Respiratory failure/ ARDS

By Dr Khaled Al Oweidat

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).

- 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.

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

- **Acute respiratory failure** :

➢ 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.

- **Chronic respiratory failure**:

➢ 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.

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

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

Normal Physiology of Respiration

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

- 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

- 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:
- $$\mathbf{PAO_2 = FIO_2 \times (PB - PH_2O) - PACO_2/R}$$

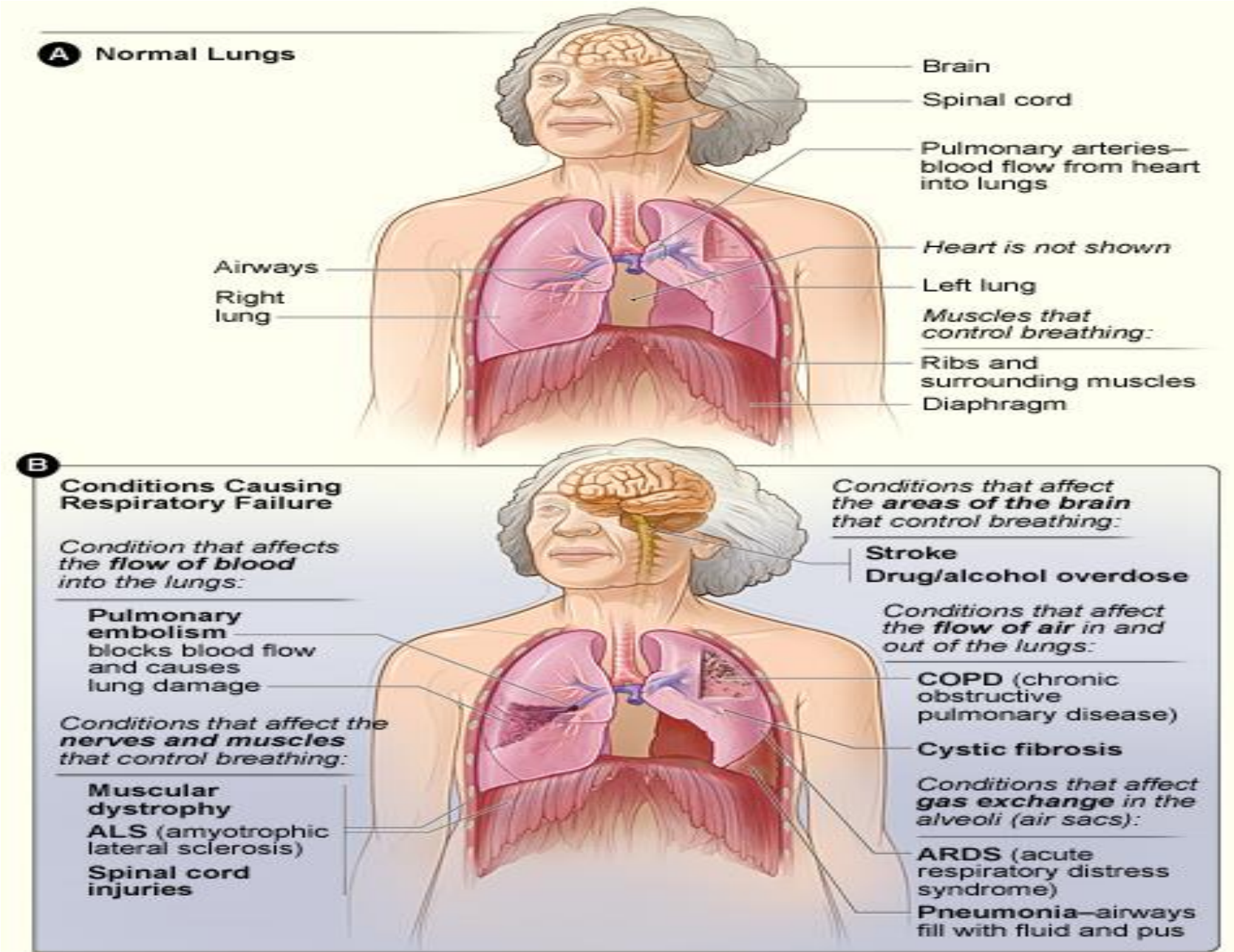
PAO_2: Alveolar PO_2

FIO_2: Fractional concentration of oxygen in inspired gas
PB: Barometric pressure

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

R: Respiratory exchange ratio.

