

# Hypoxemia

## Causes of Hypoxemia

	$P_a\text{CO}_2$	A-a Gradient	DLCO	Corrects w/ 100% $F_i\text{O}_2$ ?
Low $F_i\text{O}_2$	Normal	Normal	Normal	Yes
Hypoventilation	↑	Normal	Normal	Yes
Diffusion Impairment	Normal	↑	↓	Yes
Shunt	Normal	↑	Normal	No
V/Q Mismatch	Normal / ↑	↑	Normal	Yes

Shunt: perfusion without ventilation ( $V/Q=0$ ); see  $\downarrow p\text{O}_2$

Dead Space: ventilation without perfusion ( $V/Q=\infty$ ); see  $\uparrow p\text{CO}_2$

## Equations

### Alveolar-arterial (A-a) Gradient

$$P_{(A-a)}\text{O}_2 = P_A\text{O}_2 - P_a\text{O}_2$$

### Alveolar Gas Equation

$$P_A\text{O}_2 = F_i\text{O}_2 (P_{\text{atm}} - P_{\text{H}_2\text{O}}) - (P_a\text{CO}_2 / 0.8)$$

$$= 0.21 (760 - 47) - (40 / 0.8)$$

$$\approx \underline{100 \text{ mm Hg}}$$

#### Normal A-a Gradient:

- < 10 mm Hg ( $F_i\text{O}_2 = 0.21$ )
- < 60 mm Hg ( $F_i\text{O}_2 = 1.00$ )
- <  $(\text{age} / 4) + 4$
- a/A ratio > 0.75

#### Normal $P_a\text{O}_2$ :

- $103 - \text{age}/3$

## Causes of Hypoxemia

### 1. Low $F_i\text{O}_2$

- Altitude
- Hypoxic  $F_i\text{O}_2$  gas mixture

### 2. Hypoventilation

- Drugs (opioids, BZDs, barbiturates)
- Chest wall damage
- Neuromuscular diseases
- Obstruction (e.g. OSA, upper airway compression)

### 3. Diffusion Impairment

- Increased diffusion pathway (e.g. pulmonary edema, fibrosis)
- Decreased surface area (e.g. emphysema, pneumonectomy)
- Decreased rate of  $\text{O}_2$ -Hb association (e.g. high CO, anemia, PE)

## Causes of Hypoxemia

### 4. Shunt (i.e. perfusion w/o ventilation; $V/Q = 0$ )

- Congenital (e.g. ASD, VSD, PDA), or AVM
- ARDS, pneumonia, atelectasis

### 5. V/Q Mismatch

- Often multifactorial
- COPD, ILD, PE
- Decreased CO (e.g. MI, CHF)

### 6. Mixed Process

- Hypoxemia is often due to multiple causes.
- Example: A tourist with COPD is visiting Denver, overdoses on heroin, now s/p MVA with chest wall trauma, pulmonary hemorrhage, Hct = 15%, and LV contusion. What is the cause of hypoxemia?

## Hypoxemia in the OR

### Take a systematic approach to the diagnosis and treatment of hypoxemia in the OR!

Suggestion: *Alveoli* → *Machine*

#### 1. Listen to the lungs

- Atelectasis
- Pulmonary edema
- Bronchoconstriction
- Mucus plug or secretions
- Right mainstem ETT
- Pneumothorax
- Esophageal intubation

#### 2. Check ETT

- Cuff deflation
- Kinked/bitten ETT
- Extubation

## Hypoxemia in the OR

### 3. Check circuit

- ETT disconnect
- Circuit disconnect

### 4. Check machine

- Inspiratory & expiratory valves
- Bellows
- Minute ventilation
- $F_iO_2$
- Pipeline & cylinder pressures

### 5. Check monitors to confirm (you will probably do this 1<sup>st</sup>!)

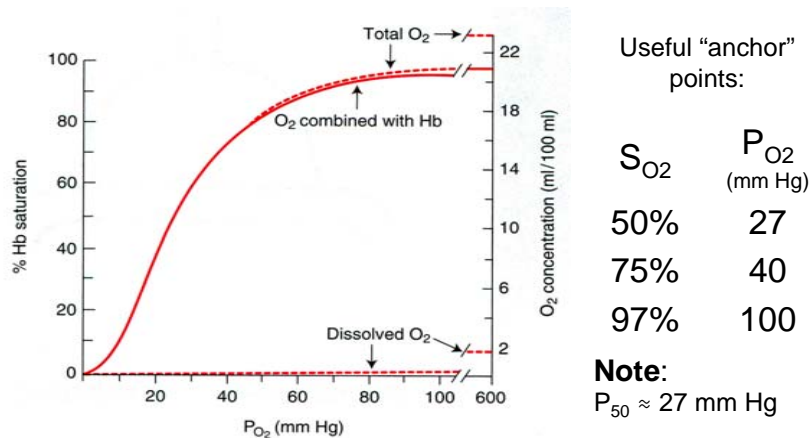
- Pulse oximeter waveform
- Gas analyzer

## Management of Hypoxemia

### Assuming proper oximeter function, placement, and waveform:

- Place patient on 100%  $O_2$ .
- Perform recruitment maneuver and/or add PEEP.
- Confirm ETT placement by auscultation, bilateral chest rise, and FOB if necessary.
- Suction airway.
- Consider cardiovascular causes and restore volume, RBCs and/or cardiac output.

