Abnormal Ventilation, Abnormal Gas Exchange

Ventilation and Gas Exchange

- Objective: to achieve adequate tissue oxygenation and remove metabolically produced CO_2.
- 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 *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_2 delivery (CaO_2 x C.O.)

Physiologic Causes of Hypoxemia

Alveolar Hypoventilation

Decreased PIO_2

Diffusion Abnormality

V/Q mismatch

Shunt

Ventilation

- Minute Ventilation (V_E)=tidal volume (V_T) x respiratory frequency
- Alveolar ventilation (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_2 ($PACO_2$)=VCO_2/V_A x K
- VCO_2=CO_2 production
- V_A=alveolar ventilation
- Normal: VCO_2/V_A=1/21.6; K=863 mmHg)
- Alveolar PCO_2=CO_2 leaving lungs after gas exchange; directly reflects arterial PCO_2
- e.g., halving alveolar ventilation with constant CO_2 production will double the alveolar PCO_2
- e.g., doubling the alveolar PCO_2 reflects halved alveolar ventilation

Hypoventilation

- Inability to inspire and expire a volume of air/gas sufficient to meet metabolic demands
- Inabilty to bring a fresh volume of O_2 with each breath to the gas exchanging unit, and inability to remove CO_2 produced by metabolism.
- *Sine qua non:* Increased arterial PCO_2 ($PaCO_2$); decreased arterial PO_2 (PaO_2) breathing room air *(parallel changes!!)*

Hypoventilation/ Alveolar hypoventilation

- All hypoventilation concerns either :
- increased dead space/tidal volume (anatomic or physiologic), or
- Decreased MINUTE ventilation (decreased tidal volume, and/or decreased respiratory rate)
- Each is considered alveolar hypoventilation if PaCO2 is elevated.

Alveolar Hypoventilation: 2 Clinical Pearls

- Does not widen the $AaDO_2$
- The hypoxemia may be readily ameliorated with supplemental O_2

Alveolar Gas Equation

- PAO2=PIO2 – PACO2/R
- PAO2=PIO2 – PACO2/R + [PCO2 x FIO2 x 1-R/R]

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- R=Respiratory Exchange Ratio: (gas R=CO2 added to alveolar gas by blood/amount of O2 removed from alveolar gas by blood; low V/Q=low R); normal=0.8

Case History

- Room air: PaO2=30 mmHg, PaCO2=90 mmHg, pH=7.08
- PAO2= 0.21 (760-47) –90/0.8

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- AaDO2=7.5 mmHg

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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

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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
- Hypoxemia MUST result

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Diffusion Abnormality

V/Q mismatch

Shunt

Climbing Everest (Decreased PIO2)

- P atm= 250 mmHg
- PaCO2=18 mmHg; R=1
- PAO2=PIO2-PCO2/R
- PAO2=.21 (250-47)-18/1=24.6

Case History

- RA: PaO_2=70, $PaCO_2$=30 mmHg

Case History

- RA: PaO_2=70, $PaCO_2$=30 mmHg
- No treatment: RA PaO_2=50 mmHg, $PaCO_2$=28 mmHg

What happened?

- $PAO_2=PIO_2 - PACO_2/R$
- 0.21 FIO2, PaO_2=50 mmHg, $PaCO_2$=28 mmHg
- PAO2=0.21(713)-28/0.8=150-35= 115 mmHg
- AaDO2=115-50= 65 mmHg

AaDO2 and Hypoxemia

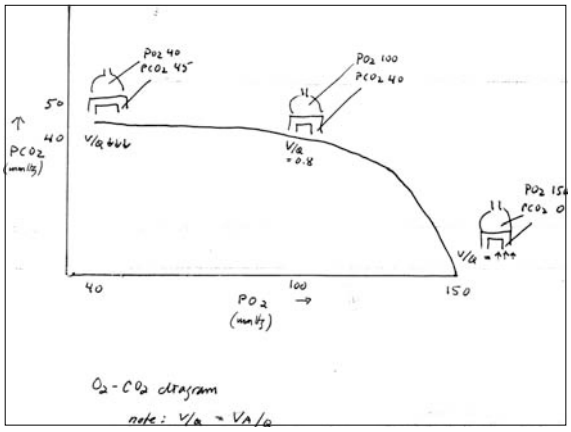
- Widened in diffusion disorder, V/Q mismatch, and shunt
- Not widened in alveolar hypoventilation and decreased PIO2
- Normal 10-15 mmHg in young adult

Hypoxemia

- No widening of $AaDO_2$: hypoventilation, low PIO_2.
- Widened AaDO2: shunt, low V/Q, low diffusing capacity
- Hypoxemia of each may be overcome with supplemental O2 *except: shunt*.
- Note: no gas exchange=no amelioration of hypoxemia with O_2, whether dead space, shunt, or no diffusion.

Low V/Q

- "Venous admixture"
- Alveolar filling: pneumonia, pulmonary edema (cardiogenic/non-cardiogenic)
- COPD a common situation of low V/Q
- Usually will involve some infinitely low V/Q (shunt) and decreased diffusion.



Low V/Q

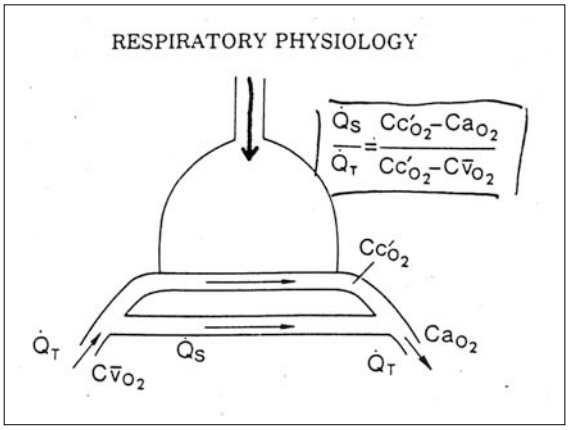
- Low relationship of V to Q; NOT low ventilation
- Low V/Q is NOT hypoventilation (unless all units are the same low V/Q)

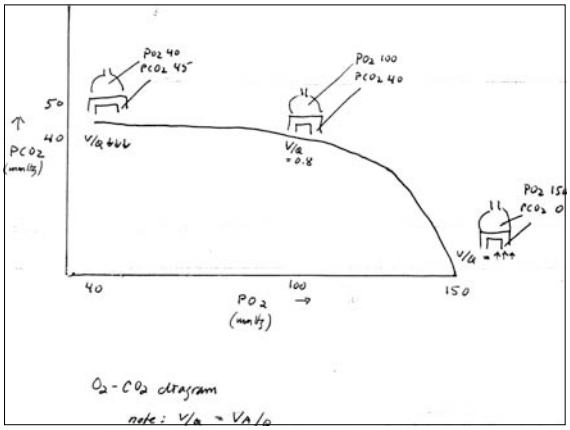
Diffusion Abnormality

- 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 O2 will not raise PaO2 with large shunt
- Clinical examples: ARDS, other severe pneumonia, cardiogenic pulmonary edema
- May also be cardiogenic R-L shunt





Hypoxemic Respiratory Failure

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

SUMMARY

- Hypoventilation: High PaCO2, Low PaO2, no widening of AaDO2
- Gas exchange abnormality: Low PaO2, normal to low PaCO2, widened AaDO2
- Hypoxemia of all hypoventilation and gas exchange abnormalities may be sufficiently overcome by supplemental O2 unless gas exchange abnormality is *absolute (eg shunt)*