Respiratory failure/ ARDS

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Definition

Types

Normal Physiology of Respiration

Pathophysiology of Hypoxemia

Pathophysiology of Hypercapnia

Treatment of Respiratory Failure

ARDS

- Respiratory dysfunction refers to **the failure of gas exchange**, i.e., decrease in arterial oxygen tension, PaO_2, lower than 60 mm Hg (hypoxemia).

respiratory failure

- It may or may not accompany hypercapnia, a $PaCO_2$ higher than 50 mm Hg (decreased CO_2 elimination).

- **Type 1** :Arterial oxygen tension **(PaO_2) lower than 60 mm Hg** with a **normal or low** arterial carbon dioxide tension (**$PaCO_2$)**

- **Type 2**:Hypercapnic respiratory failure is characterized by a **$PaCO_2$ higher than 50** mm Hg and arterial oxygen tension (PaO_2) lower than 60 mm Hg.

Lungs are supplied by tow circulations → Pulmonary circulation : Right side of the heart

Bronchial circulation : Left side of the heart

- Respiratory failure may be further classified as either acute or chronic.

- **Acute respiratory failure** : severe SOB , major drop in O_2

➢ Characterized by life-threatening derangements in arterial blood gases and acid-base status.

➢ **Acute** hypercapnic respiratory failure develops **over minutes to hours**; therefore, pH is less than 7.3.

if acute accumulation of CO_2 → respiratory acidosis → problem in acid-base balance

- **Chronic respiratory failure**: In clinic

➢ Less dramatic and may not be as readily apparent

➢ Develops over **several days or longer,** allowing time for renal compensation and an increase in bicarbonate concentration. Therefore, the pH usually is only slightly decreased.

Compensation of low O2:

➢ The clinical markers of chronic hypoxemia, such as **polycythemia** or **cor-pulmonale**, suggest a long-standing disorder.

↑ Hb production

severe vasoconstriction of the lungs due to chronic hypoxia

vasodilation every where in your body except in the lungs

- The distinction between acute and chronic hypoxemic respiratory failure cannot readily be made on the basis of arterial blood gases.

Cor- pulmonale: right sided heart failure due to a pulmonary disease

Normal Physiology of Respiration

- The **"Alveolar" oxygen tension PAO_2** remains close to 100 mmHg, while alveolar carbon-dioxide tension $PACO_2$ is maintained close to 40 mmHg.

= to blood CO_2

- There is a small difference of 5-10 mmHg between "Alveolar (A)" and "arterial (a)" oxygen tension because around **2% of the systemic cardiac output** bypasses the **pulmonary circulation** (physiologic shunt) and is not oxygenated

↳ normal shunt → from bronchial vein

↳ other normal shunt : Coronary artery

Aa gradiant

- Resulting mix of a small amount of deoxygenated blood makes the PO_2 of arterial blood (PaO_2) slightly lower than that of alveolar air (PAO_2).

- A normal A-a gradient is about < 10 mmHg. If the A-a gradient is normal, it means there is no defect in the diffusion of gases.

- **The A-a gradient** helps to outline the different causes of respiratory failure.

- At steady-state, the rate of **carbon dioxide** production within the body is constant. The $PACO_2$ depends on and is **'inversely proportional'** to the ventilation, so the increased ventilation will lead to decreased $PACO_2$, and decreased ventilation will cause increased $PACO_2$.

- The alveolar oxygen tension, PAO_2, depends on the concentration of **inhaled oxygen** (FIO_2), and alveolar carbon-dioxide tension ($PACO_2$), as in the following equation:

- $$PAO_2 = FIO_2 \times (PB - PH_2O) - PACO_2/R$$

pressure at see level; alveolar CO_2; respiratory-exchange ratio = 0.8; H_2O vapor pressure 47 mm Hg; fractional of inspired air 21%

