Respiratory failure

Content

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- Clinical Presentation
- Workup Approach
- Treatment & Management
- Medication & guidelines

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Introduction

Background, pathophysiology and etiology

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Definition

- 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**

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Hypoxemic respiratory failure (type I)

- **Type I** is characterized by an arterial oxygen tension (PaO_2) <60 mm Hg with a normal or low arterial carbon dioxide tension ($PaCO_2$)
- This is the most common form of respiratory failure

- Example:
  - cardiogenic or noncardiogenic pulmonary edema
  - Pneumonia
  - pulmonary hemorrhage

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Hypoxemic respiratory failure (type I)

- Type 1 respiratory failure is defined as a low level of oxygen in the blood (hypoxemia) without an increased level of carbon dioxide in the blood (hypercapnia), and indeed the P_aCO_2 may be normal or low.
- It is typically caused by a ventilation/perfusion (V/Q) mismatch; the volume of air flowing in and out of the lungs is not matched with the flow of blood to the lungs.

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Hypoxemic respiratory failure (type I)

This type of respiratory failure is caused by conditions that affect oxygenation such as:

- **Low ambient oxygen** (e.g. at high altitude)
- **Ventilation-perfusion mismatch** (parts of the lung receive oxygen but not enough blood to absorb it, e.g. pulmonary embolism)
- **Alveolar hypoventilation** (decreased minute volume due to reduced respiratory muscle activity, e.g. in acute neuromuscular disease); this form can also cause type 2 respiratory failure if severe
- **Diffusion problem** (oxygen cannot enter the capillaries due to parenchymal disease, e.g. in pneumonia or ARDS)
- **Shunt** (oxygenated blood mixes with non-oxygenated blood from the venous system, e.g. right to left shunt)

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Hypercapnic respiratory failure (type II)

- **Type II** is characterized by a $PaCO_2$ >50 mm Hg
- Hypoxemia is common in patients with hypercapnic respiratory failure who are breathing room air
- The pH depends on the level of bicarbonate, which, in turn, is dependent on the duration of hypercapnia

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Hypercapnic respiratory failure (type II)

- Type 2 respiratory failure is caused by inadequate alveolar ventilation; both oxygen and carbon dioxide are affected
- Defined as the buildup of carbon dioxide levels ($PaCO_2$) that has been generated by the body but cannot be eliminated

- Common etiologies of type II:
  - drug overdose
  - neuromuscular disease
  - chest wall abnormalities
  - severe airway disorders (eg, asthma and COPD)

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Hypercapnic respiratory failure (type II)

The underlying causes include:

- **Increased airways resistance** (COPD, asthma, suffocation)
- **Reduced breathing effort** (drug effects, brain stem lesion, extreme obesity)
- **A decrease in the area of the lung** available for gas exchange (such as in chronic bronchitis)
- **Neuromuscular problems** (Guillain–Barré syndrome, motor neuron disease)
- Deformed (kyphoscoliosis), rigid (ankylosing spondylitis), or flail chest.

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Prognosis

The mortality associated with respiratory failure varies according to the etiology

- ARDS, mortality is approximately 40-45%
- Younger patients (<60 y) have better survival rates than older patients
- For patients with COPD and acute respiratory failure, the overall mortality has declined from approximately 26% to 10%
- Acute exacerbation of COPD carries a mortality of approximately 30%

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Clinical presentation

History and physical examination findings

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History

- The diagnosis of acute or chronic respiratory failure begins with **clinical suspicion** of its presence
- Confirmation of the diagnosis is based on **arterial blood gas analysis**
- Evaluation of an underlying cause must be initiated early, frequently in the presence of concurrent treatment for acute respiratory failure.
- The cause of respiratory failure is often evident after a careful history and physical examination

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Physical Examination

- **Localized pulmonary findings** reflecting the acute cause of hypoxemia (eg, pneumonia, pulmonary edema, asthma, or COPD)
- **Neurologic manifestations** include restlessness, anxiety, confusion, seizures, or coma
- **Asterixis** may be observed with severe hypercapnia
- **Tachycardia** and a variety of arrhythmias may result from hypoxemia and acidosis

