Lung Plura

Each lung, like the heart, are surrounded by a membraneous sac called the pleura

- Parietal pleura is attached to the thorax wall and the diaphragm
- Visceral pleura is attached to the lungs itself.
- Inbetween the two pleura is the intrapleural cavity filled with intrapleural fluid.

Ventilation

Ventilation is the exchange of air between the surrounding air and the lungs (and hence, alveoli). This requires an flow of air from one area (outside the lungs) to another area (inside the lungs).

We have seen the definition of flow before with respect to blood flow and the physical principles are the same.

Flow = ΔP/R where

\[ ΔP = P_{atm} - P_{alv} \]

This difference is thus the difference between atmospheric pressure and alveolar (lung) pressure.

Just like wind is created by a flow of air from a high pressure system to a low pressure system, so does air flow into our lungs according to the pressure gradient.

Since we cannot change atmospheric pressure, the main determinant in ΔP is \( P_{alv} \).

Breathing is accomplished by changing the pressure in our lungs.

This change in \( P_{alv} \) is directly related to Boyle's Law

\[ \text{Pressure} \times \text{Volume} = \text{Constant} \ (\text{at constant Temp.}) \]

In other words, by changing the Volume of the lungs, we will end up changing the pressure in the lungs.

However, there are no muscles connected to the lungs to pull on them and increase their size. So how do we increase the size of the lungs?
The volume of the lungs will increase and expand depending on

- Transpulmonary pressure
- Compliance of the lungs (how easy they can be stretched or expanded)

**Intrapulmonary pressure** \(= P_{alv} \)

- pressure within the alveoli of the lungs
- rises and falls with breathing pattern but always equilibrates with atmospheric pressure at the end of an inhalation and end of exhalation

**Intrapleural pressure** \(= P_{ip} \)

- is the pressure within the pleural cavity
- always \(-4\) mm Hg with respect to atmospheric pressure (and with intrapulmonary pressure between breaths)
- the surface tension created by the thin layer of pleural fluid creates this negative pressure
- it seals visceral pleura against parietal pleura like two wet pieces of glass are held together
- if that seal is broken, lungs will collapse due to elasticity of lungs and alveoli
- two factors may collapse lungs (*Atelactasis*)
  - too much pleural fluid creates a positive pressure (due to failing lymph drainage in that area)
  - a breach of the intrapleural space does the same

**Transpulmonary pressure**

- Is the difference in pressure between Intrapulmonary and Intrapleural pressure
Inspiration or Inhalation

- Is initiated by the inspiratory muscles; diaphragm and external intercostal muscles (EIM)
- Contraction of diaphragm causes it to move downwards; increases the length-wise dimension of the thoracic cavity
- Contraction of EIM lifts rib cage and pulls sternum forward; broadens lateral dimensions of thorax

Both actions stretches the lungs and the **intrapulmonary volume** increases.
- Intrapleural pressure and Intrapulmonary pressure drops, resulting in a transpulmonary pressure going from -4 to -6 mm Hg.
- This pressure difference is enough to keep expanding the lungs and maintaining a $P_{alv}$ of -1 mm Hg below $P_{atm}$
- This tiny pressure difference between $P_{atm}$ and $P_{alv}$ is enough to move air into the lungs as air moves down its pressure gradient (outside ---> inside)
- Of the two muscles, diaphragm action is the most important

Expiration

- Is mostly a Passive process that depends on the elasticity of the lungs
- As muscles relax, lungs resume their original length, volume decreases, intrapulmonary pressure rises to about +1 mm Hg above atmospheric which forces air out
- Increased exhalation can be accomplished by internal intercostal muscles and abdominal muscles

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**Inhalation**

- Diaphragm flattens
- External intercostal muscles contract
- Volume of thoracic cavity increases
- Lungs expand
- Air flows down pressure gradient into lungs

**Normal (Passive) Exhalation**

- Muscles of inhalation relax
- Thoracic cavity recoils
- Lung volume decreases
- Air flows down pressure gradient and out of lungs
Factors Affecting Pulmonary Ventilation

