NORMAL MATERNAL RESPIRATORY CHANGES in PREGNANCY

FLAME LECTURE: 25
BURNS 5.2.18
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: makes up 90-95% of alveolar surface, blood-gas exchange, highly susceptible to damage
- Alveolar type II: produce surfactant, can transform into type I if type I’s are damaged
- Alveolar macrophages: immune function
- PMN’s: not usually seen, only in smokers/inflammation
- Mast cells
- Clara/Club cells: release secretory proteins to protect bronchioles
NORMAL GAS EXCHANGE

- **Conducting zone:** anatomic dead space (no gas exchange)
  - Trachea ➔ terminal bronchioles

- **Respiratory zone:** where gas exchange occurs
  - Respiratory bronchioles ➔ alveolar sacs
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 tissue (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 ARENT 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
- 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 effects ERV passively, but FVC doesn’t change because diaphragm excursion doesn’t change with a forced exhalation
  - Changes in FVC and FEV1 suggest underlying pulmonary pathology (asthma, bronchitis, COPD)
    - ↓ FVC/FEV1 = obstructive pulmonary dz
    - ↑ FVC/FEV1 = restrictive pulmonary dz

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TV increases in pregnancy, resulting in increased minute ventilation

- Progesterone stimulates respiratory drive by increasing chemosensitivity to $\text{CO}_2$, allowing for 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 CO₂ 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 O₂, but also for CO₂ excretion; decreased maternal P₇CO₂ creates a gradient that allows the fetus to offload CO₂.
- This is partially compensated via increased maternal renal loss of HCO₃, but blood gasses are still shifted during pregnancy.

<table>
<thead>
<tr>
<th>ABG</th>
<th>NON-PREGNANT</th>
<th>PREGNANT</th>
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<tbody>
<tr>
<td>pH</td>
<td>7.38-7.42</td>
<td>7.39-7.45</td>
</tr>
<tr>
<td>pCO₂</td>
<td>38-42</td>
<td>25-33</td>
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<tr>
<td>pO₂</td>
<td>90-100</td>
<td>92-107</td>
</tr>
<tr>
<td>BICARB</td>
<td>22-26</td>
<td>16-22</td>
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</tbody>
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RESPIRATORY ALKALOSIS

- Consider again the dissociation curve!

- Maternal respiratory alkalosis → alkalosis-stimulated ↑ in 2,3-DPG → a right shift so that Hgb binds less tightly to O₂
  - At the same time, fetal hemoglobin has a higher binding affinity for O₂
  - Thus, fetal blood can draw more O₂ from maternal blood as maternal Hgb releases more O₂
OXYGEN CONSUMPTION

- Concomitant with the increase in maternal minute ventilation is a 20-40% increase in maternal oxygen consumption. This is caused by both the increased oxygen demands of the fetus, placenta, and uterus.
- In addition to the other physiologic changes noted, this increased oxygen consumption makes pregnant women more susceptible to early decompensation.
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 & CO\(_2\) 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

1. UpToDate
2. Normal Maternal Physiology: Implications for Prenatal Care University of Utah, Department of OB/GYN
3. Clinical Gate – Neonatal-Perinatal Medicine Ch. 10 Respiratory System