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Physical Examination

- **Cyanosis**, a bluish color of skin and mucous membranes, indicates hypoxemia
- **Dyspnea**, an uncomfortable sensation of breathing, often accompanies respiratory failure
- **Pulmonary hypertension** frequently is present in chronic respiratory failure. Alveolar hypoxemia potentiated by hypercapnia causes pulmonary arteriolar constriction

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Criteria for diagnosis of ARDS

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Criteria for diagnosis of ARDS

- **Clinical presentation** - Tachypnea and dyspnea; crackles upon auscultation
- **Clinical setting** - Direct insult (aspiration) or systemic process causing lung injury (sepsis)
- **Radiologic appearance** - 3-quadrant or 4-quadrant alveolar flooding

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Criteria for diagnosis of ARDS

- **Lung mechanics** - Diminished compliance (<40 mL/cm water)
- **Gas exchange** - Severe hypoxia refractory to oxygen therapy (ratio of arterial oxygen tension to fractional concentration of oxygen in inspired gas [PaO 2/FiO 2] <200)
- **Normal pulmonary vascular properties** - Pulmonary capillary wedge pressure <18 mm Hg

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Respiratory Failure Workup

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Approach Consideration

- Chest **radiography** is essential.
- Pulmonary functions tests (**PFTs**), may be helpful, although more useful in terms of defining recovery potential
- **ECG** should be performed to evaluate the possibility of a cardiovascular cause of respiratory failure; it also may detect dysrhythmias resulting from severe hypoxemia or acidosis
- Right-sided heart **catheterization** is controversial

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Laboratory Studies

1. **ABG** analysis should be performed to confirm the diagnosis and to assist in the distinction between acute and chronic forms

2. **CBC** may indicate anemia, which can contribute to tissue hypoxia, whereas polycythemia may indicate chronic hypoxemic respiratory failure

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Laboratory Studies

3. Abnormalities in **renal and hepatic** function may either provide clues to the etiology of respiratory failure or alert the clinician to complications

4. Abnormalities in **electrolytes** such as K^+, Mg, and phosphate may aggravate respiratory failure and other organ function

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Laboratory Studies

5. Measuring **serum creatine** kinase with fractionation and troponin I helps exclude recent MI in a patient with respiratory failure

6. In chronic hypercapnic respiratory failure, serum levels of **TSH** should be measured to evaluate the possibility of hypothyroidism

   (a potentially reversible cause of respiratory failure)

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Chest X-ray

Chest radiography frequently reveals the cause

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Bilateral airspace infiltrates on chest radiograph film secondary to acute respiratory distress syndrome that resulted in respiratory failure

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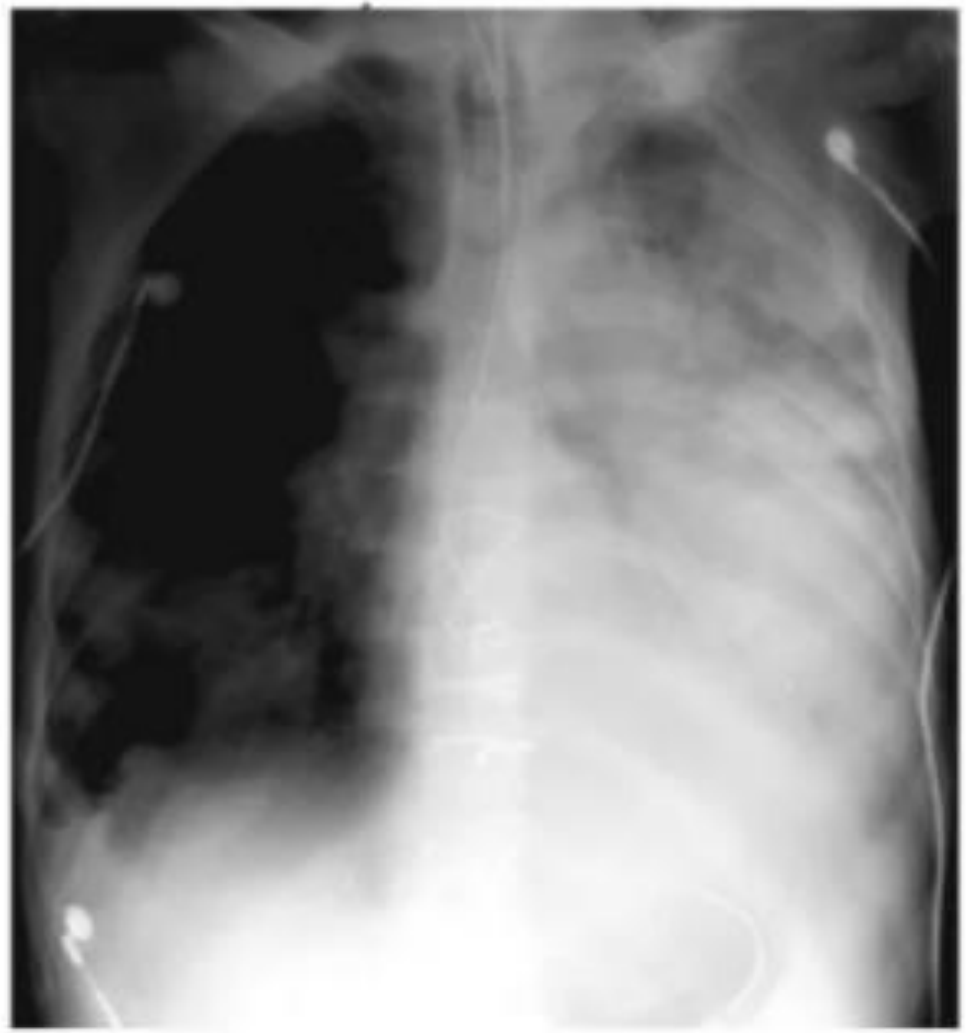

