




## Abnormal Ventilation, Abnormal Gas Exchange




## Ventilation and Gas Exchange

- ← Objective: to achieve adequate tissue oxygenation and remove metabolically produced CO<sub>2</sub>.
- ← Ventilation: concerned with delivery of fresh volume of air to gas exchanging units, and the removal of a sufficient volume of mixed gas out
- ← Gas Exchange: the ability to move gas across the alveolar-capillary membrane




## Ventilation and Gas Exchange

- ← The failure of either or both results in impaired arterial blood gases and ultimately to *respiratory failure*.
- ← Ventilatory failure: *Hypercapnic respiratory failure*
- ← Gas exchange failure: *Hypoxemic respiratory failure*
- ← *Hypoxemia is the inevitable result of both*




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  - ← Low O<sub>2</sub> delivery (CaO<sub>2</sub> x C.O.)



## Physiologic Causes of Hypoxemia

Alveolar Hypoventilation

Decreased  $P_{IO_2}$

Diffusion Abnormality

V/Q mismatch

Shunt



## Ventilation

← Minute Ventilation ( $\dot{V}_E$ ) = tidal volume ( $V_T$ ) x respiratory frequency

← Alveolar ventilation ( $\dot{V}_A$ ) = that part of minute ventilation which participates in gas exchange

← Alveolar ventilation = alveolar volume (tidal volume - dead space volume) x respiratory frequency



## Ventilation

- ← Alveolar PCO<sub>2</sub> (PACO<sub>2</sub>) =  $V_{CO_2} / V_A \times K$
- ← VCO<sub>2</sub> = CO<sub>2</sub> production
- ← V<sub>A</sub> = alveolar ventilation
- ← Normal:  $V_{CO_2} / V_A = 1/21.6$ ; K = 863 mmHg)
- ← Alveolar PCO<sub>2</sub> = CO<sub>2</sub> leaving lungs after gas exchange; directly reflects arterial PCO<sub>2</sub>
- ← e.g., halving alveolar ventilation with constant CO<sub>2</sub> production will double the alveolar PCO<sub>2</sub>
- ← e.g., doubling the alveolar PCO<sub>2</sub> reflects halved alveolar ventilation




## Hypoventilation

- ← Inability to inspire and expire a volume of air/gas sufficient to meet metabolic demands
- ← Inability to bring a fresh volume of O<sub>2</sub> with each breath to the gas exchanging unit, and inability to remove CO<sub>2</sub> produced by metabolism.
- ← *Sine qua non*: Increased arterial PCO<sub>2</sub> (PaCO<sub>2</sub>); decreased arterial PO<sub>2</sub> (PaO<sub>2</sub>) breathing room air (*parallel changes!!*)




## Hypoventilation/ Alveolar hypoventilation

- ← All hypoventilation concerns either increased dead space/tidal volume (anatomic or physiologic) : ALVEOLAR HYPOVENTILATION; or
- ← Decreased MINUTE ventilation (decreased tidal volume, decreased respiratory rate)
- ← Increased minute ventilation *may* make up for impaired alveolar ventilation; opposite not true...



## Alveolar Hypoventilation: 2 Clinical Pearls


- ← Does not widen the AaDO<sub>2</sub>
- ← The hypoxemia may be readily ameliorated with supplemental O<sub>2</sub>



## Alveolar Gas Equation

←  $PAO_2 = PIO_2 - PACO_2/R$


←  $PAO_2 = PIO_2 - PACO_2/R + [PCO_2 \times FIO_2 \times 1-R/R]$



## Alveolar Gas Equation


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←  $PIO_2: FIO_2 (P_{atm} - P_{H_2O})$



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- ←  $PACO_2 = PaCO_2$
- ←  $R = \text{Respiratory Exchange Ratio: (gas } R = CO_2 \text{ added to alveolar gas by blood/amount of } O_2 \text{ removed from alveolar gas by blood; low } V/Q = \text{low } R); \text{ normal} = 0.8$



