

NORMAL MATERNAL RESPIRATORY CHANGES in PREGNANCY

FLAME LECTURE: 25

BURNS 5.14.19

LEARNING OBJECTIVES

- ▶ Discuss the maternal physiologic and anatomic changes associated with pregnancy
- ▶ Prerequisites: NONE
- ▶ See also:
 - ▶ FLAME 24 – Normal cardiovascular changes in pregnancy
 - ▶ FLAME 89 – Asthma in pregnancy

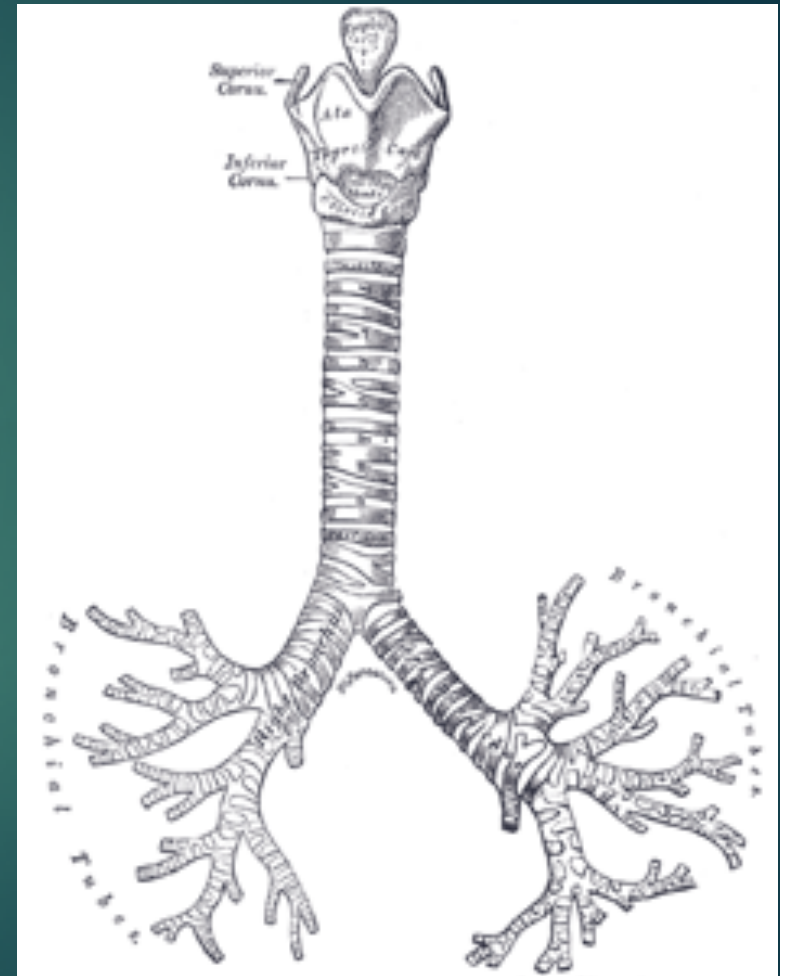
NORMAL RESPIRATORY PHYSIOLOGY

CELL TYPES

- ▶ Capillary endothelium
- ▶ Alveolar Type I pneumocytes: makes up 90-95% of alveolar surface, blood-gas exchange, highly susceptible to damage
- ▶ Alveolar Type II pneumocytes: produce surfactant, can transform into type I if type I's are damaged
- ▶ Alveolar macrophages: innate immune response to pathogens that evaded the lung's primary mechanical defenses
- ▶ PMN's: not usually seen, only in smokers/inflammation
- ▶ Mast/Clara/Club cells: secretagogue sentinel immune and exocrine cells