1. temperature is a variable

PAO_2: Alveolar PO_2

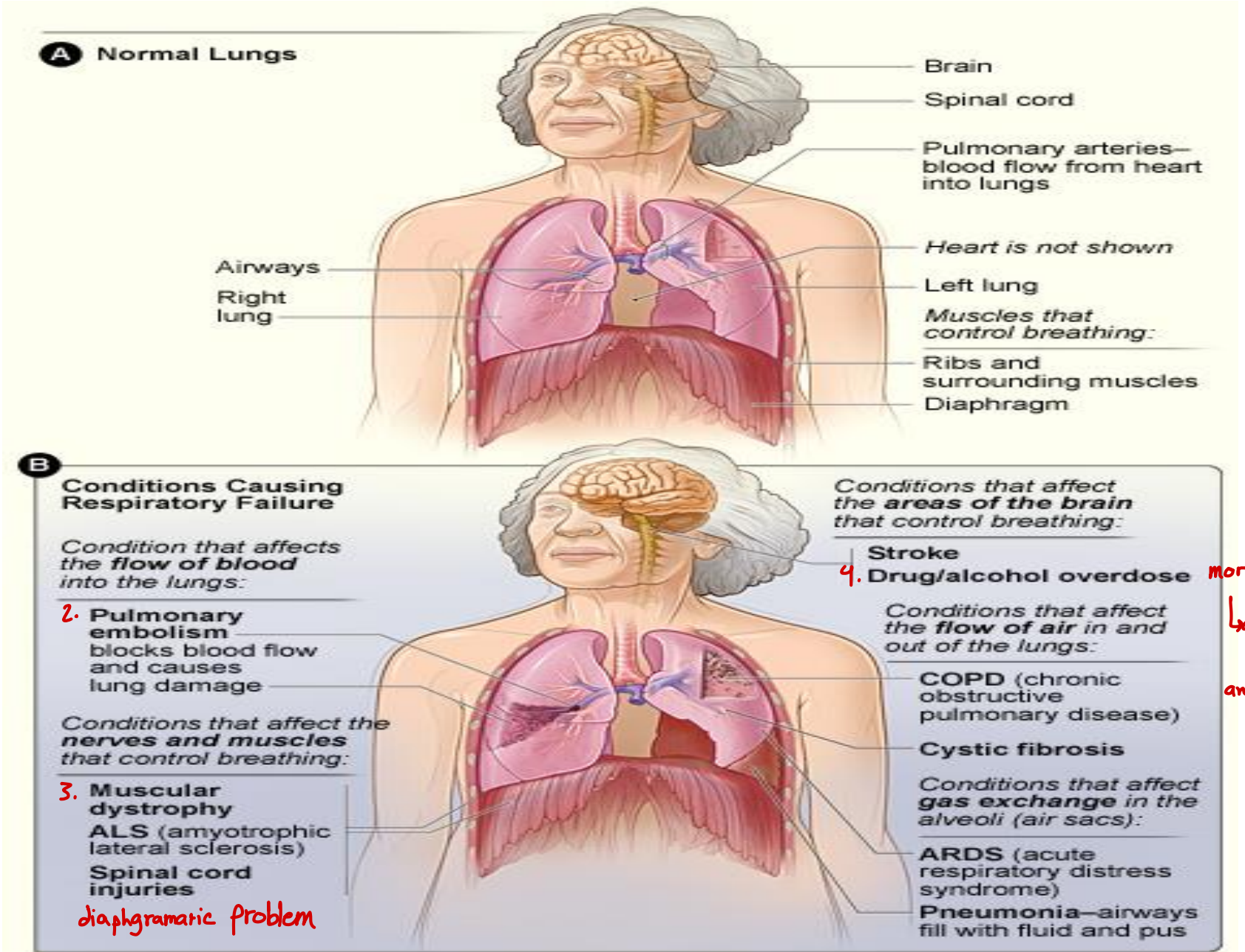
FIO_2: Fractional concentration of oxygen in inspired gas → can change : supply more O_2 to the pt.
PB: Barometric pressure

PH_2O: water vapor pressure at 37°C
$PACO_2$: Alveolar PCO_2

R: Respiratory exchange ratio.

2. altitude → if you go up PB will be low → result in → O_2 in alveola will be low

Pathophysiology of Hypoxemia



Hypoxia can be explained by:

1. central cause

morphine, benzodiazepines

antidote: Nalexone

antidote: Flumazenil

There are five important pathophysiological causes of hypoxemia and respiratory failure.

1. **Diffusion Impairment**
2. **Hypoventilation**
3. **High Altitude**
4. **Pulmonary Shunt**
5. **Ventilation – Perfusion (V/Q) Mismatch**

Pulmonary shunt(right-to-left shunt)

↳ area poorly ventilated with normal perfusion

- The venous deoxygenated blood from the right side enters the left side of the heart and systemic circulation without getting oxygenated within the alveoli.
- So, shunt refers to "**normal perfusion, poor ventilation**."
- The lungs have a normal blood supply, but ventilation is decreased or absent, resulting in failure to exchange gases with the incoming deoxygenated blood.
- The ventilation/perfusion ratio is or near to zero.

O / intact

- **The A-a gradient increases** as deoxygenated blood enter the arterial (systemic) circulation, decreasing the arterial oxygen tension, PaO_2.

- Therefore increasing the oxygen concentration does not correct the hypoxemia. The blood will bypass the lungs, no matter how high the oxygen concentration.

