Ch. 12: Respiratory Physiology

Objectives:
1. Review respiratory anatomy.
2. Understand mechanics of breathing.
3. Learn lung volumes & respiratory vocabulary
4. Learn gas exchange at lungs & at body tissues
5. Learn autonomic regulation of respiration.
7. Respiratory control of acid/base balance.

Anatomy of Respiratory System

2 Zones of Respiratory System:
1) Conduction Zone = from oral/nasal cavities to terminal bronchioles

2) Respiratory Zone = from respiratory bronchioles to alveoli.
2) Respiratory zone

Respiratory bronchioles = smallest bronchioles, branch from tertiary bronchioles.

Alveolar sacs = honey-comb shaped, 1-cell thick sacs for gas exchange.
[~300 mill in lungs! ~760 sq ft area!]

How gases are exchanged w/blood

Surrounded by arterial & venous capillaries ("capillary plexus") for gas exchange between alveoli & blood.
2 Types Alveolar Cells:

Type 1 Alveolar Cells = 97% of total lung surface area where most gas exchange occurs.

Type 2 Alveolar Cells = secrete surfactant

Surfactant↓ surface tension from water lining alveolar sacs preventing walls from collapsing during exhalation.

Surfactants ↓ intra-alveolar pressure & prevent collapse

Infant Respiratory Distress Syndrome (IRDS) ---- Clinical Application Pg 540

- 1st breath baby takes must overcome tremendous pressure (20X that of all future breaths!)
- Surfactant is produced > 28 weeks (7-8 months)
- Babies are born < 28 wks - not enough surfactant. Results in collapsed alveoli
- Tx = synthetic surfactant delivered into baby’s lungs & mechanical ventilator.

Acute Respiratory Distress Syndrome (ARDS) Pg 540

- Due to inflammation from infection (septic shock)
- Results in protein (serum) secretion in lungs. Fluid dilutes surfactant
- ↓ lung compliance
Refresher of the thoracic cavity:

Membranes of the lungs:

- **Visceral pleura**
  Covers the lungs

- **Parietal pleura**
  Lines the pleural cavities

Lungs normally fill thoracic cavity, pleura pressed together.
- Serous fluid layer between pleura decreases friction

Parietal pleura held tight against thoracic wall by surface tension of water layer.
- As thoracic cage changes volume (with breathing) so do lungs.

### 2. Mechanics of Respiration

1) **Air moves from high to low pressure**
- Depends on where pressure is greatest
- Atmospheric air pressure = constant (760 mmHg)
- Lung air pressure depends on volume of thoracic cavity

2) **Air pressure in lungs (closed chamber) changes with volume of chamber**

   “Boyle’s Law” =  
   as volume of closed chamber ↑, air pressure within ↓  
   as volume of closed chamber ↓, air pressure within ↑

**Translates to lung volume & air pressure within lungs (“intrapulmonary pressure”):**

- As thoracic volume ↑  lung volume ↑ & intrapulmonary pressure ↓
- As thoracic volume ↓  lung volume ↓ & intrapulmonary pressure ↑
**Boyle’s Law**

Chamber volume larger
BUT air pressure lower

Chamber volume smaller
BUT air pressure higher

**Muscle activity changes thoracic volume**

<table>
<thead>
<tr>
<th></th>
<th>Inspiration</th>
<th>Expiration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intrapulmonary pressure (mmHg)</td>
<td>-3</td>
<td>+3</td>
</tr>
<tr>
<td>Intrapleural pressure (mmHg)</td>
<td>-6</td>
<td>-3</td>
</tr>
<tr>
<td>Transpulmonary pressure (mmHg)</td>
<td>+3</td>
<td>+6</td>
</tr>
</tbody>
</table>

*Note: Pressures indicate mmHg below or above atmospheric pressure.*
Gas Pressure Vocabulary:

- **Intrapulmonary pressure** = pressure inside lungs
  - *During inhalation* – is lower than atmospheric pressure (-3 mmHg)
  - *During exhalation* – is above atmospheric pressure (+3 mmHg)

- **Intrapleural pressure** = pressure between the pleural membranes due to elastic recoil (parietal pleura sticks to wall)
  - *During inhalation* – is lower than atmospheric (-6 mmHg)
  - *During exhalation* – is still lower atmospheric (-3 mmHg)

- **Transpulmonary pressure** = difference between intrapulmonary & intrapleural pressure (is ALWAYS above atmospheric pressure).

