## PULMONARY PHYSIOLOGY

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### I. Lung volumes



- A. Tidal volume (TV) is the volume of air entering and exiting the lungs during each breath
  - 1. Approximately 500mL

B. Inspiratory reserve volume (IRV) is the volume of air that can be inhaled (forcibly using inspiratory muscles) beyond normal inspiration (beyond normal TV)

C. Inspiratory capacity (IC) = TV + IRV

D. Expiratory reserve volume (ERV) is the volume of air that can be exhaled (forcibly using expiratory muscles) beyond normal expiration (beyond normal TV)

E. Vital capacity (VC) is the maximum volume of air that can be exhaled forcibly after maximal inspiration (VC = IRV + TV + ERV)

1. Forced vital capacity (FVC) is the total volume expired from maximal inspiration to maximal expiration and is normally 80-120% of the TV

F. Residual volume (RV) is the remaining volume of air that can't be exhaled forcibly after maximal inspiration (RV = FRC - ERV)

G. Functional residual capacity (FRC) is the volume of air remaining in the lungs after normal passive expiration (FRC = ERV + RV)

1. FRC is the lung's physiological reserve/reservoir (resting volume of the lung) for gas exchange

2. Decreased lung compliance reduces FRC  $\rightarrow$  alveolar collapse

3. Positive end-expiratory pressure (PEEP) is applied at end-expiration and increases FRC by preventing alveolar collapse (atelectrauma)

a. IN ARDS, lung compliance is reduced (lungs are stiffer and more difficult to inflate  $\rightarrow$  reduction in FRC to a point that is less than closing capacity/volume and results in airway closure  $\rightarrow$ low V/Q  $\rightarrow$  hypoxemia.

b. PEEP recruits lung units, increases FRC, decreases venous admixture and improves oxygenation

H. Total lung capacity (TLC) is the volume of air in the lungs after maximal inspiration (TLC = VC + RV)

I. Minute volume is the volume of air exhaled every minute

J. Forced expiratory volume in 1 second ( $FEV_1$ ) is the volume of air that can be forcefully expired in 1 second

1.  $FEV_1 = 80\%$  of the VC normally

2. Reduced in obstructive lung disease (increased airway resistance) and in restrictive lung disease (low VC)

- K. Spirometry measures TV, IRV, IC, ERV, and VC directly
  - 1. FVC maneuver (maximal inspiration followed by maximal expiration)
    - a. Normal airway resistance =  $FEV_1/FVC > 70\%$
    - b. Obstructive lung disease (asthma, COPD) = ratio decreased
    - c. Restrictive lung disease (pulmonary fibrosis) = ratio normal or increased
- II. Ventilation (rate at which gas enters and leaves the lungs)
  - A. Respiratory (minute) ventilation (MV) is the total volume of gas entering the lungs per minute

1. TV (mL/breath) x respiratory rate (RR in breaths/min) =  $500mL \times 12 = 6L/min$  of gas/air breathed in and out at rest

B. Dead space ventilation is the volume of gas per unit time that does not reach the alveoli and is not involved in gas exchange but remains in the proximal conducting airways

- 1. Dead space (V<sub>D</sub>) x RR
- 2. Dead space = 0.15L (volume in conducting airways is anatomic dead space)
- 3. 1.8L/min at rest

C. Alveolar ventilation is the volume of gas per unit time that reaches the alveoli (where gas exchange occurs)

1.  $(TV - V_D) \times RR = (500mL - 150mL) \times 12 \text{ breaths/min} = 4.2L/\text{min} \text{ at rest}$ 

2. Increasing TV (slow and deep breaths) is more efficient method to increase alveolar ventilation as increasing RR (fast and shallow) increases alveolar ventilation as well, but also increases dead space

a. In ALI/ARDS, tidal volumes of 6 mL/kg result in significant atelectasis leading to wasted ventilation  $\rightarrow$  increase RR to control PaCO<sub>2</sub>

### III. Gas exchange

A. Primary purpose of the respiratory system is to exchange oxygen and carbon dioxide across the alveolar epithelium and capillary endothelium via passive diffusion