- **This failure to increase PaO_2 after oxygen administration** is a very important point and helps with a differential diagnosis between impaired diffusion and other causes of hypoxemia that resolve with supplemental oxygen.

- For example, in **atelectasis**, the collapsed lung is not ventilated, and the blood within that segment fails to oxygenate. but there is a perfusion

  ↳ Why small atelectasis can improve with giving O_2? O_2 will go to the unaffected areas and improve its oxygenation // but Large atelactasis or bilateral atelactasis → no matter how much you give O_2 it will not improve
- In **cyanotic heart diseases**, the blood from right side bypasses (shunts) the lungs and enters the left side, causing **hypoxemia** and **cyanosis**.

  ↳ no benefit if you give them O_2

ARDS → also an example of shunt: the infectious material will cover the lung, no matter how much you give O_2 there will be a barrier and shunting of blood.

Pneumonia → shunting (if multilobal pneumonia → giving O_2 will not improve it)

Lung circulation

Ventilation – Perfusion (V/Q) Mismatch

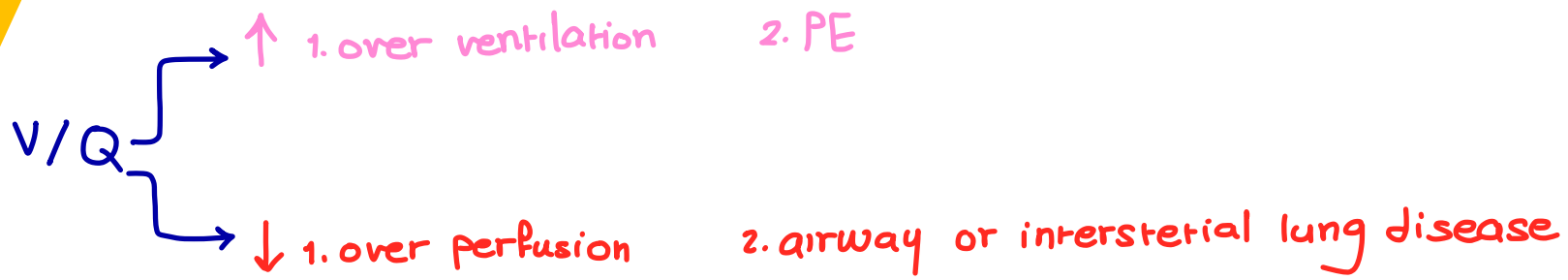
ventilation is usually less than perfusion الأجزاء الغير مستعملة بالرئة

- The **V/Q ratio** in normal individuals is around 0.8, but this ratio alters if there are significant ventilation or perfusion defects.

➢ **The decreased V/Q ratio (< 0.8)** may occur either from decreased ventilation (airway or interstitial lung disease) or from over-perfusion.

- In these cases, the blood is wasted because it fails to properly oxygenate.

- In extreme conditions, when ventilation decreases significantly, and V/Q approaches zero, it will behave as a pulmonary shunt.



- **The increased V/Q ratio (> 0.8)** usually occurs when perfusion is decreased (a pulmonary **embolism** prevents blood flow distal to obstruction) or over-ventilation.

- The air is wasted in these cases and is unable to diffuse within the blood.

- In extreme conditions, when perfusion decreases significantly, and V/Q approaches 1, the alveoli will act as dead space, and no diffusion of gases occurs.

بس هاد ما بصير كتير المريض رح يموت

- Therefore, the increased mismatch in ventilation and perfusion within the lung impairs gas exchange processes, ultimately leading to hypoxemia and respiratory failure.

Diffusion Impairment

→ decreased surface area

↳ increased thickness of alveolar membranes

- There is a structural problem within the lung.

- There may be decreased surface area (as in ① **emphysema**).

منع ventilation & perfusion أما المنطقة نفسها، أو الشبكة جالغشاء،

- Or increased thickness of alveolar membranes (as in ② **fibrosis** and ③ **restrictive lung diseases**) that impairs the diffusion of gases across the alveoli, leading to an increased alveolar-arterial gradient.

higher level of alveolar PO_2 compared to the arterial

- In an **increased A-a gradient**, the alveolar PO_2 will be normal or higher, but arterial PO_2 will be lower. The greater the structural problem, the greater the alveolar-arterial gradient will be.

- Since the diffusion of gases is directly proportional to the concentration of gases; therefore increasing the concentration of inhaled oxygen will correct PaO_2, but the increased A-a gradient will be present as long as the structural problem is present.