Pathophysiology of Hypoxemia



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)

- 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.

- **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.

- In **cyanotic heart diseases**, the blood from right side bypasses (shunts) the lungs and enters the left side, causing **hypoxemia** and **cyanosis**.

Ventilation – Perfusion (V/Q) Mismatch

- 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.

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

Diffusion Impairment

- There is a structural problem within the lung.
- There may be decreased surface area (as in **emphysema).**
- Or increased thickness of alveolar membranes (as in **fibrosis** and **restrictive lung diseases)** that impairs the diffusion of gases across the alveoli, leading to an increased alveolar-arterial gradient.

- 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)

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

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

- 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.

Hypoventilation

- 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.

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

- 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.

- 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, **hypoventilation will lead to increased $PaCO_2$.**

- 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.

Causes of Hypoxemia			
Cause	**PaO_2**	**A-a gradient**	**PaO_2 response to supplemental oxygen**
Hypoventilation	Decreased	Normal	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	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.

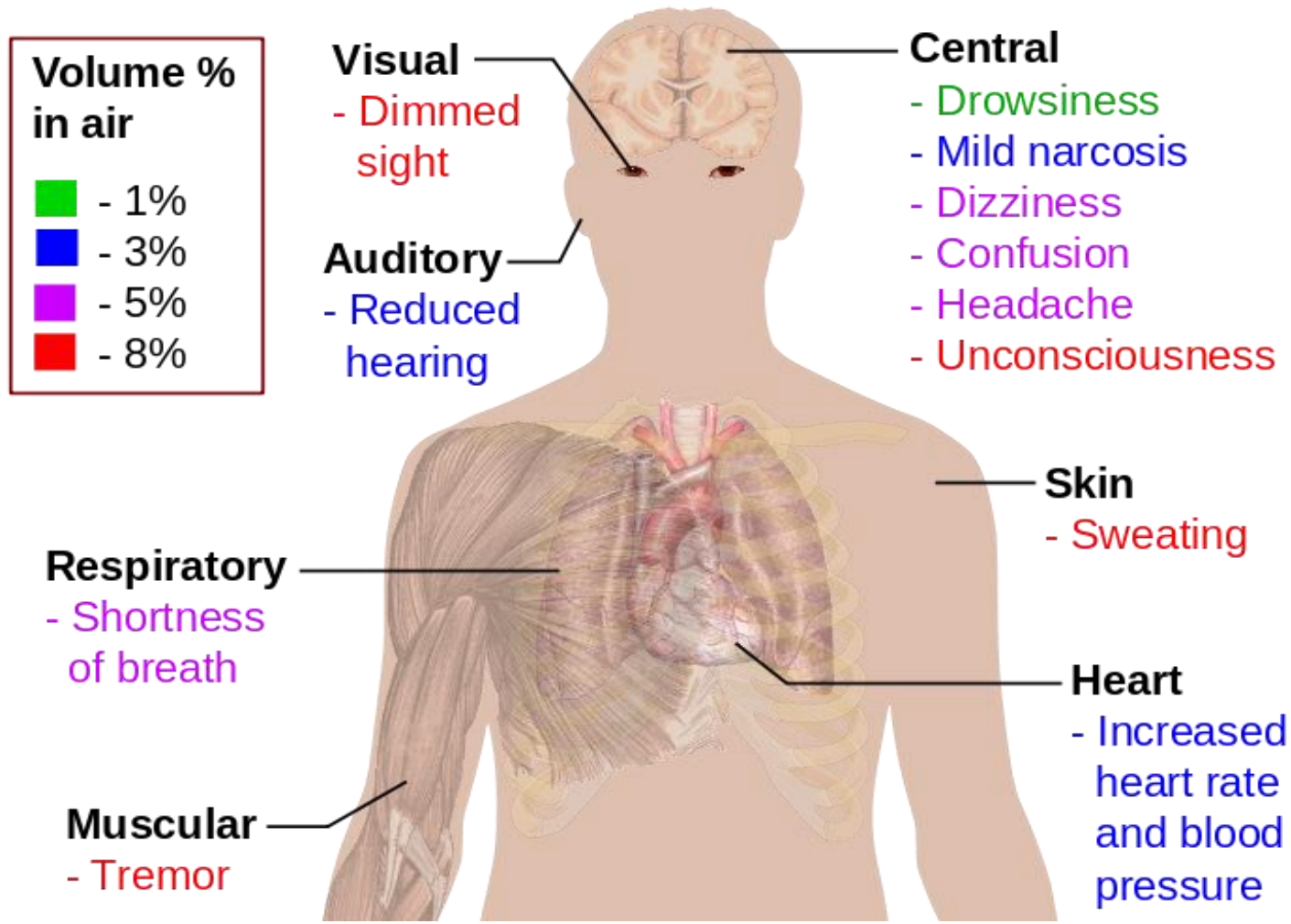
- The $PACO_2$ depends on and is inversely proportional to ventilation, so decreased ventilation will cause increased $PACO_2$ and vice versa.

