



# Respiratory Failure in the Acute Care Setting

---

A Clinical Guide

Silya Mazigh, MD

Riverside Advanced Practice Provider Conference

02/08/2025

# Disclosures

- I have no financial relationships to disclose.

# Objectives

- Define respiratory failure and its types.
- Discuss the pathophysiology and common causes.
- Identify clinical signs and apply evidence-based diagnostic tools.
- Systematically interpret ABGs and identify respiratory failure patterns.
- Review management protocols in acute care.

# Case presentation

- **Patient:** 68-year-old male
- **PMH:** severe COPD on 2L/min NC at baseline, OSA on CPAP (poor compliance)
- **Chief Complaint:** Increased shortness of breath
- **HPI:**
  - Increased sputum production (yellow/green), wheezing, and worsening dyspnea over 48 hours
  - Family reports new confusion and lethargy
  - No fever, chest pain, or leg swelling

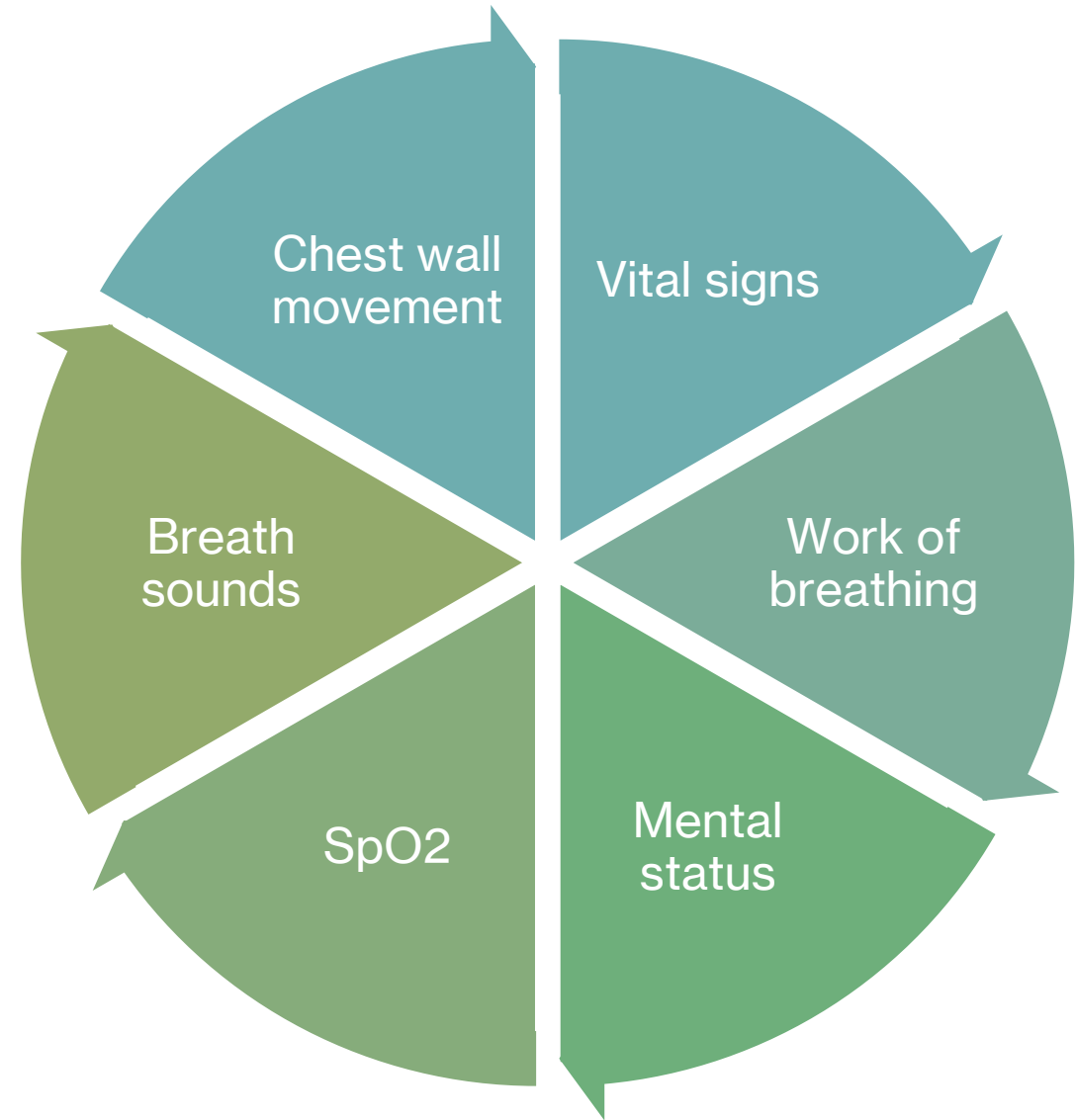


# **Diagnostic Approach**

---



# Initial Assessment





# Physical Exam

- **Vitals:** BP: 145/85 mmHg, HR: 98 bpm, RR: 28/min, SpO<sub>2</sub>: 86% on 2L NC, Temp: 37.0°C