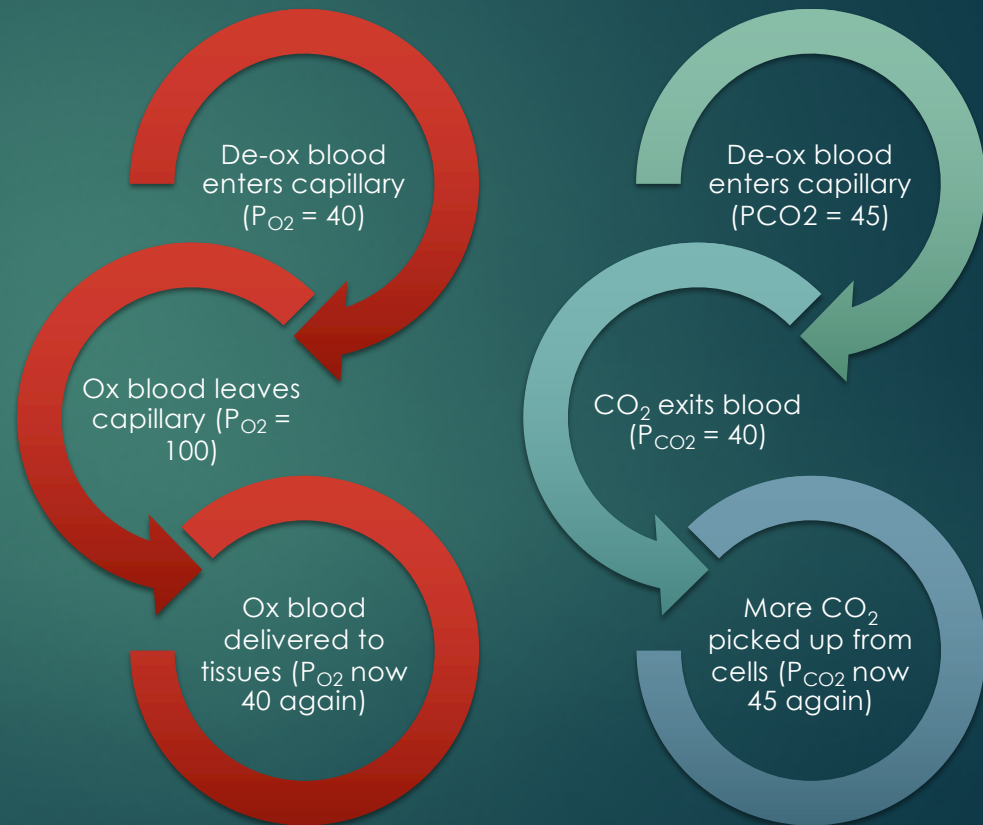
NORMAL GAS EXCHANGE

- ▶ **Conducting zone:** anatomic dead space (no gas exchange)
 - ▶ Trachea → terminal bronchioles
- ▶ **Respiratory zone:** where gas exchange occurs
 - ▶ Respiratory bronchioles → alveolar sacs



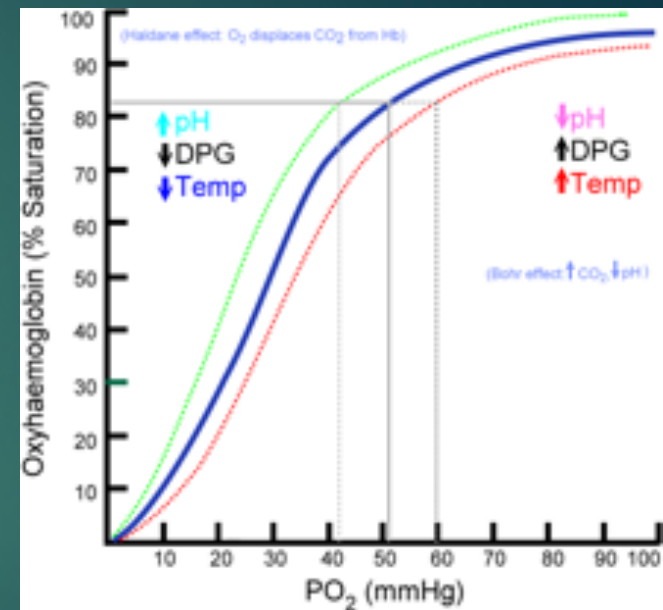
NORMAL GAS EXCHANGE

- ▶ Gas exchange (via diffusion) is driven by partial pressure differences of O_2 & CO_2 in alveoli vs. blood



NORMAL GAS TRANSPORT IN BLOOD

- ▶ 98.5% of O₂ is bound to Hgb / 1.5% is dissolved in plasma
 - ▶ Recall hemoglobin binding curve. Hgb binds O₂ in a sigmoidal distribution so that the more O₂ molecules bound, the more favorable it is to bind another O₂ molecule
 - ▶ However, Hgb has the capacity to release more oxygen if the tissue's (OR FETUS') needs are greater (**right shift**)
 - ▶ Conversely, Hgb can withhold more O₂ if tissue needs are decreased (**left shift**)
- ▶ Carbon Dioxide transport:
 - ▶ Bicarbonate (HCO₃) – 60%,
Carbaminohemoglobin (bound to Hgb) – 30%,
Dissolved in plasma – 10%