High Altitude(Low inspired FiO2)

المشكلة بال supply موجود بال lung

- At high altitudes, the **barometric pressure (PB)** decreases, which will lead to decreased alveolar PO_2 as in the equation: and this leads to hypoxia

volume

- **$PAO_2 = FIO_2 \times (PB - PH_2O) - PACO_2/R$**

- The decreased alveolar PAO_2 will lead to decreased arterial PaO_2 and hypoxemia, but the A-a gradient remains normal since there is no defect within the gas exchange processes. Under these conditions, additional oxygen (increasing the FIO_2) increases the PAO_2 and corrects the hypoxemia.

- When a person **suddenly ascends to the high altitude**, the body responds to the hypoxemia by hyperventilation, causing **respiratory alkalosis**. The concentrations of **2, 3-diphosphoglycerate** (DPG) are increased, shifting the oxygen-hemoglobin dissociation curve to the right.

- Chronically, the acclimatization takes place, and the body responds by increasing the oxygen-carrying capacity of the blood (**polycythemia**). The kidneys excrete bicarbonates and maintain the pH within normal limits.

→ as a reflex of chronic hypoxia

Hypoventilation

$CO_2 \uparrow$ → hypoventilation [if AA normal → pure hypoventilation]

if AA gradiant is high → hypoventilation + other mechanism

& $CO_2 \uparrow$

muscle fatigue

- The minute ventilation depends on the respiratory rate and the **tidal volume**, which is the amount of inspired air during each normal breath at rest.

V_E = RR x V_T

- **Minute ventilation = Respiratory rate x Tidal volume**

↓ in minute ventilation

Low O_2 and ↑ in CO_2

if RR reduced by: medication, fracture flial chest

if tidal volume decreased by: airway obstruction, muscle weakness

- The normal respiratory rate is about 12 breaths per minute, and the normal tidal volume is about 500 mL. Therefore, the minute respiratory volume normally averages about 6 L/min.

less → hypoventilation

more → hyperventilation

- Occurs when there is a **decrease** in the respiratory rate and/or tidal volume so that a lower amount of air is exchanged per minute.

- There will be decreased oxygen entry within the alveoli and the arteries, leading to decreased PaO_2.

- The $PaCO_2$ is inversely proportional to the ventilation. Hence, **<u>hypoventilation will lead to increased $PaCO_2$</u>.**

- The **alveolar-arterial gradient** will **be normal and** less than 10 mmHg since there is no defect in the diffusion of gases. In these cases, increasing the ventilation and/or increasing the oxygen concentration will correct the deranged blood gases. for a limit → you may use P-PaP

Causes of Hypoxemia			
Cause	**PaO_2**	**A-a gradient**	**PaO_2 response to supplemental oxygen**
Hypoventilation	Decreased	Normal ↑ CO_2	Increases
Diffusion Impairment	Decreased	Increased	Increases
Shunt	Decreased	Increased	Does not increase.
V/Q Mismatch	Decreased	Increased	Usually increases (depends on V/Q mismatch type)
High Altitude	Decreased	Normal normal CO_2	Increases

Pathophysiology of Hypercapnia

- **Hypercapnia** occurs when carbon-dioxide tension (PCO_2) increases to more than 50 mmHg. As explained above, at a steady-state,

- The rate of carbon dioxide production within the body is constant.

- The $PACO_2$ depends on and is inversely proportional to ventilation, so decreased ventilation will cause increased $PACO_2$ and vice versa.

hypoventilation

$$PaCO_2 = VCO_2 \times K/V_A$$

K → constant = 863 mm Hg

V_A → alveolar ventilation: the part of minute ventilation which participate in gas exchange