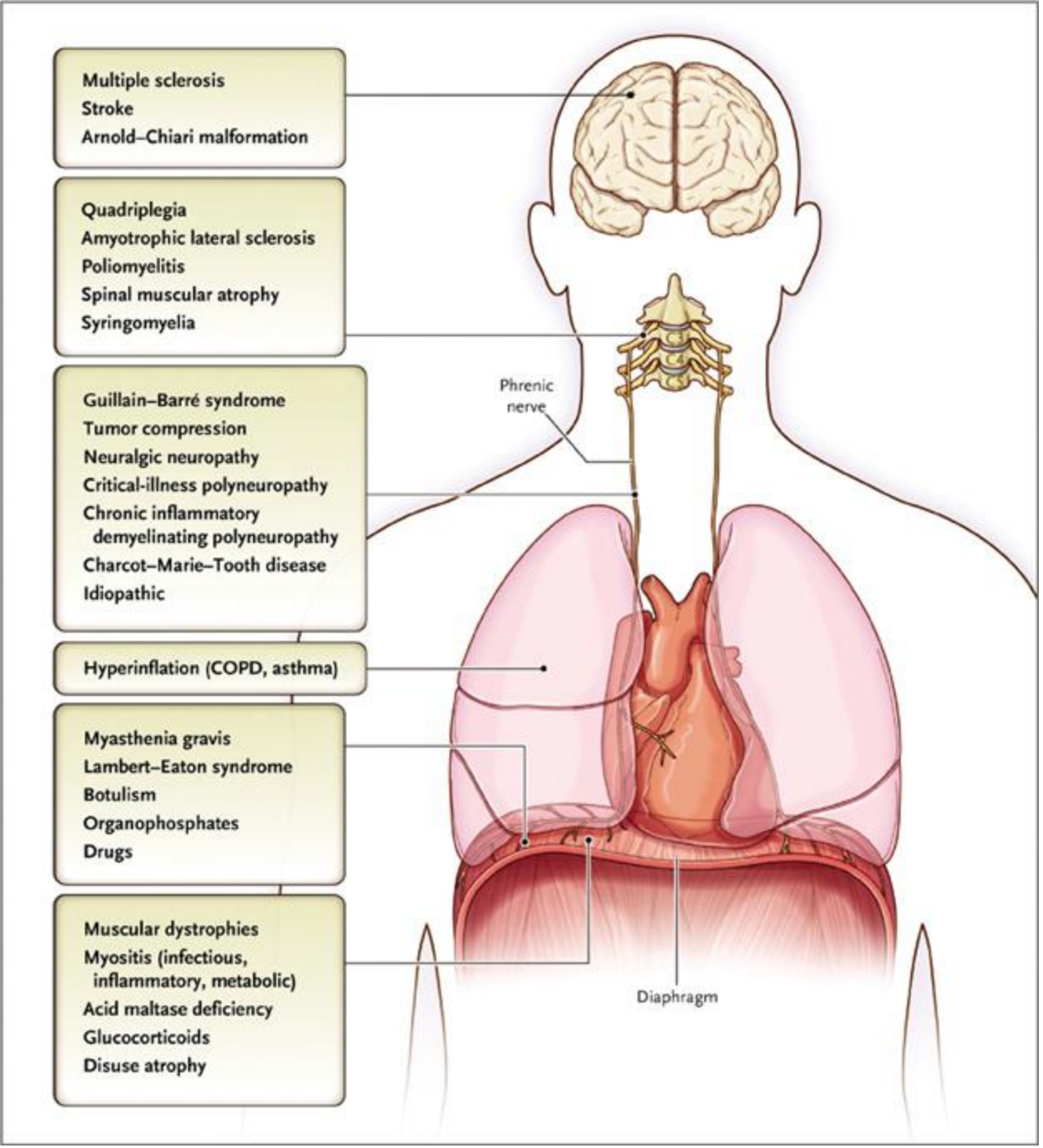
$$PaCO_2 = VCO_2 \times K/V_A$$

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

Main symptoms of

Carbon dioxide toxicity




Multiple sclerosis
Stroke
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
Phrenic nerve
Diaphragm

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.
- 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%.