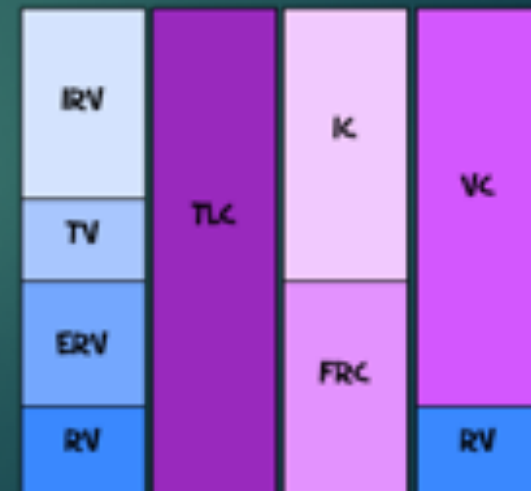
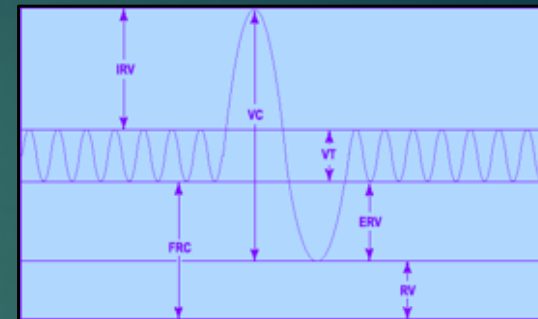
Factors that shift the curve right (more O₂ released):

- < pH
- > DPG
- < body temp

NORMAL LUNG VOLUMES

THIS IS JUST A REVIEW, DON'T GET BOGGED DOWN IN THE DETAILS

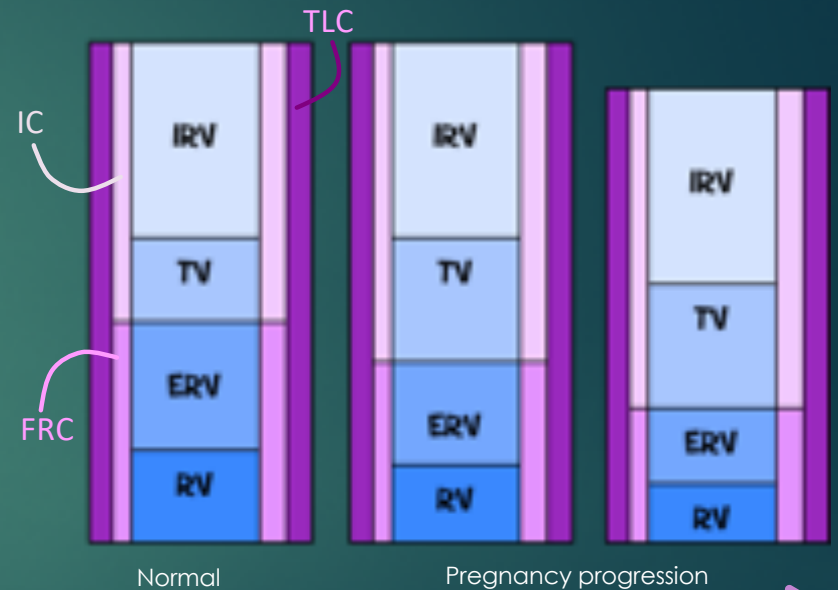
- ▶ The lung has 4 different volumes:
 - ▶ **IRV** (inspiratory reserve volume) - maximal volume that can be inhaled from the end-inspiratory level
 - ▶ **TV** (tidal volume) - volume of air moved into or out of the lungs during quiet breathing
 - ▶ **ERV** (expiratory reserve volume) - maximal volume of air that can be exhaled from the end-expiratory position
 - ▶ **RV** (residual volume) - volume of air remaining in the lungs after a maximal exhalation
- ▶ 2 or more volumes together = a capacity:
 - ▶ **TLC**: total lung capacity = IRV + TV + ERV + RV
 - ▶ **VC**: vital capacity = IRV + TV + ERV
 - ▶ **IC**: inspiratory capacity = IRV + TV
 - ▶ **FRC**: functional reserve capacity = ERV + RV
- ▶ **FEV₁**: volume of air expired in 1st second of forceful expiration
- ▶ **Minute ventilation**: volume of air inspired/expired per minute
 - ▶ Can increase with deeper breaths or more frequent breaths



LUNG VOLUMES IN PREGNANCY

THE CHANGES BELOW AREN'T OVERWHELMINGLY IMPORTANT CLINICALLY!