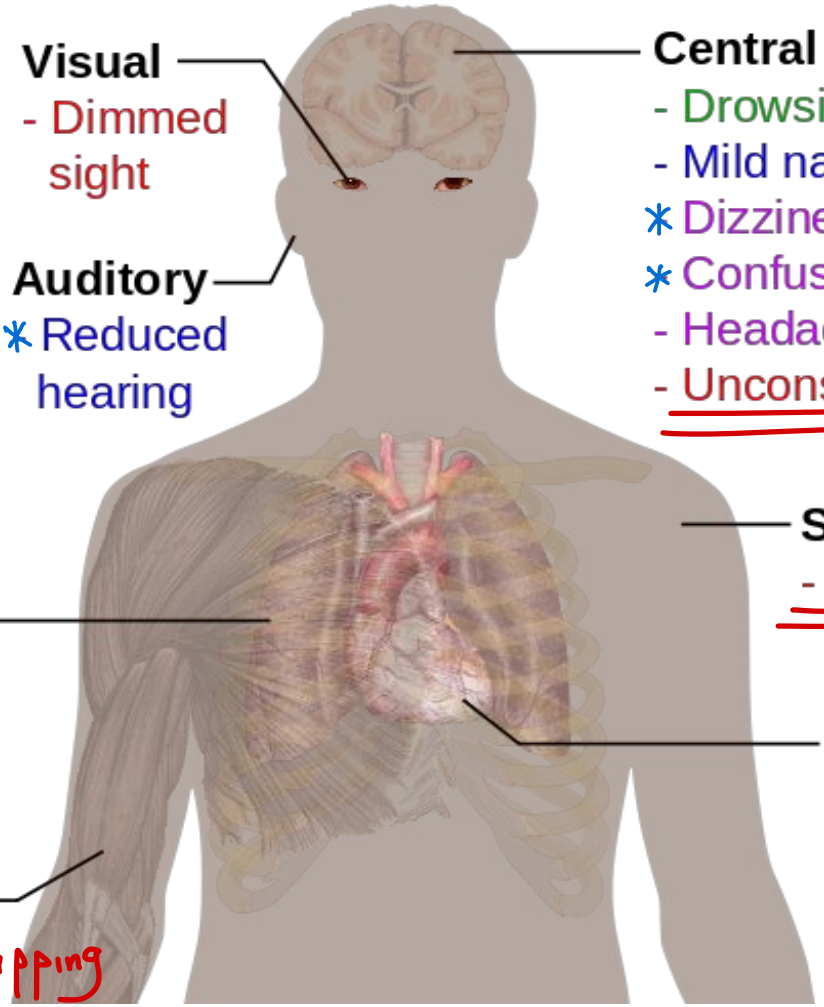
VCO_2 → CO_2 production (from lactate buffering by bicarbonate)

V_A = alveolar volume = (tidal volume − dead space volume) × respiratory frequency

- Therefore, hypercapnia (along with **hypoxemia**, Type II respiratory failure) occurs, usually due to conditions that decrease ventilation.

Main symptoms of

Carbon dioxide toxicity



Volume % in air

- 1%
- 3%
- 5%
- 8%

severe signs of CO_2 narcosis

Visual
- Dimmed sight

Auditory
* Reduced hearing

Central
- Drowsiness
- Mild narcosis
* Dizziness
* Confusion
- Headache
- Unconsciousness

Skin
- Sweating

Respiratory
- Shortness of breath

Heart
- Increased heart rate and blood pressure

Muscular
- Tremor Flapping

↳ any pt. with unconsciousness you have to do an ABGs test for him:
1. if CO_2 ↑ → CO_2 narcosis
2. might be drug overdose
3. " " massive stroke
4. " " ARTN Hial chest

Causes of hypoventilation:

- Multiple sclerosis
- Stroke → CO_2 retention
- Arnold–Chiari malformation

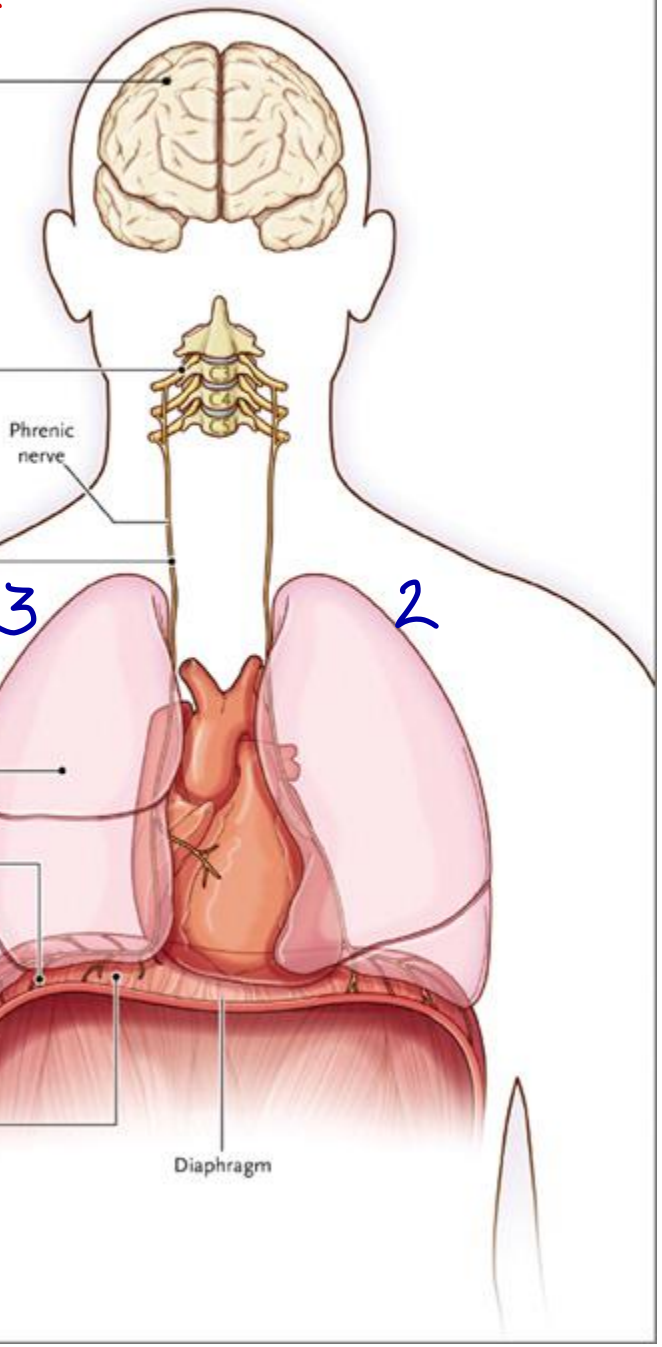
- Quadriplegia
- Amyotrophic lateral sclerosis
- Poliomyelitis
- Spinal muscular atrophy
- Syringomyelia