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

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

- 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.

- **These criteria** are based on timing of symptom onset (within one week of known clinical insult or new or worsening respiratory symptoms)
- Bilateral opacities on chest imaging that are not fully explained by effusions, lobar or lung collapse, or nodules;
- The likely source of pulmonary edema (respiratory failure not fully explained by cardiac failure of fluid overload);
- Oxygenation as measured by the ratio of partial pressure of arterial oxygen (Pao2) to fraction of inspired oxygen (Fio2).

Severity

- **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.

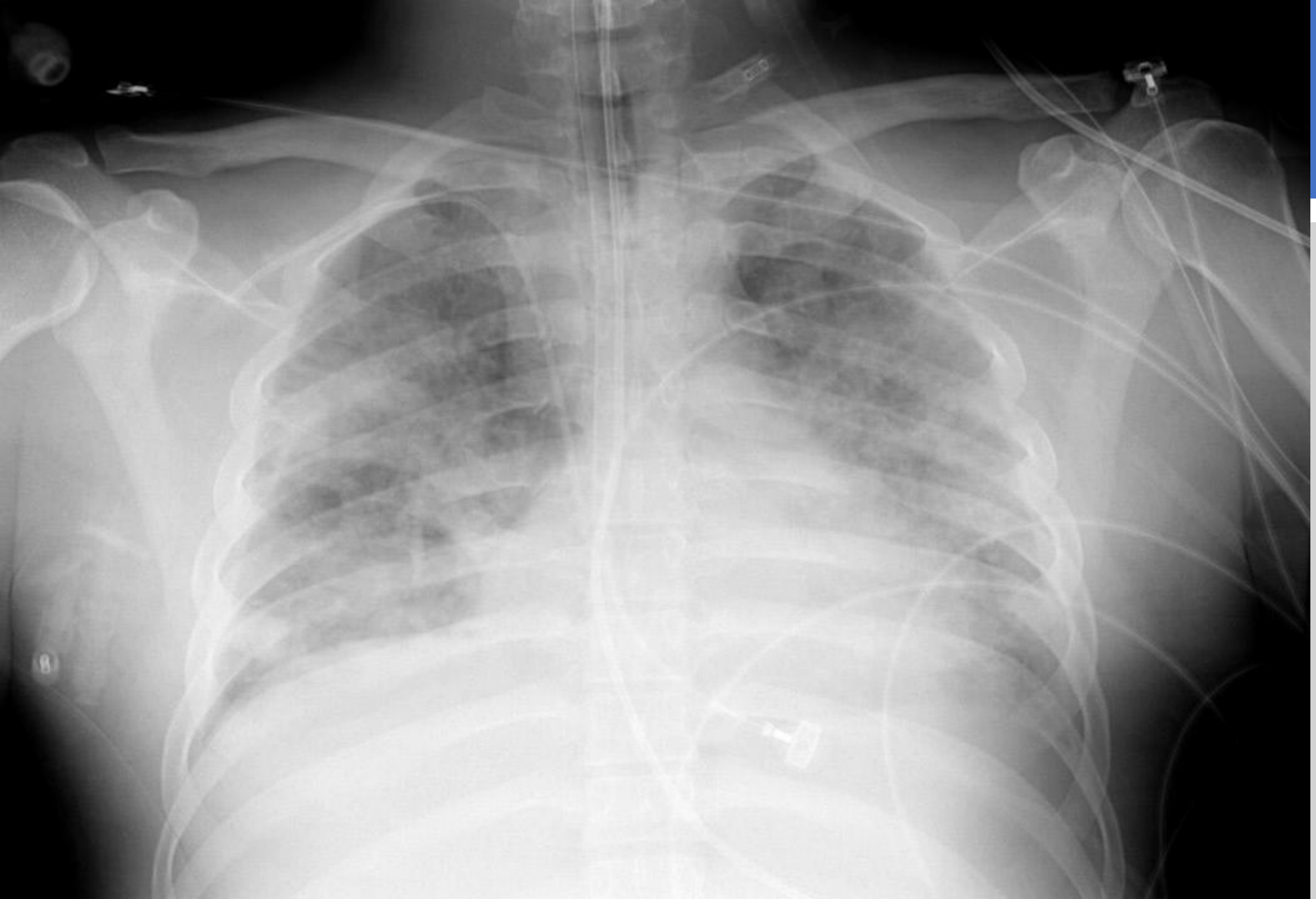
- **Severe**: Pao2/Fio2 ratio ≤ 100 mm Hg with PEEP ≥ 5

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%).