Extensive left-lung pneumonia caused respiratory failure; the mechanism of hypoxia is intrapulmonary shunting

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A 44-year-old woman developed acute respiratory failure and diffuse bilateral infiltrates. She met the clinical criteria for the diagnosis of acute respiratory distress syndrome. In this case, the likely cause was urosepsis

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This patient developed acute respiratory failure that turned out to be the initial presentation of SLE. The lung pathology evidence of diffuse alveolar damage is the characteristic lesion of acute lupus pneumonitis

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Treatment & Management

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Approach Consideration

- Correction of Hypoxemia
- Principles of Mechanical Ventilation
- Ventilation Approaches for Specific Diseases
- Noninvasive Ventilatory Support
- Weaning From Ventilator
- Long-Term Monitoring

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Correction of Hypoxemia

- The first objective in the management of respiratory failure is to reverse and/or prevent tissue hypoxia
- Many experts believe that hypercapnia should be tolerated until the arterial blood pH falls < 7.2

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Correction of Hypoxemia

- Patient with acute respiratory failure generally should be **admitted to ICU**
- Most patients with chronic respiratory failure can be treated at home with oxygen supplementation and/or ventilatory assist devices along with therapy for their underlying disease

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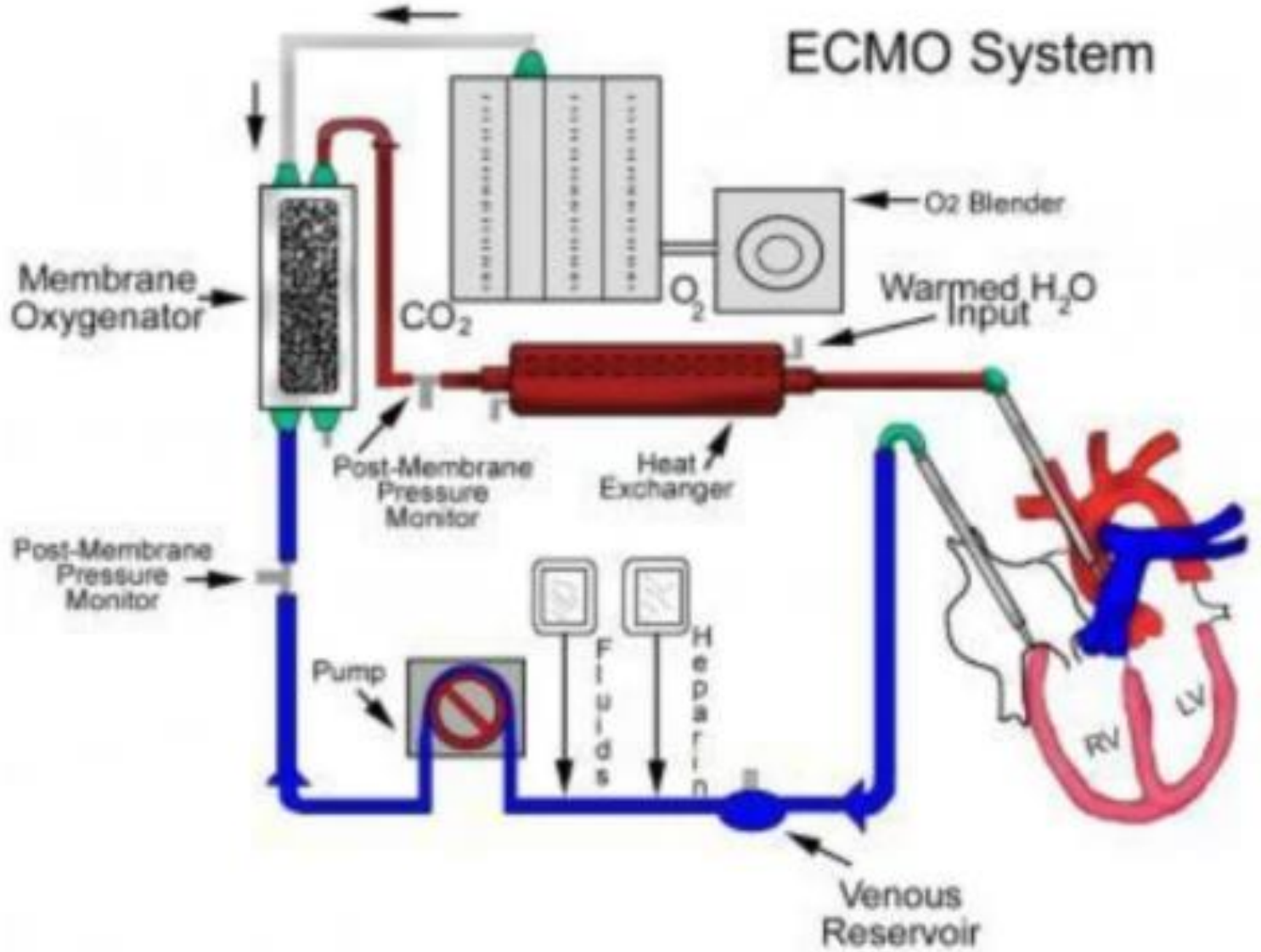
Correction of Hypoxemia



Extracorporeal membrane oxygenation (**ECMO**) may be more effective than conventional management for patients with severe but potentially reversible respiratory failure