- **General:** Drowsy, but arousable
- **Respiratory:**
  - Labored breathing, accessory muscle use
  - Diffusely diminished breath sounds, expiratory wheezing
  - Prolonged expiratory phase
- **Neurologic:**
  - A&O x 2 (disoriented to place)
  - No focal deficits

# **Preliminary diagnoses**

---



# Definition and Classification of Respiratory Failure

**Definition:** Failure of the respiratory system to maintain oxygenation or ventilation.

## Types:

- **Type 1: Hypoxic**
  - PaO<sub>2</sub> < 60 mmHg on room air
  - SpO<sub>2</sub> ≤ 90% on room air (SpO<sub>2</sub> of 91% correlates with PO<sub>2</sub> of 60 mmHg)
  - A-a gradient > 20 mmHg
- **Type 2: Hypercapnic**
  - PaCO<sub>2</sub> > 45 mmHg with a pH < 7.35 acutely

Acute vs. Chronic vs. Acute-on-Chronic.

# Definition and Classification of Respiratory Failure

## Chronic:

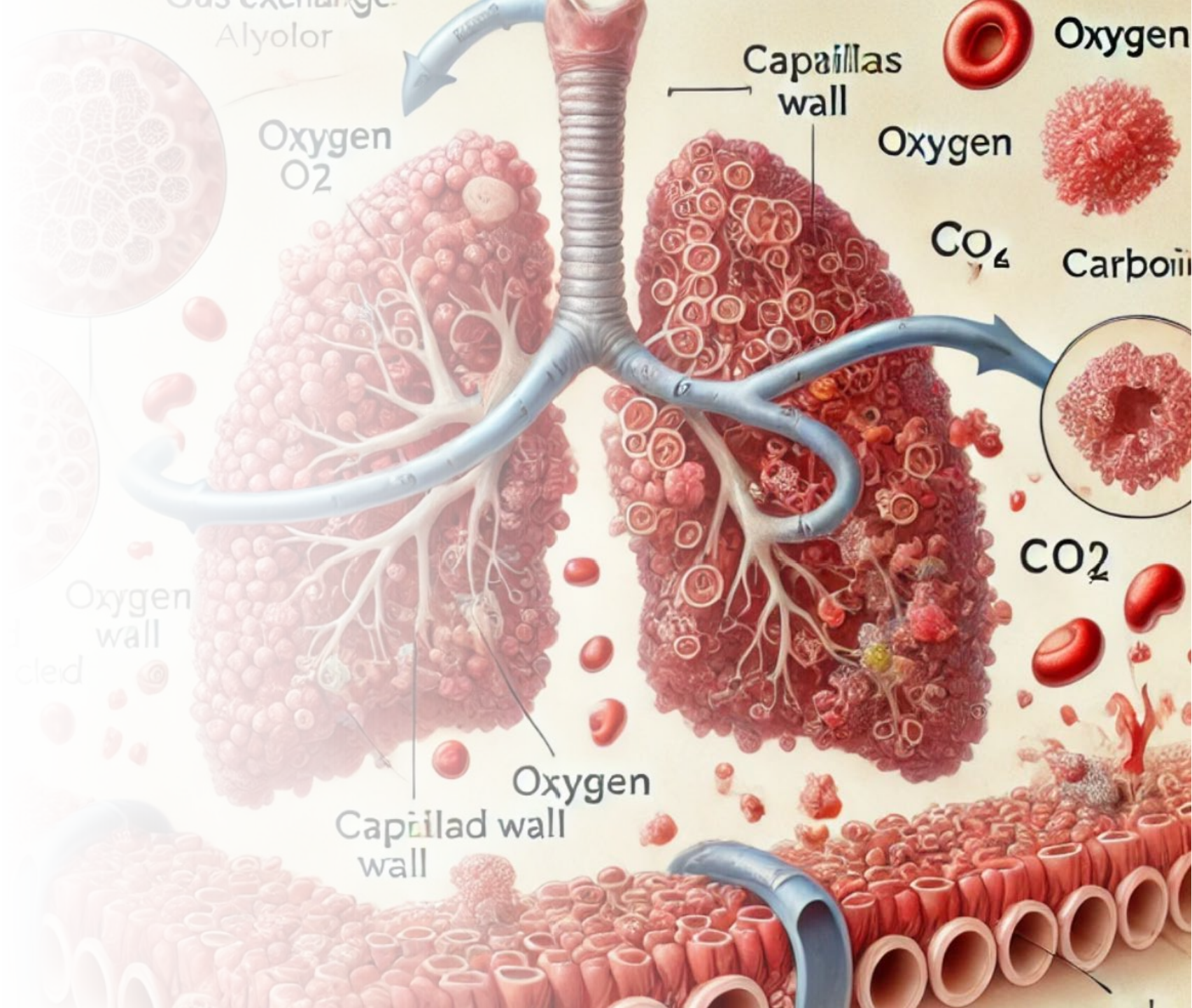
- **Hypoxic:**
  - Dependence on continuous home O<sub>2</sub>
- **Hypercapnic:**
  - Dependence on home non-invasive mechanical ventilation (BiPAP, AVAPS, etc.)

