normal. In hypoventilation, breathing supplemental O_2 raises arterial P_{O_2} by raising the alveolar P_{O_2}.
- **Diffusion defects** (e.g., fibrosis, pulmonary edema) cause hypoxemia by increasing diffusion distance or decreasing surface area for diffusion. Equilibration of O_2 is impaired, Pa_{O_2} is less than PA_{O_2}, and the A – a gradient is increased, or widened. With diffusion defects, breathing supplemental O_2 raises arterial P_{O_2} by raising alveolar P_{O_2} and increasing the driving force for O_2 diffusion.
- **$\dot{V}/\dot{Q}$ defects** always cause hypoxemia and increased A – a gradient. Recall that $\dot{V}/\dot{Q}$ defects usually present as a constellation of abnormalities that may include regions of dead space, high $\dot{V}/\dot{Q}$, low $\dot{V}/\dot{Q}$ and shunt. Recall also that high $\dot{V}/\dot{Q}$ regions have a high P_{O_2} and low $\dot{V}/\dot{Q}$ regions have a low P_{O_2}. The question may then arise: *In $\dot{V}/\dot{Q}$ defects, why don't regions of high $\dot{V}/\dot{Q}$ compensate for regions of low $\dot{V}/\dot{Q}$ so that the P_{O_2} of blood leaving the lungs is normal?* The answer is that while high $\dot{V}/\dot{Q}$ regions have blood with a high P_{O_2}, blood flow to those regions is low (i.e., high $\dot{V}/\dot{Q}$ ratio) and contributes little to total blood flow. Low $\dot{V}/\dot{Q}$ regions, where P_{O_2} is low, have the highest blood flow and the greatest overall effect on P_{O_2} of blood leaving

the lungs. In $\dot{V}/\dot{Q}$ defects, supplemental O_2 can be helpful, primarily because it raises the P_{O_2} of low $\dot{V}/\dot{Q}$ regions where blood flow is highest.

- **Right-to-left shunts** (right-to-left cardiac shunts, intrapulmonary shunts) always cause hypoxemia and increased A – a gradient. Shunted blood completely bypasses ventilated alveoli and cannot be oxygenated (see Fig. 5-27). Because shunted blood mixes with, and dilutes, normally oxygenated blood (nonshunted blood), the P_{O_2} of blood leaving the lungs must be lower than normal. Supplemental O_2 has a limited effect in raising the P_{O_2} of systemic arterial blood because it can only raise the P_{O_2} of normal nonshunted blood; the shunted blood continues to have a dilutional effect. Therefore, the ability of supplemental O_2 to raise the P_{O_2} of systemic arterial blood will depend on the size of the shunt: The larger the shunt, the less effective is supplemental O_2.

  Another feature of treating right-to-left shunts with supplemental O_2 is that it never corrects the increased A – a gradient; in fact, as supplemental O_2 is administered, the A – a increases or widens because $P_{A_{O_2}}$ increases faster than Pa_{O_2} increases.

Hypoxia

Hypoxia is decreased O_2 delivery to the tissues. Because O_2 delivery is the product of cardiac output and O_2 content of blood, hypoxia is caused by decreased cardiac output (blood flow) or decreased O_2 content of blood. Recall that O_2 content of blood is determined primarily by the amount of O_2-hemoglobin. Causes of hypoxia are summarized in Table 5-6.

Table 5–6 Causes of Hypoxia

Cause	Mechanism	Pa_{O_2}
↓ Cardiac output	↓ Blood flow	—
Hypoxemia	↓ Pa_{O_2} ↓ O_2 saturation of hemoglobin ↓ O_2 content of blood	↓
Anemia	↓ Hemoglobin concentration ↓ O_2 content of blood	—
Carbon monoxide poisoning	↓ O_2 content of blood Left shift of O_2-hemoglobin curve	—
Cyanide poisoning	↓ O_2 utilization by tissues	—

A **decrease in cardiac output** and a decrease in regional (local) blood flow are self-evident causes of hypoxia. **Hypoxemia** (due to any cause; see Table 5-5) is a major cause of hypoxia. The reason that hypoxemia causes hypoxia is that a decreased Pa_{O_2} reduces the percent saturation of hemoglobin (see Fig. 5-20). O_2-hemoglobin is the major form of O_2 in blood; thus, a decrease in the amount of O_2-hemoglobin means a decrease in total O_2 content. **Anemia,** or decreased hemoglobin concentration, also decreases the amount of O_2-hemoglobin in blood. **Carbon monoxide (CO) poisoning** causes hypoxia because CO occupies binding sites on hemoglobin that normally are occupied by O_2; thus, CO decreases the O_2 content of blood. **Cyanide poisoning** interferes with O_2 utilization of tissue; it is one cause of hypoxia that does not involve decreased blood flow or decreased O_2 content of blood.

SUMMARY

- Lung volumes and capacities are measured with a spirometer (except for those volumes and capacities that include the residual volume).
- Dead space is the volume of the airways and lungs that does not participate in gas exchange. Anatomic dead space is the volume of conducting airways. Physiologic dead space includes the anatomic dead space plus those regions of the respiratory zone that do not participate in gas exchange.
- The alveolar ventilation equation expresses the inverse relationship between $P_{A_{CO_2}}$ and alveolar ventilation. The alveolar gas equation extends this relationship to predict $P_{A_{O_2}}$.
- In quiet breathing, respiratory muscles (diaphragm) are used only for inspiration; expiration is passive.
- Compliance of the lungs and the chest wall is measured as the slope of the pressure-volume relationship. As a result of their elastic forces, the chest wall tends to spring out and the lungs tend to collapse. At FRC, these two forces are exactly balanced and intrapleural pressure is negative. Compliance of the lungs increases in emphysema and with aging. Compliance decreases in fibrosis and when pulmonary surfactant is absent.
- Surfactant, a mixture of phospholipids produced by type II alveolar cells, reduces surface tension so that the alveoli can remain inflated despite their small radii. Neonatal respiratory distress syndrome occurs when surfactant is absent.
- Airflow into and out of the lungs is driven by the pressure gradient between the atmosphere and the alveoli and is inversely proportional to the resistance of the airways. Stimulation of β_2-adrenergic receptors