- Guillain–Barré syndrome
- Tumor compression
- Neuralgic neuropathy
- Critical-illness polyneuropathy
- Chronic inflammatory demyelinating polyneuropathy
- Charcot–Marie–Tooth disease
- Idiopathic

- Hyperinflation (COPD, asthma)

- Myasthenia gravis
- Lambert–Eaton syndrome
- Botulism
- Organophosphates
- Drugs

- Muscular dystrophies
- Myositis (infectious, inflammatory, metabolic)
- Acid maltase deficiency
- Glucocorticoids
- Disuse atrophy



Treatment of Respiratory Failure

- Patients with acute respiratory failure have an increased risk of **hypoxic tissue damage** and should be admitted to a respiratory/intensive care unit.

- The patient's airway, breathing, and circulation (ABCs) must be assessed and managed first, similar to all emergencies.
  - ↳ Airway
  - ↳ Breathing
  - ↳ Circulation

- The first goal is to correct hypoxemia and/or prevent tissue hypoxia by maintaining an arterial oxygen tension (PaO_2) of 60 mm Hg or arterial oxygen saturation (SaO_2) greater than 90%.

if the pt. is CO_2 retainer → you keep SaO_2 btw 80–92%
We don't want to over oxygenate him bcs this will cause more CO_2 retention

if the pt. is not CO_2 retainer → keep it btw 92 - 96%

- Usually, initially providing supplemental oxygen and mechanical ventilation, which is provided by facial mask (non-invasive) or by tracheal intubation, is effective.

  ↳you bypass the obstruction, you overcome the muscle weakness

  ↳if consnousniss level is less than 10 → invasive

- Specific respiratory failure treatment **depends on the underlying cause.**

  ↳it's not a diagnosis, it's a reflection of other process : e.g., infection pneumonia → treat by antibiotics, Asthma → steroid & bronchodilator, PE → heparin or thrombolytic therapy
  COPD → " "

- Therefore, we should try to identify the underlying pathophysiologic disturbances that led to respiratory failure and correct them by providing specific treatment, such as **steroids** and **bronchodilators** for COPD and asthma, **antibiotics** for pneumonia, and **heparin** for **pulmonary embolism.**

Acute respiratory distress syndrome (ARDS)

- A rapidly progressive **noncardiogenic pulmonary edema** that initially manifests as dyspnea, tachypnea, and hypoxemia, then quickly evolves into respiratory failure.

↳ mechanism: shunting

- **These criteria** are based on timing of symptom onset (within one week of known clinical insult or new or worsening respiratory symptoms)

How to diagnose ARDS?

1 Bilateral opacities on chest imaging that are not fully explained by effusions, lobar or lung collapse, or nodules;

2 The likely source of pulmonary edema (respiratory failure not fully explained by cardiac failure of fluid overload);

3 Oxygenation as measured by the ratio of partial pressure of arterial oxygen (Pao2) to fraction of inspired oxygen (Fio2).

PF ratio

$PaO_2 / FiO_2 = P/F\ ratio$

if room air → $FiO_2 = 0.21$

if you give the pt. 50% $O_2 = 0.5$

Severity

PF ratio:

- normal : > 300

- **Mild**: 200 mm Hg < Pao2/Fio2 ratio ≤ 300 mm Hg with positive end-expiratory pressure (PEEP) or continuous positive airway pressure ≥ 5 cm H2O.