- 1. Rate of gas transfer dependent on:
  - a. The difference in partial pressure of the gases across the membrane
  - b. The surface area of the membrane
  - c. The thickness of the membrane
  - d. The molecular weight and the solubility of the gas crossing the membrane

aa.  $CO_2$  is 20X more soluble than  $O_2$ 



B. Efficiency of gas exchange across the lung calculated using alveolar gas equation

1. The partial pressure of  $CO_2$  in arterial blood (PaCO<sub>2</sub>) increases when minute ventilation decreases  $\rightarrow$  decreases alveolar PAO<sub>2</sub> and arterial PaO<sub>2</sub> because CO<sub>2</sub> displaces oxygen in the alveolus

2.  $PAO_2 = FIO_2 \times (P_{atm} - P_{H2O}) - (PaCO_2/RQ)$  where RQ = the respiratory quotient (O<sub>2</sub> consumed to CO<sub>2</sub> produced when nutrients are metabolized) and varies from 0.7-1.0 (1.0 carbohydrate, 0.7 protein, and 0.6 fat)

= 0.21 x (760 mm Hg - 47 mm Hg) - (40 mm Hg/0.8)

= 100 to 110 mm Hg

### C. Dead space

1. Alveolar dead space is when air contacts the alveoli but lack blood flow in adjacent pulmonary capillaries (ventilation without perfusion as in PE) and therefore no gas exchange

- 2. Physiologic dead space = anatomic dead space + alveolar dead space
  - a. Wasted portion of breath that does not participate in CO<sub>2</sub> exchange



b. PE, pulmonary vasoconstriction, and high tidal volume ventilation act to increase physiologic dead space

c. With lung disease, dead space varies compared to tidal volume.  $V_D/V_T$  ratio helps determine how much respiratory effort is being wasted

aa. normal ratio is 0.15 to 0.35

bb. in critically ill, may exceed 0.7 (70% is wasted ventilation)

D. Diffusing capacity of the lung for carbon monoxide ( $D_LCO$ ) – volume of CO transferred across the alveolar-capillary membrane (ease with which oxygen moves from inhaled air to the RBC in the pulmonary capillaries) per minute per unit alveolar partial pressure. Normal 17-25 mL/min/mmHg

1. Decreased in COPD; normal or high in asthma

## IV. Lung zones, V/Q, and Shunt

A. Zones reflect influence of gravity on degree of alveolar distention and blood flow

1. in the upright position (standing, sitting), Zone 1 at the apices, Zone 3 at bases

2. in the supine position, Zone 1 is the most anterior, Zone 3 is most posterior

3.  $P_A$  = Alveolar pressure.  $P_a$  = arterial pressure.  $P_v$  = venous pressure.

**Zone 1:**  $P_A > P_a > P_v$ .

Gravity stretches the Zone 1 alveoli most; negative pressure created by inspiration is greatest in Zone 1; distention of alveoli (and therefore alveolar pressure) greatest in Zone 1. Theoretically with maximal alveolar distention, alveolar pressure would be greater than arterial pressure and therefore prevent blood flow. Likely occurs more in the hypovolemic state rather than in normal euvolemic lungs. Ventilation (V) is highest and perfusion (Q) is lowest in Zone 1, so V/Q ratio is highest here.

# **Zone 2:** $P_a > P_A > P_v$ .

Arterial pressure overcomes alveolar pressure, allowing blood flow, but with distention alveolar pressure still higher than venous pressure and may inhibit flow. Ventilation and perfusion are best matched in Zone 2.

# Zone 3: $P_a > P_v > P_A$ .

Perfusion is greatest but ventilation poorest in Zone 3, so V/Q ratio is lowest here. Zone 3 affects gas exchange most, as it is more dependent on blood flow.

4. Manipulation of the lung zones is the basis of rotational therapy for mechanically ventilated patients and for prone positioning of ARDS patients. By shifting Zones 1 and 3 back and forth, overall V/Q mismatch is decreased, and gas exchange is improved.

## B. Ventilation/Perfusion (V/Q) mismatch

1. V/Q mismatch described in terms of the "A-a gradient": the difference in partial pressure of oxygen between the Alveoli (A) and arterial blood (a).

a. anatomic shunt (aka true shunt)

i. about 3% of blood reaching the left heart has not gone through the pulmonary capillary circulation (i.e. anatomic shunt)

1. bronchial blood flow

2. thebesian veins going directly to left heart

ii. because of the anatomic shunt, the paO2 of normal arterial blood (breathing room air) is reduced from about 100 mmHg in the pulmonary veins to about 95 mmHg after leaving the heart

b. In Zone 1 (high V/Q, lower blood flow so more opportunity for gas exchange), paO2 of blood leaving alveoli is > 100 mmHg, while in Zone 3 (low V/Q, higher blood flow but less ventilation), paO2 is < 100 mmHg