## Acute on Chronic:

- **Hypoxic**
  - Drop in PaO<sub>2</sub> > 10 mmHg from baseline
  - Increased O<sub>2</sub> requirements from baseline to maintain SpO<sub>2</sub> > 88%
- **Hypercapnic**
  - PaCO<sub>2</sub> > 10 mmHg above baseline and pH < 7.35

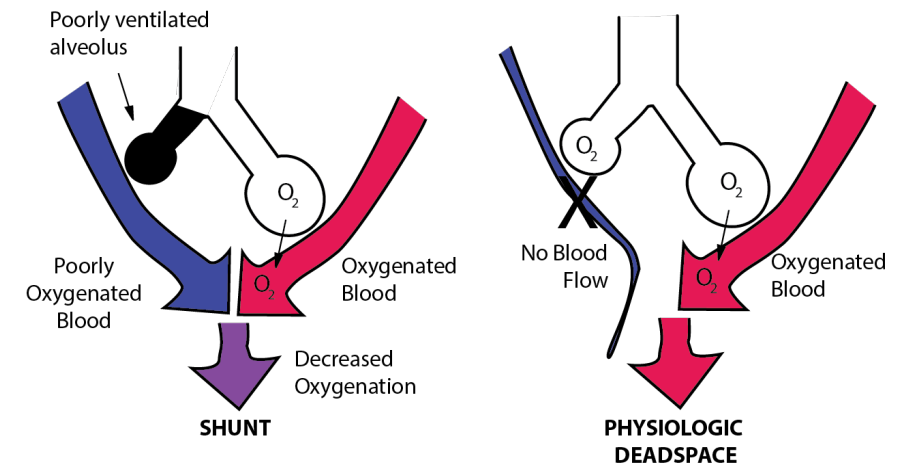
# Pathophysiology of Hypoxia

- Ventilation/perfusion (V/Q) mismatch.
- Right-to-Left Shunt (mechanical or physiologic).
- Dead Space Ventilation.
- Diffusion Limitation.
- Reduced inspired oxygen tension (high altitude sickness).