- ▶ **RV**, **ERV** (and thus **FRC**) all decrease due to uterus compressing + elevating diaphragm
- ▶ **VC** and **TLC** maintained until late pregnancy
 - ▶ **IC** initially increases slightly to offset FRC decrease and maintain TLC (2nd diagram)
- ▶ **FVC*** and **FEV₁** are UNCHANGED during pregnancy
 - ▶ ***VC** can be measured as slow **vital capacity (VC)** or **forced vital capacity (FVC)**; **VC** decreases because diaphragm elevation affects **ERV** passively, but **FVC** doesn't change because diaphragm excursion doesn't change with a forced exhalation
- ▶ Thus, changes in **FVC** and **FEV₁** suggest underlying pulmonary pathology (asthma, bronchitis, COPD)
 - ▶ ↓ FVC/FEV₁ = obstructive pulmonary dz
 - ▶ ↑ FVC/FEV₁ = restrictive pulmonary dz



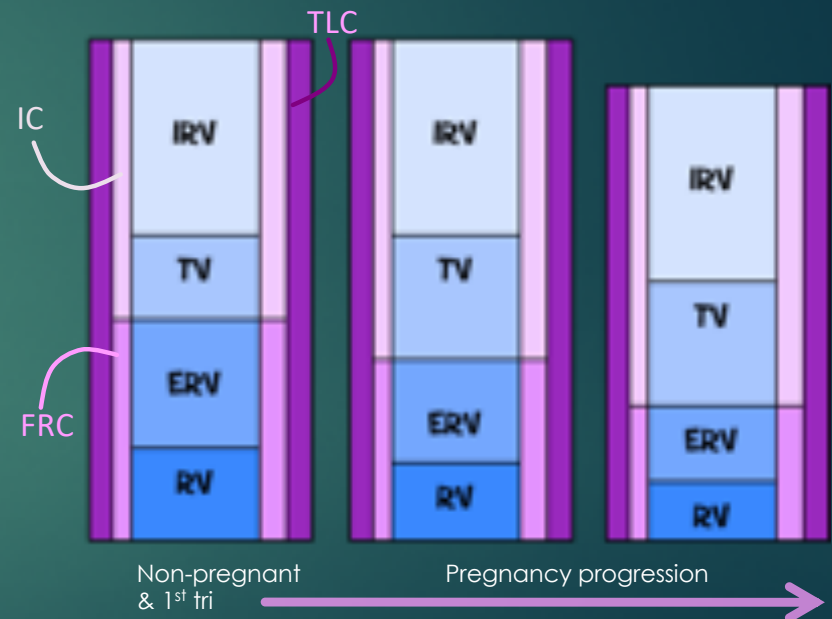
First, RV, ERV, & FRC decrease but TV (and therefore IC) compensate so no change in TLC

Later, diaphragm elevation from enlarging uterus. TV can't compensate, so TLC also decreases

LUNG VOLUMES IN PREGNANCY

NOTE: TIDAL VOLUME & MINUTE VENT ARE MORE IMPORTANT!

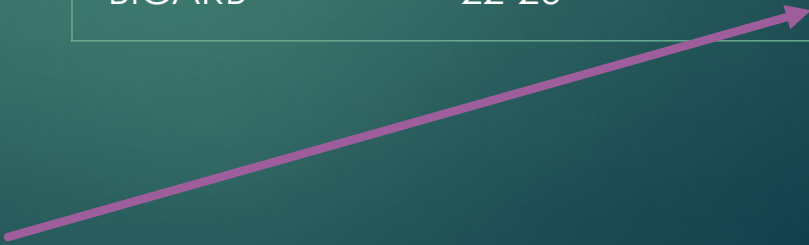
- ▶ **TV** increases in pregnancy, resulting in increased minute ventilation
 - ▶ Progesterone stimulates respiratory drive by lowering the respiratory drive center threshold to CO_2 , allowing for an increase in ventilation
 - ▶ Thus, minute ventilation is increased through **more volume in/out with each breath** and *not* by increased respiratory rate (RR largely remains unchanged in pregnancy)
 - ▶ Also, the chest wall broadens to facilitate increased TV
 - ▶ This occurs both from the expanding abdomen and via the same protein *relaxin* that allows for relaxation of pelvic ligaments to expand pelvis