Physical properties of the lungs:

A) **Surface tension** = pressure resulting from thin film of water lining alveoli that resists their expansion.

  **Law of Laplace & surface tension in alveoli:**
  - Pressure is greater the smaller alveoli become (w/exhalation).
  - Pressure is smaller in larger alveoli (w/inhalation).

B) **Compliance** = ability of lungs to expand with skeletal muscle activity.
[Allows changes in thoracic volume to change lung volume.]
  - more lung compliance = greater capacity for “stretchiness”
  - less lung compliance = less capacity for “stretchiness”

C) **Elasticity/Recoil** = tendency of lungs to return to normal shape after either inflating (inhalation) or deflating (exhaling).
A) Surface Tension & Law of La Place

“Law of LaPlace” = air pressure within alveolar sac depends on surface tension and size of alveolar sac:

- Large alveoli – have lower surface tension & air pressure within them (expanding with air is easier w/large alveoli)
- Small alveoli – have higher surface tension & air pressure within them. (expanding with air is harder w/small alveoli)

**anything that functionally ↓ alveolar size:
- ↑ surface tension & intra-alveolar pressure
- ↓ gas exchange &
- ↓ respiratory capacity

Cystic Fibrosis (Clinical App Pg 136)
- Genetic disorder affecting Cl- channels on alveoli membrane.

Results in buildup of mucus within alveoli causing:
- ↓ decreased functional alveolar size
- ↑ surface tension & intra-alveolar pressure (harder for alveoli to expand)
- ↓ with gas exchange
- Warmth & moisture (mucus) aids bacterial growth. (Vulnerable to pneumonia)
B) Lung compliance

Factors that increase compliance:
- pulmonary “surfactants”

Disease:
Ex: Emphysema = causes excessive lung compliance!
Destroyed alveoli & alveolar walls weakened,
- Alveoli expand easily, but also collapse easily.

Factors that decrease compliance:
- Pulmonary fibrosis = buildup of fibrous tissue in lungs stiffens them (Ex. from breathing in small particles that accumulate in lungs):
  
  Ex: Silicosis = (inhalation of fine glass, rock, or sand particles)
  Ex: Anthracosis (black lung disease) = inhalation of coal dust.

CLINICAL APPLICATIONS

If air enters the intrapleural space and thereby raises the intrapleural pressure, the difference in pressure between the inside of the lungs (intrapulmonary pressure) and the outside of the lungs (intrapleural pressure) is abolished. As a result, the lung is no longer stuck to the thoracic wall; this is like releasing a stretched rubber band, and the lung’s elastic recoil causes it to collapse. The condition of air entering the intrapleural space and causing the collapse of a lung is known as a pneumothorax. Fortunately, a pneumothorax usually causes only one lung to collapse, because each lung is contained in a separate pleural compartment.
“pneumothorax” = a chest wound ("traumatic pneumothorax") causes air from inside the lungs enters intrapleural space. Trapped air presses inward on lung – collapsing it.

- intrapleural pressure ↑ to equal that of atmospheric pressure.

***No pressure gradient between atmospheric air pressure & intraplural pressure.

Result = can’t get air to enter lungs!

Review

- The respiratory system
  – The respiratory zone (respiratory bronchioles & alveoli)
    • External respiration, internal respiration
  – The conducting zone (oral cavity to 3° bronchioles)
    • Warming/humidification, filtration, cleaning
  – Ventilation, gas exchange, oxygen utilization
    • Intrapulmonary pressure, intrapleural pressure, transpulmonary pressure
    • Boyle’s Law
- Physical properties of the lungs
  – Lung compliance, elasticity, & surface tension
    • Role of surfactant
Spirometry = clinical evaluation of pulmonary (respiratory) function, which allows diagnosis of lung disorders.