## O<sub>2</sub>-Hb Dissociation Curve



## O<sub>2</sub>-Hb Curve Shifts

### Right Shift

(lower affinity for O<sub>2</sub> = increased unloading at tissues)

- Acidosis
- Hyperthermia
- Hypercarbia
- Increased 2,3-DPG
- Sickle Cell Hb
- Pregnancy
- Volatile anesthetics
- Chronic anemia

### Left Shift

(higher affinity for O<sub>2</sub> = decreased unloading at tissues)

- Alkalosis
- Hypothermia
- Hypocarbica
- Decreased 2,3-DPG
- CO-Hb
- Met-Hb
- Sulf-Hb
- Fetal Hb
- Myoglobin

## Factors Affecting Tissue Oxygenation

- Hb concentration
- O<sub>2</sub> Saturation
- Cardiac Output
- O<sub>2</sub> Consumption
- O<sub>2</sub>-Hb Affinity (P<sub>50</sub>)
- Dissolved O<sub>2</sub> in plasma (little effect)

See "Equations" for a mathematical explanation of these factors.

## Equations

### Arterial O<sub>2</sub> Content

$$\begin{aligned}
 C_aO_2 &= O_2\text{-Hb} + \text{Dissolved } O_2 \\
 &= (Hb \times 1.36 \times S_aO_2/100) + (P_aO_2 \times 0.003) \\
 &= (15 \times 1.36 \times 100\%) + (100 \times 0.003) \\
 &\approx \underline{20 \text{ cc } O_2/\text{dl}}
 \end{aligned}$$

### Mixed Venous O<sub>2</sub> Content

$$\begin{aligned}
 C_vO_2 &= O_2\text{-Hb} + \text{Dissolved } O_2 \\
 &= (Hb \times 1.36 \times S_vO_2/100) + (P_vO_2 \times 0.003) \\
 &= (15 \times 1.36 \times 75\%) + (40 \times 0.003) \\
 &\approx \underline{15 \text{ cc } O_2/\text{dl}}
 \end{aligned}$$

## Equations

### O<sub>2</sub> Delivery

$$\begin{aligned} \text{DO}_2 &= \text{CO} \times \text{C}_a\text{O}_2 \\ &= 5 \text{ L/min} \times 20 \text{ cc O}_2/\text{dl} \\ &\approx \underline{1 \text{ L O}_2/\text{min}} \end{aligned}$$

### O<sub>2</sub> Consumption (Fick Equation)

$$\begin{aligned} \text{VO}_2 &= \text{CO} \times (\text{C}_a\text{O}_2 - \text{C}_v\text{O}_2) \\ &= 5 \text{ L/min} \times 5 \text{ cc O}_2/\text{dl} \\ &\approx \underline{250 \text{ cc O}_2/\text{min}} \end{aligned}$$

### O<sub>2</sub> Extraction Ratio

$$\begin{aligned} \text{ER}_{\text{O}_2} &= (\text{VO}_2 / \text{DO}_2) \times 100 \\ &= 250 / 1000 \\ &\approx \underline{25\% \text{ (normal 22-30\%)}} \end{aligned}$$

## Other Concepts

**Diffusion Hypoxia** = low P<sub>A</sub>O<sub>2</sub> as a result of breathing air, in combination with the washout of N<sub>2</sub>O into the alveoli, upon termination of an anesthetic.

**Absorption Atelectasis** = the tendency for airways to collapse if proximally obstructed; poorly soluble N<sub>2</sub> normally stents alveoli open, but patients on 100% O<sub>2</sub> have greater tendency toward atelectasis.

**Bohr Effect** = a property of Hb in which increasing CO<sub>2</sub>, temperature, and acidosis promote decreased O<sub>2</sub>-Hb affinity (i.e. right-shift of O<sub>2</sub>-Hb curve).

**Haldane Effect** = a property of Hb in which O<sub>2</sub> promotes dissociation of CO<sub>2</sub> from Hb to the plasma (e.g. as when venous blood enters the lungs).

## References

- Gaba DM, Fish KJ, and Howard SK. *Crisis Management in Anesthesiology*. Philadelphia: Churchill Livingstone, Inc., 1994.
- West JB. *Respiratory Physiology: The Essentials, 7th ed.* Philadelphia: Lippincott Williams & Wilkins, 2005.
- West JB. *Pulmonary Pathophysiology: The Essentials, 6th ed.* Philadelphia: Lippincott Williams & Wilkins, 2003.