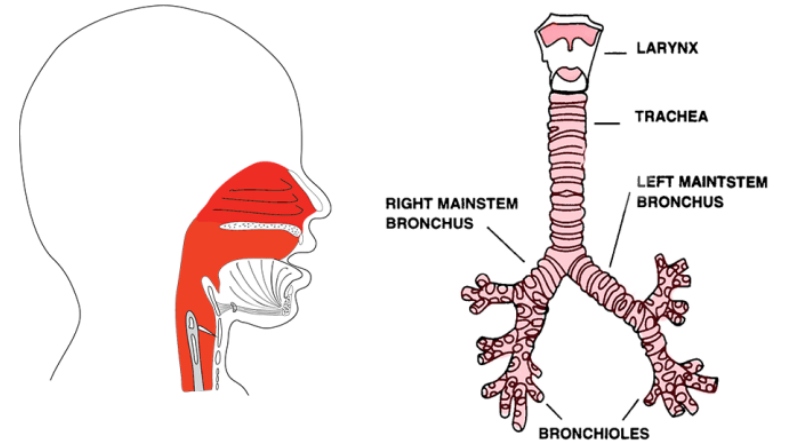
# Ventilation/perfusion (V/Q) mismatch

- The balance between **ventilation (V)** (air reaching the alveoli) and **perfusion (Q)** (blood flow to the alveoli) is disrupted.
- **Low V/Q (Shunt-like Physiology)**
  - **Poor Ventilation, Normal Perfusion**
  - Shunt is perfusion of poorly ventilated alveoli
  - **Pathophysiology:** Alveoli are poorly ventilated but still perfused → Blood remains deoxygenated, leading to hypoxemia.
- **High V/Q (Dead Space-like Effect)**
  - **Poor Perfusion, Normal Ventilation**
  - Physiologic dead space is ventilation of poor perfused alveoli.
  - **Pathophysiology:** Alveoli are ventilated but receive little or no perfusion → Wasted ventilation.



# Dead Space

- **Tidal volume:** the amount of air that move in and out of the lungs with each respiratory cycle.
- **Dead Space:** the parts of the respiratory system that do not participate in gas exchange.
  - **Anatomic:** the fixed parts of the respiratory system that are ventilated but not perfused. Anatomic dead space is about 1/3 of normal tidal volume in an adult.
  - **Physiologic:** alveoli that are ventilated, but not perfused. Physiologic dead space can change as blood flow increases.
  - **Equipment:** the volume inside the ventilation mask or endotracheal tube, the connecting elbow, and the breathing circuit. This volume becomes significant in small patients or small tidal volumes.



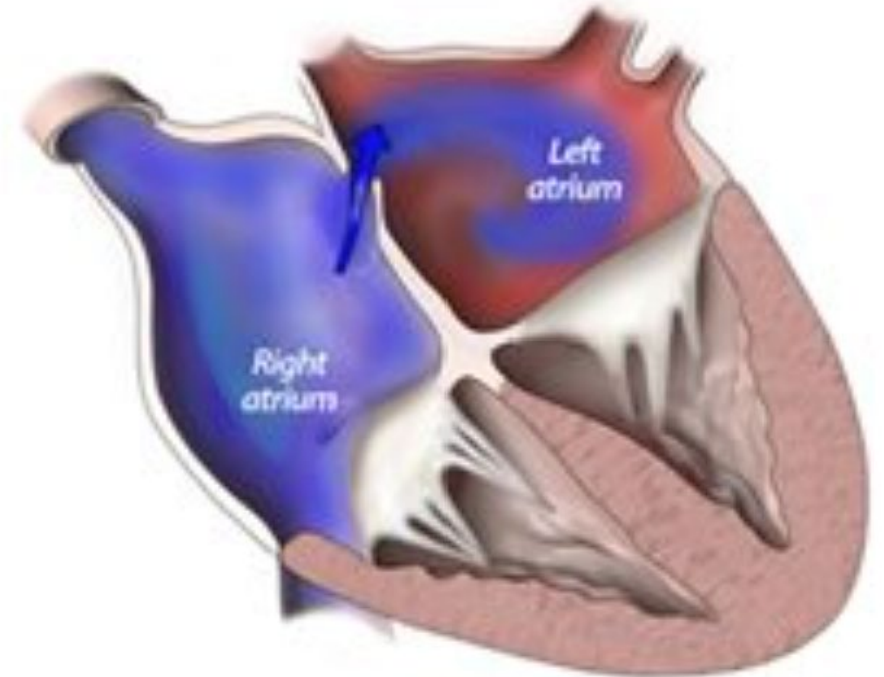


---

# Intracardiac Shunt

---

- Allows deoxygenated blood from the right side of the heart to mix with oxygenated blood on the left side, bypassing the lungs and resulting in a lower overall oxygen level in the bloodstream → Right-to-left shunt.
- Causes of right to left shunt: patent foramen ovale, atrial septal defect, ventricular septal defect, congenital heart disease.

