



Foundations Frameworks

Approach to Hypoxemia

FOUNDATIONS
of Emergency Medicine

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Is your patient hypoxic, hypoxemic, or both?

Hypoxia is insufficient delivery of oxygen to the tissues

- Due low cardiac output, low Hb concentration, or low SaO₂

Hypoxemia is an abnormally low arterial oxygen tension in the blood

- Defined as PaO₂ ≤ 60 mmHg (with the patient on room air)

Can occur independently:

- Patient with polycythemia can have low PaO₂ (hypoxemia) but no hypoxia due to increased O₂ delivery
- Patient that with severe anemia can have normal PaO₂ but hypoxia due to decreased O₂ delivery

Hypoxemia can be caused by four major categories of disease:

1. Hypoventilation
2. V/Q mismatch (shunt physiology)
3. Delivery/uptake mismatch
4. Diffusion impairment

The alveolar-arterial O₂ gradient¹ is the best tool to differentiate these, so an ABG may be helpful in an otherwise undifferentiated hypoxemic patient. However, this is only obtainable in intubated patients whose FiO₂ can be definitively determined, and additionally these categories can significantly overlap, so this analysis is more applicable to the ICU than to the ED.

Hypoventilation

This is mainly from the following three categories:

- Brainstem respiratory depression (e.g. drug-induced, obesity-hypoventilation syndrome)
- Peripheral neuropathy (e.g. Guillain-Barre syndrome)
- Muscle weakness (e.g. myasthenia gravis, myopathy)

Note that hypoventilation is the ONLY cause of hypoxemia that does not alter the A-a gradient (under normal atmospheric partial pressures of oxygen, which can be decreased by e.g. fires or altitude).

V/Q Mismatch

Pretty much any lung disease can be included in this category, but the big four include:

- Blood (e.g. alveolar hemorrhage)
- Water (e.g. pulmonary edema, ARDS, CHF)
- Pus (e.g. pneumonia, empyema)
- Atelectasis (including COPD/asthma due to blood flow to ineffectively ventilated lung)

Note that cardiac abnormalities (e.g. PFO, VSD) can create shunt physiology as well (i.e. ventilation of the lungs without perfusion, with V/Q close to zero). Conversely, V/Q can approach infinity, creating perfusion without ventilation, or dead space (e.g. PE, pneumothorax).

¹ A-a gradient = PAO₂ – PaO₂. PAO₂ (ideal alveolar O₂) is PiO₂ – PaCO₂/0.8. Because the A-a gradient increases with age, a “normal” gradient can be estimated as < (age in years/4) + 4.

Delivery/uptake Mismatch

The mismatch here only really matters when the lungs are impaired from extracting O₂ from the air, because during normal gas exchange a low mixed venous O₂ will be compensated by increased alveolar uptake. The mismatch can be from the following two categories:

- Decreased O₂ delivery (e.g. anemia, low-output shock, impaired O₂ binding)
- Increased tissue O₂ utilization (e.g. sepsis, thyrotoxicosis, hyperthermia, etc.)

Diffusion Impairment

This is a rare cause of acute hypoxemia, as it is frequently due to chronic structural lung problems (e.g. emphysema and pulmonary fibrosis). Still, acute pathology producing hypoventilation, V/Q mismatch, or delivery/uptake mismatch may complicate an underlying diffusion impairment.

Approach to Supplemental Oxygen

This only works reliably for hypoventilation, diffusion impairment, and delivery/uptake mismatch (not VQ mismatch)

- Hypoventilation: supplemental O₂ increases amount of alveolar O₂, which increases uptake by alveolar capillary blood
- Delivery/uptake mismatch: supplemental O₂ saturates available hemoglobin and increases the plasma saturation, increasing the amount of O₂ deliverable to tissues
- Diffusion impairment: supplemental O₂ helps overcome the lung's underlying difficulty in transporting oxygen into the alveolar capillary blood
- V/Q mismatch can *sometimes* be alleviated by supplemental O₂ if V/Q < 1, but it can also act as a shunt (V/Q = 0). In shunt physiology, blood circulates to alveoli unavailable for gas exchange, so supplemental O₂ won't help you here. Instead, you need to modify your respiratory support to treat the structural problem (e.g. PEEP/BiPAP, thrombolysis, etc.).

General Treatment for Hypoxia

1. Administer supplemental O₂:
 - This will improve most etiologies of hypoxemia except for severe V/Q mismatch leading to shunt physiology
2. Simultaneously address underlying pathophysiology:
 - Hypoventilation: give naloxone or other targeted drug antidote, provide NIPPV or intubate for impending (or actual) ventilatory failure
 - V/Q Mismatch: thrombolysis (or catheter-directed therapy) for PE, NIPPV for obstructive lung disease or CHF pulmonary edema, intubate as necessary
 - Delivery/Uptake Mismatch: blood transfusion for anemia, pressors for shock, 100% O₂ or hyperbaric therapy for CO poisoning, methylene blue for methemoglobinemia, propranolol for thyroid storm, etc.
 - Diffusion Impairment: be aware of underlying pathology that may be worsened by other causes of hypoxemia (e.g. prostaglandin-dependent pulmonary hypertension)

References:

Marino, Paul. *The ICU Book*. Chapter 20: Hypoxemia and Hypercapnia.