## Case History

← Room air: PaO<sub>2</sub>=30 mmHg, PaCO<sub>2</sub>=90 mmHg, pH=7.08

← PAO<sub>2</sub>= 0.21 (760-47) -90/0.8



## Case History

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← AaDO<sub>2</sub>=7.5 mmHg




## Alveolar Hypoventilation

← CNS: central hypoventilation; infectious,  
traumatic, vascular damage to medullary  
centers; pharmacologic and sleep suppression  
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## Alveolar Hypoventilation

- ← CNS: central hypoventilation; infectious, traumatic, vascular damage to medullary centers; pharmacologic and sleep suppression of ventilatory drive
- ← Peripheral nervous system/myoneural junction: poliomyelitis, Guillain-Barre, myasthenia gravis



## Alveolar Hypoventilation


Respiratory muscles: muscular dystrophy, ALS, increased inspiratory loading (eg emphysema)



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Airway obstruction: upper airway, lower airway

Increased dead space ventilation: pulmonary embolism; COPD



## Hypercapnic Respiratory Failure

← Primary deficit=hypoventilation without gas exchange abnormality, until late



## Physiologic Causes of Hypoxemia

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Decreased  $PIO_2$

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V/Q mismatch

Shunt



## Climbing Everest

←  $P_{atm} = 250$  mmHg

←  $P_{aCO_2} = 18$  mmHg;  $R = 1$

←  $PAO_2 = PIO_2 - PCO_2/R$

←  $PAO_2 = .21 (250 - 47) - 18/1 = 24.6$



## Case History

← RA: PaO<sub>2</sub>=70, PaCO<sub>2</sub>=30 mmHg



## Case History

← RA: PaO<sub>2</sub>=70, PaCO<sub>2</sub>=30 mmHg

← No treatment: RA PaO<sub>2</sub>=50 mmHg,  
PaCO<sub>2</sub>=28 mmHg



## What happened?

←  $PAO_2 = PIO_2 - PACO_2/R$

← 0.21 FIO<sub>2</sub>, PaO<sub>2</sub>=50 mmHg,  
PaCO<sub>2</sub>=28 mmHg

←  $PAO_2 = 0.21(713) - 28/0.8 = 150 - 35 =$   
115 mmHg

←  $AaDO_2 = 115 - 50 = 65$  mmHg




## AaDO<sub>2</sub> and Hypoxemia

← Widened in diffusion disorder, V/Q mismatch, and shunt


← Not widened in alveolar hypoventilation and decreased PIO<sub>2</sub>

← Normal 10-15 mmHg in young adult



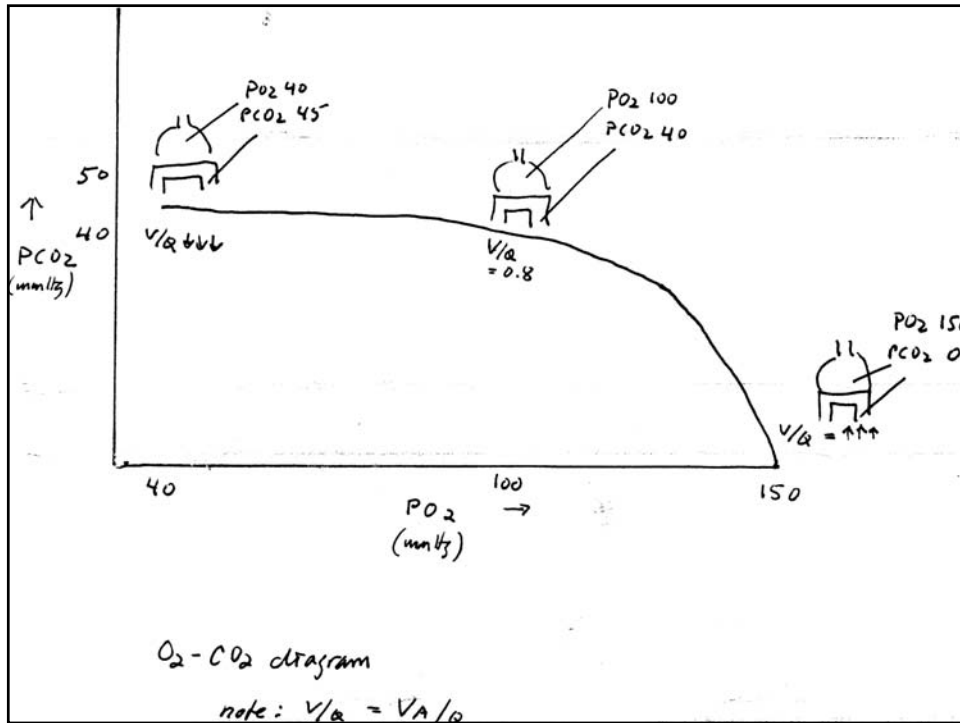
## Hypoxemia

- ← No widening of AaDO<sub>2</sub>: hypoventilation, low P<sub>IO</sub><sub>2</sub>.
- ← Widened AaDO<sub>2</sub>: shunt, low V/Q, low diffusing capacity
- ← Hypoxemia of each may be overcome with supplemental O<sub>2</sub> *except: shunt*.
- ← Note: no gas exchange=no amelioration of hypoxemia with O<sub>2</sub>, whether dead space, shunt, or no diffusion.




## Low V/Q

- ← “Venous admixture”
- ← Alveolar filling: pneumonia, pulmonary edema (cardiogenic/non-cardiogenic)
- ← COPD
- ← Usually will involve some infinitely low V/Q (shunt) and decreased diffusion.




## Low V/Q

- ← Low relationship of V to Q; NOT low ventilation
- ← That is, hypoventilation NOT low V/Q
- ← Low V/Q NOT hypoventilation