- **Moderate**: 100 mm Hg < Pao2/Fio2 ratio ≤ 200 mm Hg with PEEP ≥ 5 cm H2O.

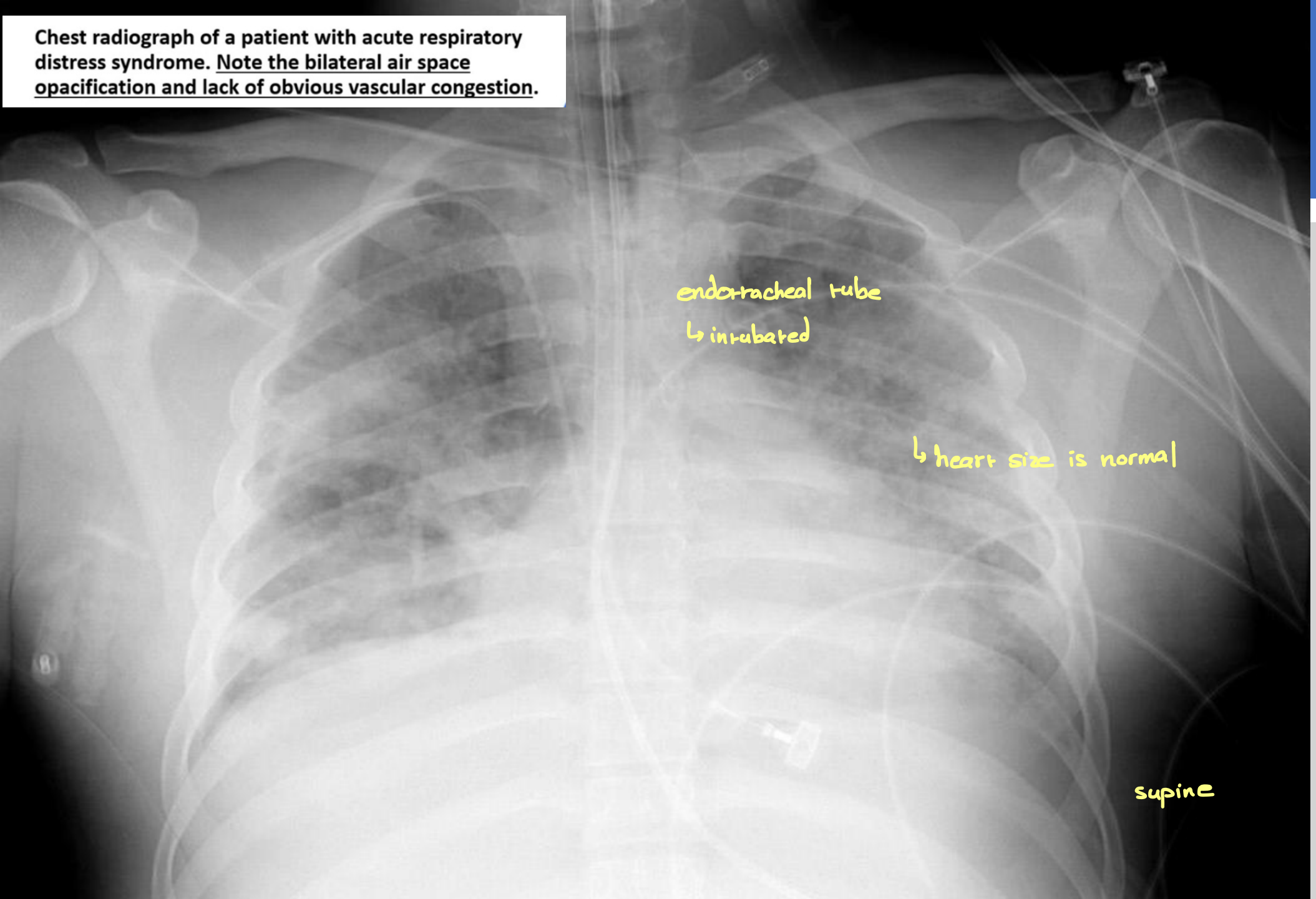
- **Severe**: Pao2/Fio2 ratio ≤ 100 mm Hg with PEEP ≥ 5

cm H2O.

- ARDS often must be differentiated from pneumonia and congestive heart failure, which typically has signs of fluid overload.

- ARDS is responsible for one in 10 admissions to intensive care units and one in four mechanical ventilations. In-hospital mortality for patients with severe ARDS ranges from 46% to 60%.

- Most cases of ARDS in adults are associated with pneumonia with or without sepsis (60%) or with non-pulmonary sepsis (16%).