### TABLE 12.1  Terms Used to Describe Lung Volumes and Capacities

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lung Volumes</td>
<td>The four nonoverlapping components of the total lung capacity</td>
</tr>
<tr>
<td>Tidal volume</td>
<td>The volume of gas inspired or expired in an unforced respiratory cycle</td>
</tr>
<tr>
<td>Inspiratory reserve volume</td>
<td>The maximum volume of gas that can be inspired during forced breathing in addition to tidal volume</td>
</tr>
<tr>
<td>Expiratory reserve volume</td>
<td>The maximum volume of gas that can be expired during forced breathing in addition to tidal volume</td>
</tr>
<tr>
<td>Residual volume</td>
<td>The volume of gas remaining in the lungs after a maximum expiration</td>
</tr>
<tr>
<td>Lung Capacities</td>
<td>Measurements that are the sum of two or more lung volumes</td>
</tr>
<tr>
<td>Total lung capacity</td>
<td>The total amount of gas in the lungs after a maximum inspiration</td>
</tr>
<tr>
<td>Vital capacity</td>
<td>The maximum amount of gas that can be expired after a maximum inspiration</td>
</tr>
<tr>
<td>Inspiratory capacity</td>
<td>The maximum amount of gas that can be inspired after a normal tidal expiration</td>
</tr>
<tr>
<td>Functional residual capacity</td>
<td>The amount of gas remaining in the lungs after a normal tidal expiration</td>
</tr>
</tbody>
</table>
Additional Respiratory Vocabulary:

**Apnea** = absence of breathing  
**Dyspnea** = labored or difficult breathing  
**Eupnea** = normal breathing at rest  
**Hyperventilation** = excessively rapid ventilation (will decrease alveolar CO2)  
**Hypoventilation** = low ventilation (will increase alveolar CO2)  
**Pneumothorax** = presence of gas in intrapleural space causing lung collapse

**Respiratory Disorders**

**Chronic Obstructive Pulmonary Disease (COPD)**
= chronic bronchiole inflammation (bronchitis)
- Leads to bronchiole scar-tissue (fibrosis), which narrows bronchioles (bronchoconstriction)
- mucus buildup ↑ resistance & ↓ compliance  
- ↓ expiratory reserve volume  
  (90% SMOKERSI)

**Asthma** = inflammation of bronchioles.
- Bronchoconstriction (bronchospasms)
- Tx= anti-inflammatories & epinephrine as bronchodilator (or B2 agonist *albuterol* – rescue inhaler).
Lung damage from smoking causes chronic bronchitis and formation of scar tissue (fibrosis)
4. Basics of Gas Exchange at Lungs and at Body Tissues

Gas exchange between 2 structures is dependent on pressure gradient of dissolved O2 & CO2

* Gas moves from side with higher pressure (from dissolved gases) to side with lower pressure & visa versa

*Gas wants to move “downhill” from high to low pressure!

Gas exchange between lung alveoli & pulmonary vessels:

> Alveolar PO2 = 105 mmHg, higher than that in pulmonary arteries (40 mmHg)

> Alveolar PCO2 = 40 mmHg, lower than that in pulmonary arteries (46 mmHg)
Gas exchange between systemic capillaries & tissues:

> Tissue PO$_2$ (<100 mmHg) = lower than O$_2$-rich arterial blood (100 mmHg)

> Tissue PCO$_2$ (>40 mmHg) = higher than that in arterial blood (40 mmHg)

**Review**

- Pulmonary function tests (spirometry)
- Alveolar PO$_2$ lower than atmospheric
- Gas exchange at tissues & at alveoli of lungs
  Depends on differences in partial pressures of O$_2$ and CO$_2$
Motor neurons from 2 brain areas control breathing muscles:

1) Voluntary Breathing
   = primary motor cortex of frontal cerebral lobe.

2) Involuntary Breathing = Medulla – respiratory center regulates respiratory rate.  
                            Pons  – apneustic center (stimulate inhalation)  
                            – pneumotaxic center (inhibit inhalation)

What happens to minute ventilation after:

• Hypoventilation?

• Hyperventilation?