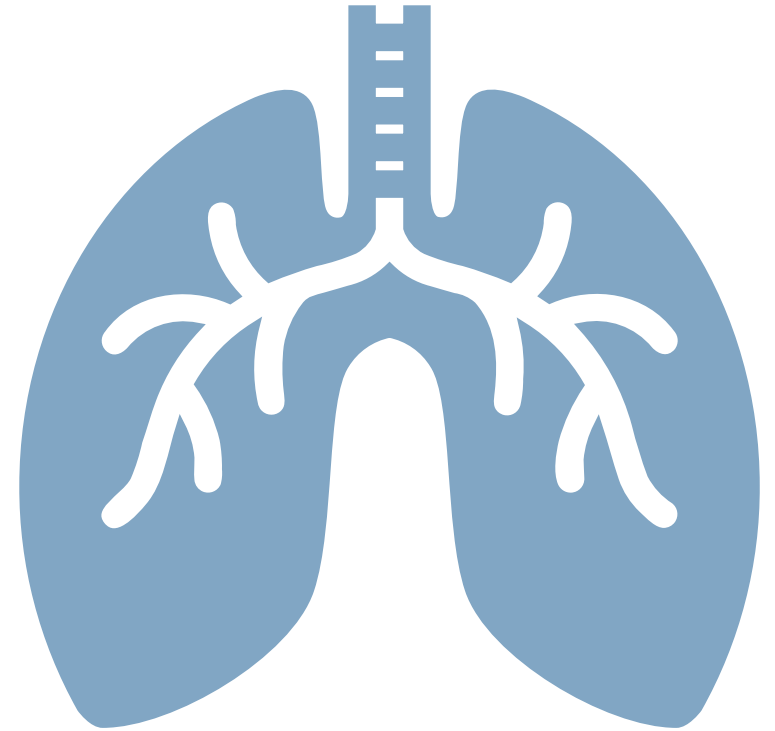


---

# Diffusion Limitation

---

- Oxygen transport across the alveolar-capillary membrane is impaired.
- Typically results from thickening or destruction of the alveolar-capillary interface → slower diffusion of oxygen from alveoli into the blood.
- Common causes:
  - Interstitial lung disease (ILD)/Pulmonary fibrosis
  - Sarcoidosis
  - Asbestosis
  - Scleroderma





# Reduced inspired oxygen tension

- High altitude sickness



# Case presentation

---

Which mechanism is the main cause of hypoxia?

Additional diagnoses?

---

## **Immediate Testing**

---

ABG/VBG

CXR

CBC, BMP

Lactate

ECG

# ABG Basics

• pH: 7.35-7.45

• PaCO<sub>2</sub>: 35-45 mmHg

• PaO<sub>2</sub>: 80-100 mmHg

• HCO<sub>3</sub>: 22-26 mEq/L

• Base Excess: ±2

# Case Presentation - ABG

Parameter	Value	Interpretation
pH	7.25	
PaCO <sub>2</sub>	72 mmHg	
PaO <sub>2</sub>	50 mmHg	
HCO <sub>3</sub> <sup>-</sup>	30 mEq/L	
FiO <sub>2</sub>	21% (room air)	

# ABG Interpretation



Assess Oxygenation

PaO<sub>2</sub>  
A-a gradient  
P/F ratio



Evaluate pH

< 7.35: Acidosis  
> 7.45: Alkalosis



Identify primary disorder

Respiratory: PaCO<sub>2</sub>  
Metabolic: HCO<sub>3</sub>



Check compensation

Winter's formula  
Expected HCO<sub>3</sub> change



Calculate A-a gradient

Bohr equation

# Bohr Equation

- Physiologic dead space:

$$VD/VT = \frac{P_aCO_2 - P_ECO_2}{P_aCO_2}$$

VD: Dead space volume.

VT: Tidal volume.

PaCO<sub>2</sub>: Partial pressure of carbon dioxide in arterial blood.