2. P<sub>A</sub> calculated by the alveolar gas equation:

 $P_AO2 = (FIO2 \times [P_{atmos} - P_{H2O}]) - (PaCO2 / RQ)$ 

FIO2 = fraction of inspired oxygen

 $P_{atmos}$  = atmospheric pressure, i.e. 760 mm Hg at sea level

 $P_{H2O}$  = water vapor pressure at 37 degrees, i.e. 47 mm Hg

PaCO2 = partial pressure of arterial CO2

RQ = respiratory quotient, i.e. ratio of CO2 eliminated to O2 consumed, which is normally 0.8

### C. Shunt

- 1. Anatomic shunt discussed in IV.B.1.a.
- 2. Transpulmonary shunt

a. shunting of blood through the pulmonary circulation without first being oxygenated (or being suboptimally oxygenated)

b. occurs with perfusion of nonventilated or hypoventilated alveoli (e.g. infection, pulmonary edema, atelectasis), or by inability of O2 to diffuse across the alveolar membrane into the blood (e.g. interstitial fibrosis)

c. shunted blood is not further oxygenated, mixes with the remaining pulmonary circulation and lowers the overall paO2 (i.e. hypoxemia)

d. degree of shunting calculated by the shunt fraction (Qs/Qt)

 $Qs/Qt = \frac{CcO2 - CaO2}{CcO2 - CvO2}$ 

CcO2 = O2 content in pulmonary capillary blood (i.e. just postalveolus and maximally oxygenated)

CaO2 = O2 content in arterial blood (i.e. after leaving the heart, so includes blood from the anatomic shunt whose O2 content is

normally a bit lower than the CcO2, and from the transpulmonary [pathological] shunt)

CvO2 = O2 content in venous blood

e. CcO2 not directly measurable clinically, so shunt fraction can be estimated from existing graphs

f. the normal shunt fraction is < 10%

# V. Oxygen Transport

A. Oxygen delivery (DO2)

= the rate of blood flow (cardiac output) x O2 content of blood (CaO2)

= CO x CaO2

= CO x 10 x (Hgb x 1.34 x O2 sat) + 0.003(paO2), where:

- CO is in liters/minute
- Hgb is hemoglobin in g/dL
- 1.34 is the amount of O2 in mL per g of Hgb
- O2 saturation is in decimal form (98% = 0.98)
- 0.003 is the amount of O2 dissolved in plasma, i.e. 0.003 mLO2 per dL plasma, per mmHg paO2
- 10 is the conversion factor to give the result in mL/minute

## 1. The dissolved amount of O2 is negligible, and can be omitted for general use.

a. This is why paO2 is not clinically relevant to tissue oxygenation. Interventions/ventilator adjustments should not be done to "treat" paO2. The paO2 is only a measure of efficiency of gas exchange by the lungs.

- 2. Equation abbreviated to  $DO2 = CO \times 10 \times Hgb \times 1.34 \times O2sat$ 
  - a. Normal CaO2 = 20mL/dL
  - b. DO2 with CO of 5L/min = 5 x 10 x 20 = 1000mL/min

B. O2/Hgb dissociation curve

1. A graphical plot of the oxygen saturation of Hgb (y axis) to oxygen tension/paO2 (x axis)

a. Sigmoidal (S-shaped) curve

b. memory device: "90-60, 60-30, 75-40"

i. each 1st number is O2 sat, 2nd is paO2, so a 90% sat corresponds to a paO2 of 60 mmHg, and so on

2. Curve shifts - certain factors increase or decrease hemoglobin's affinity for O2

a. Rightward shift - decreased affinity for O2; oxygen is released more easily to tissues

i. Need higher paO2 to get same O2 sat;

ii. Think "Right-Release"

iii. Increases in temperature, acidosis, and 2,3-DPG shift curve to right

iv. Think of exercise: body heats up, produces acid (lactate), so curve shifts to right to release more O2 to tissues

b. Leftward shift - increased affinity for O2; oxygen more tightly bound

i. Decreased temperature and 2,3-DPG, and alkalosis shift curve to left

ii. Alkalosis worse for tissue oxygenation based on the curve shift (less offloading of O2 to tissues)