• Exercise?
Autonomic motor control breathing involves:

Chemoreceptors:

- Aorta & carotid artery chemoreceptors
- Medulla chemoreceptors

6. Effect of Hemoglobin on Gas Transport

Hemoglobin =

- 4 protein chains w/4 iron-containing heme (pigments)
- Each heme group binds with 1 O2 molecule
- Each RBC has ~280 million hemoglobin molecules (each RBC can carry ~billion O2 molecules! (4 X 280 million)
- Hemoglobin bound to O2 = “oxyhemoglobin” (Arterial blood 97% saturated w/oxyhemoglobin = bright red)
- Hemoglobin lacking O2 = “deoxyhemoglobin” (venous blood dull red or maroon)
Hemoglobin Disorders:

**Carbon Monoxide** = odorless, color-less gas that binds w/hemoglobin to create **carboxyhemoglobin** in RBCs.

Carboxyhemoglobin has lower affinity for O2.

**Result:**
> Hypoxia (called carboxyhemoglobinemia)
> Death
**Methemoglobinemia** = disorder in which hemoglobin’s iron (a component of heme) is “ferric” rather than “ferrous”.
> this hemoglobin called **methemoglobin** (pronounce as “met-hemoglobin”)
> Methemoglobin has ↓ ability to release (unload) O2 at tissues.
> Patients are hypoxic & **BLUE**!

“**Blue baby syndrome**” = babies turn blue (hypoxia) from drinking milk made w/nitrate contaminated water. Nitrate causes formation of methemoglobin.

**Neonatal jaundice** (Clinical App Pg 563)
- At birth switch from **hemoglobin-F** (fetal) to **hemoglobin-A** (adult)
- Body removes RBCs with hemoglobin f.
- Liver removes biliruben from destroyed hemoglobin f.
- Liver sometimes not mature enough to remove biliruben.
- Biliruben builds up.
- Baby turns yellow. (happens in up to 50% newborns)

**Treatment:**
“blue light exposure” – breaks biliruben down to water-soluble form excreted by kidneys.
**Sickle Cell Anemia** = homozygous recessive condition in which body produces RBCs with **hemoglobin-S** rather than **hemoglobin-A**.
- Hemoglobin-S turns RBCs into sickle-shape.
- Sickled RBCs carry less O2 (cause hypoxia)
- Sickled RBCs tend to form clots (thrombus)
- Patients more prone to embolism.
- More prone to ischemic events.
Review

• Regulation of breathing
  – Medulla & pons
• Chemoreceptors
  – central, peripheral
• Hemoglobin O₂ transport:
  – Oxyhemoglobin & deoxyhemoglobin
  – Abnormal hemoglobin (carboxyhemoglobin, methemoglobin)
  – Neonatal jaundice
  – Sickle cell

7. Respiratory Control of Acid/Base Balance

Normal Blood pH = 7.35 – 7.45
Blood pH maintained by buffering CO₂ with HCO₃⁻

Blood Acid/Base balance has 2 components:

1) Respiratory component = where CO₂ (a volatile acid) in blood eliminated by lungs (exhalation).
   - Increased respiratory rate ↑ blood pH.
   - Decreased respiratory rate ↓ blood pH.

2) Metabolic component = non-volatile acids in blood (i.e. lactic acid, fatty acids, ketones) eliminated by liver, kidneys, or other organs.
Acidosis = increased acids in blood (pH below 7.35)
Alkalosis = decreased acids in blood (pH above 7.45)

Respiratory acidosis = ↓ blood pH due to ↓ respiratory rate (hypoventilation) – not enough CO2 waste exhaled by lungs.

Respiratory alkalosis = ↑ blood pH due to ↑ respiratory rate (hyperventilation) – too much CO2 exhaled by lungs.

Metabolic acidosis = excess metabolic production of acids (i.e. ketosis) OR loss of bases (i.e. bicarbonate) from chronic diarrhea or kidney problems (excrete too much HCO3-)

Metabolic alkalosis = too much bicarbonate (not enough excreted by kidneys) OR loss of metabolic acids such as with chronic vomiting (lose HCL).

Review

• Acid / Base imbalance
  – Metabolic Acidosis & Alkalosis versus
  – Respiratory Acidosis & alkalosis