## Diffusing Abnormality

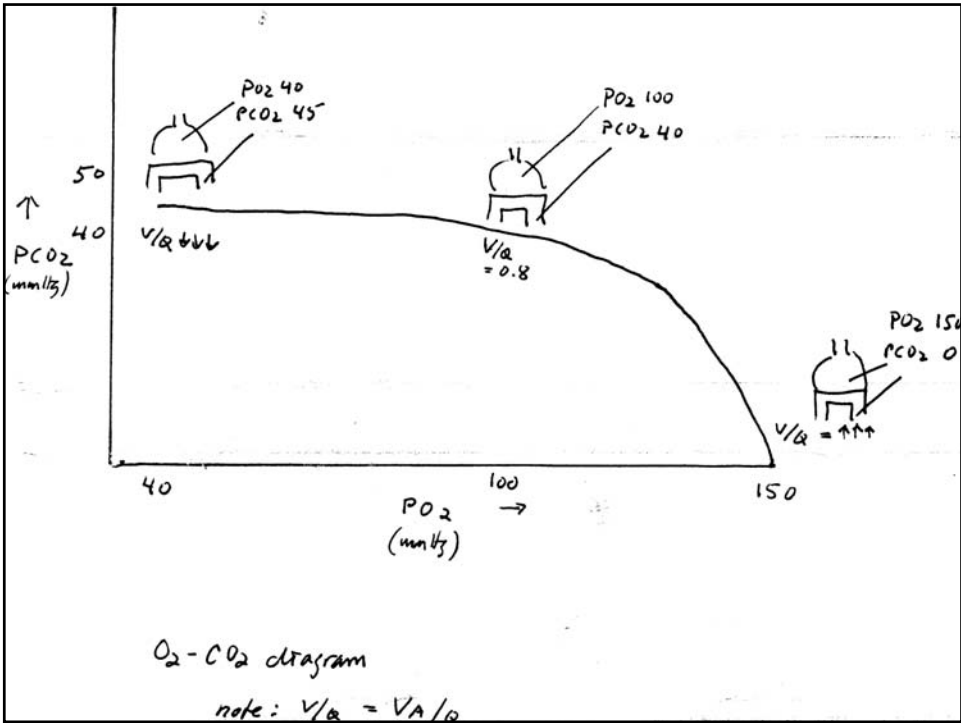
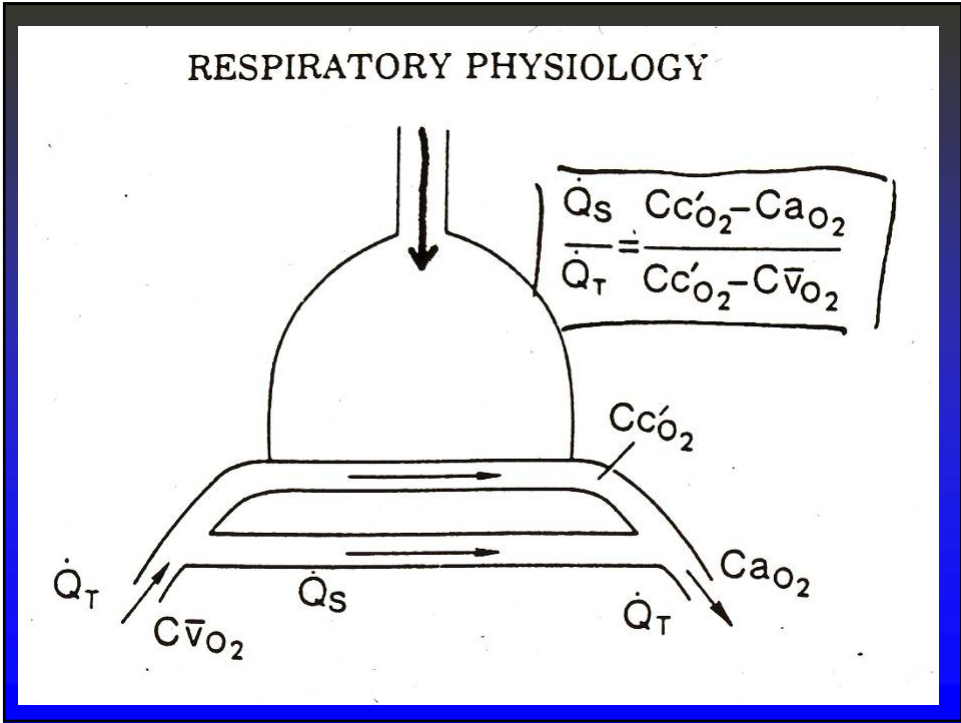
- ← Alveolar-capillary membrane thickening (pulmonary hypertension, pulmonary vasculitis, pulmonary embolism)
- ← Alveolar-capillary membrane destruction (emphysema)
- ← Pulmonary interstitial thickening (pulmonary fibrosis)
- ← Alveolar filling




## Shunt

- ← Infinitely low  $V/Q$
- ← Supplemental  $O_2$  will not raise  $PaO_2$  with large shunt
- ← Clinical examples: ARDS, other severe pneumonia, cardiogenic pulmonary edema
- ← May also be cardiogenic R-L shunt


# RESPIRATORY PHYSIOLOGY





## Hypoxemic Respiratory Failure

- ← Primary deficit=hypoxemia without hypoventilation, until late
- ← Gas exchange abnormality: shunt, low V/Q, low diffusing capacity, all...



## SUMMARY

- ← Hypoventilation: High PaCO<sub>2</sub>, Low PaO<sub>2</sub>, no widening of AaDO<sub>2</sub>
- ← Gas exchange abnormality: Low PaO<sub>2</sub>, normal to low PaCO<sub>2</sub>, widened AaDO<sub>2</sub>
- ← Hypoxemia of all hypoventilation and gas exchange abnormalities may be sufficiently overcome by supplemental O<sub>2</sub> unless gas exchange abnormality or hypoventilation is *absolute* (eg *shunt* or *dead space*)