PeCO<sub>2</sub>: Partial pressure of carbon dioxide in exhaled air.



# Compensation Principles



**Metabolic compensation:** The kidneys adjust bicarbonate ( $\text{HCO}_3^-$ ) levels in response to respiratory disorders. This is a slower process, taking hours to days.



**Respiratory compensation:** The lungs adjust carbon dioxide ( $\text{PaCO}_2$ ) levels in response to metabolic disorders. This is a faster process, occurring within minutes to hours.

# Respiratory Disorders

Primary Disorder	Compensation Mechanism	Expected Compensation
Respiratory Acidosis ( $\uparrow$ PaCO <sub>2</sub> )	Kidneys retain HCO <sub>3</sub> <sup>-</sup> ( $\uparrow$ HCO <sub>3</sub> <sup>-</sup> )	- Acute: $\uparrow$ HCO <sub>3</sub> <sup>-</sup> by 1 mEq/L per 10 mmHg $\uparrow$ PaCO <sub>2</sub> - Chronic: $\uparrow$ HCO <sub>3</sub> <sup>-</sup> by 3.5 mEq/L per 10 mmHg $\uparrow$ PaCO <sub>2</sub>
Respiratory Alkalosis ( $\downarrow$ PaCO <sub>2</sub> )	Kidneys excrete HCO <sub>3</sub> <sup>-</sup> ( $\downarrow$ HCO <sub>3</sub> <sup>-</sup> )	- Acute: $\downarrow$ HCO <sub>3</sub> <sup>-</sup> by 2 mEq/L per 10 mmHg $\downarrow$ PaCO <sub>2</sub> - Chronic: $\downarrow$ HCO <sub>3</sub> <sup>-</sup> by 4-5 mEq/L per 10 mmHg $\downarrow$ PaCO <sub>2</sub>

# Metabolic Disorders

Primary Disorder	Compensation Mechanism	Expected Compensation (Winter's Formula)
Metabolic Acidosis ( $\downarrow$ $\text{HCO}_3^-$ )	Hyperventilation ( $\downarrow$ $\text{PaCO}_2$ )	$\text{PaCO}_2 = (1.5 \times \text{HCO}_3^-) + 8 \pm 2$
Metabolic Alkalosis ( $\uparrow$ $\text{HCO}_3^-$ )	Hypoventilation ( $\uparrow$ $\text{PaCO}_2$ )	$\text{PaCO}_2$ increases by $\sim 0.6$ mmHg per 1 mEq/L $\text{HCO}_3^-$

# Simplified Rules for Expected Compensation

Primary Disorder	Compensation Mechanism	Expected Change
Metabolic Acidosis	Decrease in $P_aCO_2$	$P_aCO_2 = (HCO_3^- + 15) \pm 5$
Metabolic Alkalosis	Increase in $P_aCO_2$	$P_aCO_2 = (HCO_3^- + 15)$
Respiratory Acidosis (Acute)	Increase in $HCO_3^-$	$\Delta HCO_3^- = 1$ per 10 mmHg $P_aCO_2$
Respiratory Acidosis (Chronic)	Increase in $HCO_3^-$	$\Delta HCO_3^- = 4$ per 10 mmHg $P_aCO_2$
Respiratory Alkalosis (Acute)	Decrease in $HCO_3^-$	$\Delta HCO_3^- = 2$ per 10 mmHg $P_aCO_2$
Respiratory Alkalosis (Chronic)	Decrease in $HCO_3^-$	$\Delta HCO_3^- = 5$ per 10 mmHg $P_aCO_2$



**Additional  
diagnoses?**

# Test Results

- ☑ **CXR:** hyperinflation, no acute cardiopulmonary process
- ☑ **ECG:** sinus tachycardia, no ST-T changes
- ☑ **CBC:** WBC 12.5K
- ☑ **BMP:** significant electrolyte imbalances, normal renal function
- ☑ **Lactic acid:** 1.2 mmol/L



## Further Diagnostics

---

CT chest

D-dimer

BNP

Procalcitonin

Blood  
cultures



# Management Protocol – Hypoxemia

---

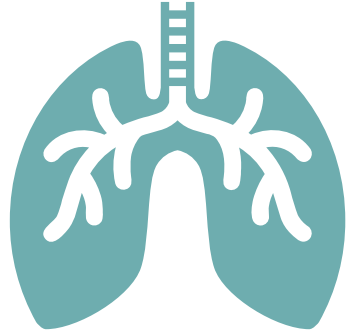
Initiate  
Oxygen  
Therapy

Monitor  
Parameters

Indications  
for  
intubation

# Risk Stratification

---



Need for immediate  
intubation?