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Correction of Hypoxemia

- Once the airway is secured, attention is turned toward correcting the underlying hypoxemia
- The **goal** is to assure adequate oxygen delivery to tissues, generally achieved with an PaO2 of 60 mm Hg or an SaO2 >90%
- **Supplemental oxygen** is administered via nasal prongs or face mask
- In patients with severe hypoxemia, intubation and **mechanical ventilation** are often required

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Mechanical Ventilation

Mechanical ventilation is used for two essential reasons:

1. to increase PaO2 and
2. to lower PaCO2

- Mechanical ventilation also rests the respiratory muscles and is an appropriate therapy for respiratory muscle fatigue

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Ventilation Approaches for Specific Diseases

- The mode of ventilation should be suited to the needs of the patient

- After the initiation of mechanical ventilation, ventilator settings should be adjusted on the basis of:
  - the patient's lung mechanics
  - underlying disease process
  - gas exchange
  - response to mechanical ventilation.

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Ventilation Approaches for Specific Diseases

- **SIMV** and assist control ventilation are often used for the initiation of mechanical ventilation
- In patients with intact respiratory drive and mild-to-moderate respiratory failure, **PSV** may be a good initial choice
- The lowest FiO2 that produces an SaO2 >90% and a PaO2 >60 mm Hg generally is recommended
- The prolonged use of an FiO2 <0.6 is unlikely to cause pulmonary oxygen toxicity

SIMV: Synchronized Intermittent Mechanical Ventilation
PSV: Pressure support ventilation

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Acute respiratory distress syndrome

Ventilation Approach


Thank
you