Why do we keep oxygen saturation in COPD Patients within 88-92%?

The conventional belief is that **oxygen therapy depresses the respiratory drive** in chronic CO? retainers (i.e., COPD patients), leading to **hypoventilation** , **CO? retention** , and **type 2 respiratory failure** . This is based on the idea that these patients rely primarily on **hypoxic respiratory drive** rather than PaCO? levels to stimulate breathing.

This is a myth.
Most COPD patients—even chronic CO? retainers—retain an **elevated respiratory drive** , especially during exacerbations. The cause of oxygen-induced hypercapnia is **not** simply a loss of hypoxic drive.

The Real Mechanisms Behind Oxygen-Induced Hypercapnia:

1. Worsening of V/Q Mismatch

Mechanism:

- In COPD, airflow limitation and alveolar destruction result in poorly ventilated lung areas.
- The body compensates through **hypoxic pulmonary vasoconstriction** —diverting blood flow away from poorly ventilated alveoli to better-ventilated ones.
- When high concentrations of oxygen are administered:
    - Hypoxic vasoconstriction is **reversed** .
    - Blood is redirected back to **poorly ventilated alveoli** .
    - This causes **ventilation-perfusion (V/Q) mismatch** and increased **physiologic dead space** .

Result:

- Impaired gas exchange.
- **Increased CO? retention** , even in patients who are not chronic CO? retainers.
- Affects **all COPD patients** , but **more pronounced** in those with advanced disease.

2. The Haldane Effect

Mechanism:

- Hemoglobin's affinity for CO? depends on its oxygenation state:
    - **Deoxygenated hemoglobin** binds CO? more readily (as carbamino compounds).
    - **Oxygenated hemoglobin** has reduced CO?-carrying capacity.
- Administering supplemental oxygen:
    - Increases hemoglobin saturation with O?.
    - Reduces the blood's capacity to carry CO?.
    - Leads to **increased PaCO?** , even if minute ventilation remains unchanged.

Result:

- **CO? retention** occurs because hemoglobin offloads CO? less efficiently.
- The **Haldane effect** accounts for approximately **25% of the total PaCO? increase** seen with oxygen therapy in severe COPD.

Clinical Implication: Why Target SpO? = 88–92%

Maintaining oxygen saturation in the **88–92%** range:

- Ensures **adequate oxygen delivery** to tissues without significantly disrupting V/Q matching.
- Minimizes the impact of the **Haldane effect** on CO? retention.
- Reduces the risk of **acute hypercapnia** , **respiratory acidosis** , and **ventilatory failure** .

Key Point: The goal is to provide **just enough oxygen** to relieve hypoxemia **without disturbing the body's compensatory mechanisms** in gas exchange.

Evidence & Guidelines:

- These studies demonstrate that **high-flow oxygen** in COPD exacerbations is associated with:
  - Higher **PaCO?**
  - Increased **acidosis**
  - **Higher mortality**

Takeaway:

"In COPD, aim for oxygen saturation between 88–92% not to prevent loss of respiratory drive, but to prevent V/Q mismatch and limit the Haldane effect, both of which increase PaCO? and risk respiratory failure."