CHANGES IN BLOOD GASSES

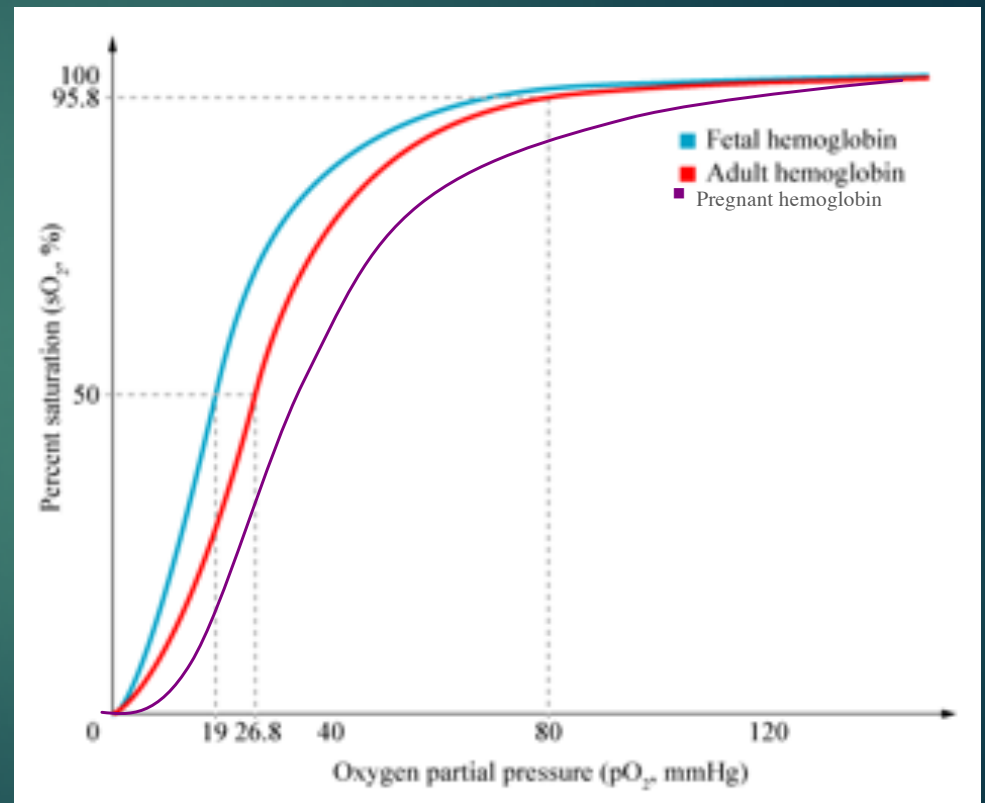
- ▶ The increase in **minute ventilation** allows for more CO_2 to be blown off causing a mild **respiratory alkalosis**
- ▶ **AND WHY IS PROGESTERONE MEDDLING IN RESPIRATORY PHYSIO?**
 - ▶ Because the fetus not only depends upon the maternal respiratory system for obtaining O_2 , but also for CO_2 excretion; decreased maternal P_{CO_2} creates a gradient that allows the fetus to offload CO_2
 - ▶ This is partially compensated with increased maternal renal loss of HCO_3^- , but blood gasses are still shifted during pregnancy

ABG	NON-PREGNANT	PREGNANT
pH	7.38-7.42	7.39-7.45
p CO_2	38-42	25-33
p O_2	90-100	92-107
BICARB	22-26	16-22



RESPIRATORY ALKALOSIS

- ▶ Consider again the hemoglobin binding curve!
- ▶ Maternal respiratory alkalosis causes a right shift so that Hgb binds less tightly to O_2
 - ▶ At the same time, fetal hemoglobin has a higher binding affinity for O_2
 - ▶ Thus, fetal blood can draw more O_2 from maternal blood as maternal Hgb releases more O_2



TAKE HOME POINTS

1. The progesterone-mediated increase in minute ventilation allows mom to blow off more CO_2 thus becoming more alkalotic at baseline. This alkalosis is what drives efficient O_2 and CO_2 exchange between mom and baby.
2. With exception of the above, most pregnancy-related anatomic and physiologic changes are not of great clinical importance to mom, UNLESS she gets sick!

PREVIEW TO PATHOLOGIC LECTURES:

1. When pulmonary pathology arises in mom (asthma, CAP, etc), gas exchange will be impaired, and she will start to retain CO_2 . Thus, her pH will first normalize to that of a non-pregnant patient before becoming more acidotic
2. Thus, it is of critical importance to understand that:
 1. A pH in the normal range of a non-pregnant patient is NOT normal for mom. Do not miss her early decline in respiratory status, or she can spiral downwards
 2. If mom is increasingly retaining CO_2 , baby is also becoming acidemic! Because now the gradient is not allowing for baby to offload CO_2 either



REFERENCES

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2. *Normal Maternal Physiology: Implications for Prenatal Care* University of Utah, Department of OB/GYN
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4. Soma-Pilay P, et al. Physiological changes in pregnancy. *Cardiovasc J Afr.* 2016; 27(2):89-94.
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