Treatment

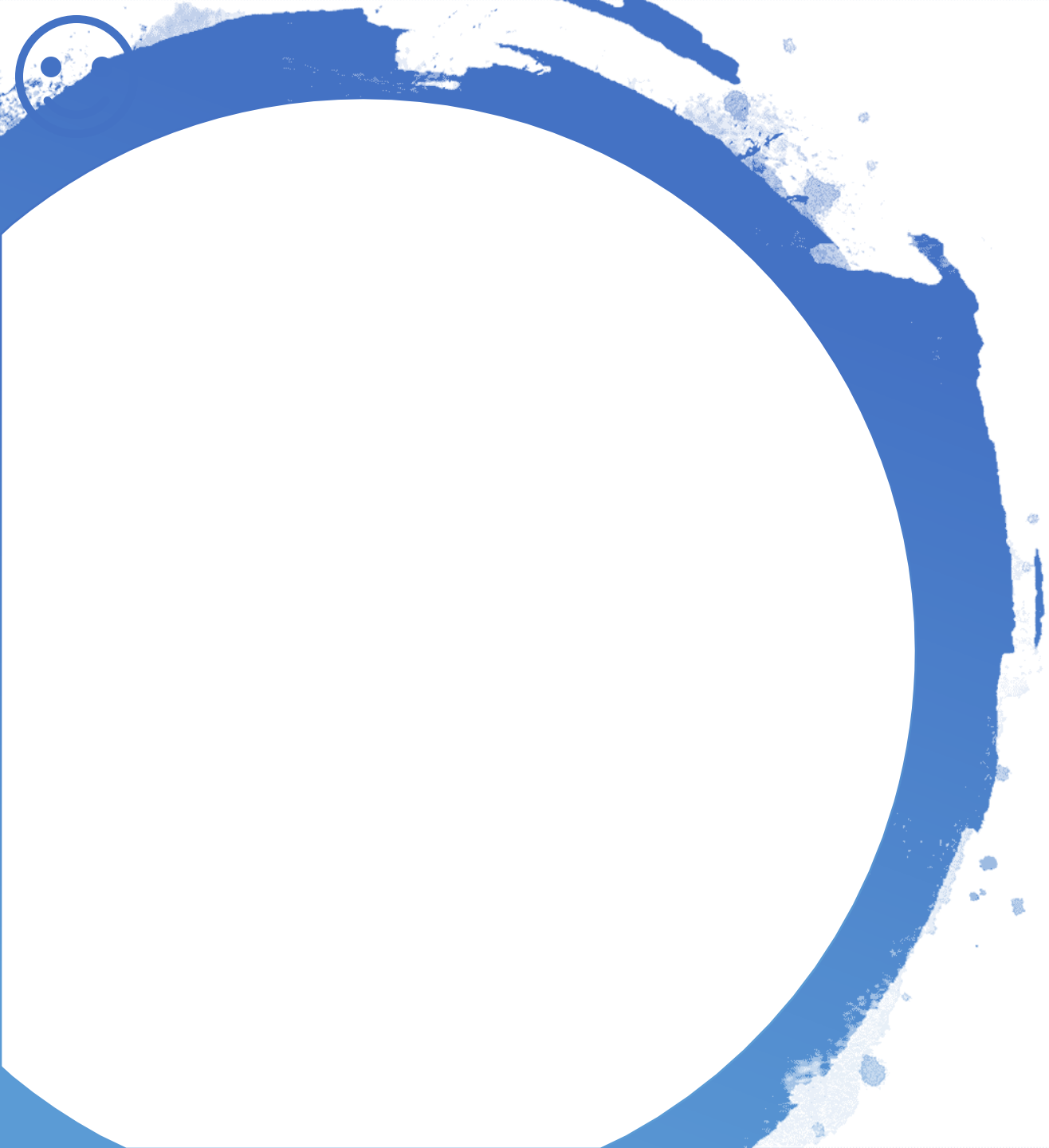
- supportive and includes:

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

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

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

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



Thanks