Chest radiograph of a patient with acute respiratory distress syndrome. Note the bilateral air space opacification and lack of obvious vascular congestion.
endotracheal tube
↳ intubated
↳ heart size is normal
supine

Treatment

- supportive and includes:

- **mechanical ventilation**, prophylaxis for stress ulcers and venous thromboembolism, nutritional support, and treatment of the underlying injury.

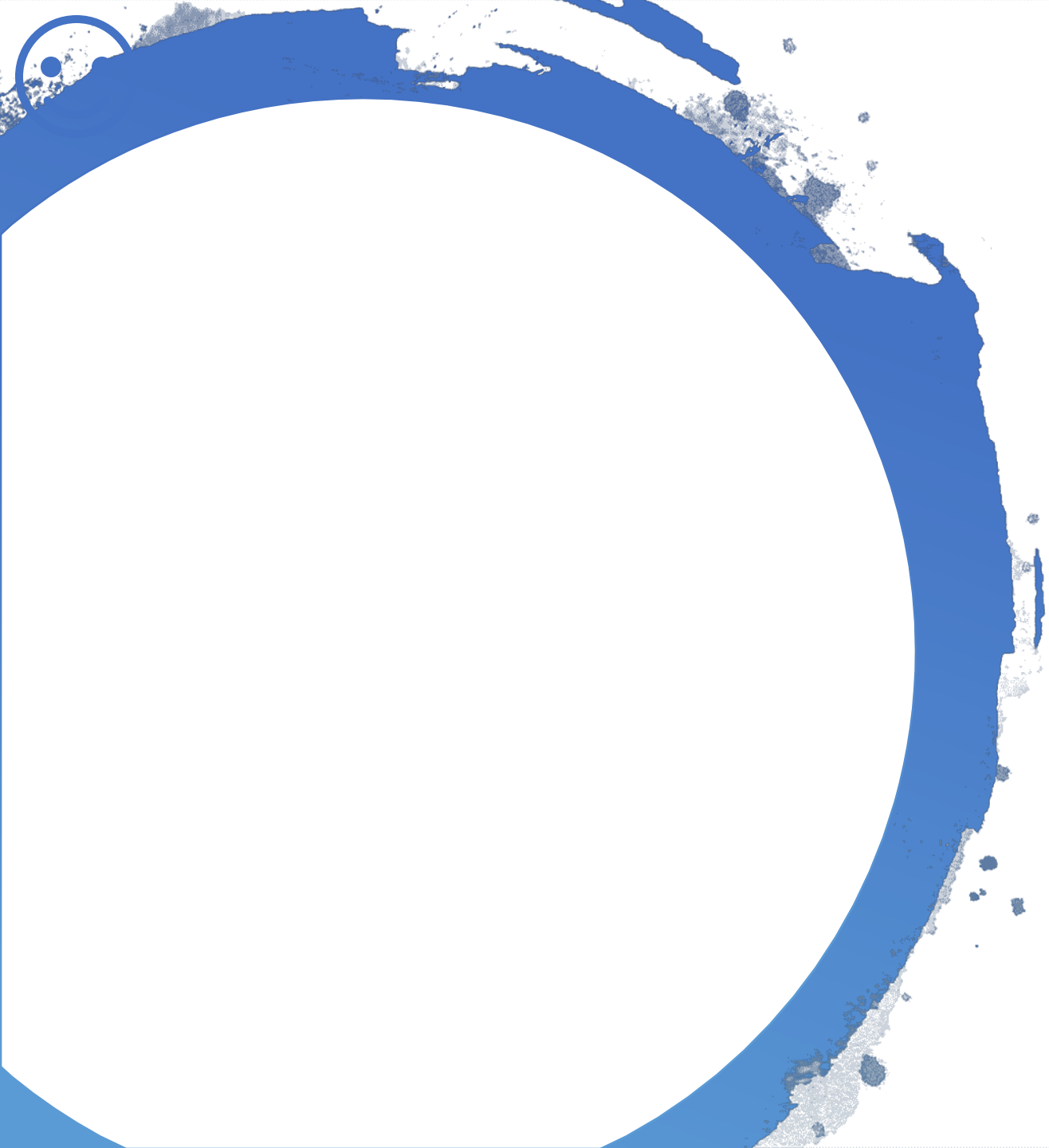
  O2 usually is not effective

- Low tidal volume and high positive end-expiratory pressure improve outcomes.

- Prone positioning is recommended for some moderate and all severe cases.

  بتنوم المريض على بطنه وبتعمله ventilation

- As patients with ARDS improve and the underlying illness resolves, a spontaneous breathing trial is indicated to assess eligibility for ventilator weaning.



Thanks