Trial of non-invasive  
support?

# Oxygen therapy

Nasal Cannula (1-6 L/min)

High-Flow NC (30-60 L/min)

Non-invasive ventilation

- Initial IPAP: 10 cmH<sub>2</sub>O
- Initial EPAP: 5 cmH<sub>2</sub>O
- FiO<sub>2</sub>: Titrate to SpO<sub>2</sub> 88-92%
- Rate: 12-15/min

Mechanical ventilation

# Monitor Parameters

---

SpO<sub>2</sub>

Work of  
breathing

Mental status

Hemodynamics

Serial ABGs

# Case Presentation - ABG

Parameter	Value	Interpretation
pH	7.20	
PaCO <sub>2</sub>	80 mmHg	
PaO <sub>2</sub>	65 mmHg	
HCO <sub>3</sub> <sup>-</sup>	30 mEq/L	
O <sub>2</sub>	5 L/min NC	

# Management Protocol – Hypercapnia

## 1. NIV Strategy

- IPAP: Start 10, increase to effect
- EPAP: Start 5, titrate for OSA/COPD
- Monitor tolerance and air leak

## 2. Medical Management

- Bronchodilators
- Steroids if indicated
- Treat underlying cause

## 3. Monitoring

- Serial ABGs
- Continuous capnography if available
- Mental status
- Work of breathing

# Indications for Intubation

---

Persistent hypoxemia despite maximal non-invasive support

Severe work of breathing

Altered mental status

Hemodynamic instability





# Case Studies

# Case Presentation

- **Scenario:** 55-year-old female admitted with COVID-19 pneumonia now with increased work of breathing.
- **Clinical Findings:**
  - ABG: pH 7.48, PaCO<sub>2</sub> 32 mmHg, PaO<sub>2</sub> 58 mmHg on 60L/min HFNC.
  - Chest X-ray: Bilateral patchy opacities.
- **Management**

# Case Presentation

- **Scenario:** 74-year-old female with CHF and COPD presenting with admitted with acute decompensation
- **Clinical Findings:**
  - ABG: pH 7.37, PaCO<sub>2</sub> 54 mmHg, PaO<sub>2</sub> 55 mmHg on 2L/min NC.
  - Chest X-ray: Bilateral pulmonary vascular congestion.
- **Management**

# References

- Roca O, Riera J, Torres F, Masclans JR. High-flow oxygen therapy in acute respiratory failure. *Respir Care* 2010; 55:408.
- Fujishima, S. Guideline-based management of acute respiratory failure and acute respiratory distress syndrome. *J Intensive Care* 11, 10 (2023). <https://doi.org/10.1186/s40560-023-00658-3>
- Griffiths MJD, McAuley DF, Perkins GD, Barrett N, Blackwood B, Boyle A, et al. Guidelines on the management of acute respiratory distress syndrome. *BMJ Open Respir Res.* 2019;6:e000420.
- O'Driscoll BR, Howard LS, Earis J, et al. BTS guideline for oxygen use in adults in healthcare and emergency settings. *Thorax* 2017; 72:ii1.
- Su L, Zhao Q, Liu T, et al. Efficacy of High-Flow Nasal Cannula Oxygen Therapy in Patients with Mild Hypercapnia. *Lung* 2021; 199:447.
- Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global Strategy for the Diagnosis, Management and Prevention of Chronic Obstructive Pulmonary Disease: 2024 Report. [www.goldcopd.org](http://www.goldcopd.org) (Accessed on November 16, 2023).
- Illustrations:
  - <https://airwayjedi.com/2017/01/06/ventilation-perfusion-mismatch/>
  - <https://airwayjedi.com/2024/07/08/understanding-anatomic-dead-space-impact-on-ventilation/>
  - <https://www.nationaljewish.org/conditions/intracardiac-shunts>