1. Airway resistance

- amount of air that moves is directly proportional to the pressure difference \( (P_{\text{atm}} - P_{\text{alv}}) \)
- gas flow however will decrease inversely with resistance

\[
\text{Flow} = \frac{P_{\text{atm}} - P_{\text{alv}}}{\text{Resistance}}
\]

- resistance is determined by the diameter of conducting zone tubing
- as in CVS, greatest resistance is encountered in medium sized bronchi and bronchioles
- Parasympathetic stimulation will constrict smooth muscles in all bronchiolar walls
  (irritants, histamine have the same effect); this can shut down the complete passage ways
- Symp. stimulation causes relaxation of smooth muscles and dilates (under influence of
  norepinephrine, epinephrine)
- Other factors such as tumors, increased mucus may cause resistance to increase

2. Alveolar surface tension

- alveolar surface are coated with watery film
- if it was only water, high surface tension of water would cause alveoli to collapse
- alveolar film contains surfactant, complex of phospholipids, lipids and proteins, that reduces
  surface tension just like a detergent does
- premature babies tend to have insufficient produced surfactant to keep their lungs open
- also, chlorine, smoke, tends to break down surfactant

Read about LaPlace equation from your PPT slides.

3. Lung Compliance

- The ease with which lungs can be stretched is called compliance
- So the more a lung expand in volume for a given change in transpulmonary pressure, the greater the compliance
- Compliance is determined by elasticity of lung tissue, surrounding thoracic cage and surface tension of alveoli
- Compliance is diminished by any factor that reduces
  - lung elasticity
  - surfactant production
  - flexibility of thoracic cage or its ability to expand
  - the smaller respiratory passages
- The higher the compliance, the more efficient the ventilation

Diseases that are the result of reduced lung compliance are called restrictive Lung diseases
**Respiratory Volumes**

Definitions of the Respiratory Volumes
- **Tidal Volume (TV)**
  - amount of air that moves in and out of the lungs per breath (~500 ml) during quiet normal respiration
- **Inspiratory Reserve Volume (IRS)**
  - amount of air that can be inspired forcibly beyond the TV (~2100 to 3200 ml)
- **Expiratory Reserve Volume (ERS)**
  - amount of air that can forcibly expelled beyond normal TV (~1000 to 1200 ml)
- **Residual Volume (RV)**
  - amount of air that remains in the lungs after strenuous expiration (~1200 ml)

**Respiratory Capacities**
- **Inspiratory Capacity (IC):** IC = TV + IRS (~3600 ml max)
- **Functional Residual Capacity (FRC):** FRC = ERS + RV (~2400 ml max)
- **Vital Capacity (VC):** VC = IRS + TV + ERS (~4800 ml max) (is total exchangeable air possible)
- **Total Lung Capacity (TLC):** TLC = VC + RV (sum of all lung volumes = 6000 ml)

FEV1 = the amount air expired within 1 sec after completely filling the lungs to capacity.
FEV1 is usually around 80% of Vital capacity. People with **obstructive lung diseases** (air has problem going through the conducting zone) will show reduced FEV1 and a reduced ERS.

People with restrictive lung disease will show reduced IRV and IC.
Emphysema is a problem where the lungs can easily expand but will not recoil due to loss of elasticity. Which of the volumes discussed above will show dramatic changes?
Concept of Dead Space

- Some of the inspired air never contributes to gas exchange
- It's the air located in the conducting zones and make up the anatomical dead space and is close to 150 ml
- So if TV = 500 ml, then only 350 ml (500 – 150) is involved in alveolar gas exchange

The consequences of this anatomical dead space is that during an average breath, only (500-150) ml of air reaches our alveolar areas (the actual respiratory zones). This 350 ml mixes with the 2400 ml of air that during normal respiration stays behind (1200 ml ERV + 1200 ml RV).

So, during each tidal volume breath, only 12 % of the air in the lungs is replaced. (350 of (2400+350) = 12 %)

Although this seems at first illogical, it prevents large fluctuations in O\textsubscript{2} and CO\textsubscript{2} content; it buffers the lung contents. This is reflected in the rather constant composition of the air we breathe out (see table on atmospheric and alveolar gas content below).

Ventilation Rates

\textit{Minute ventilation rate}

- MVR = the total air that flows in and out of the lungs in one minute.
  - MVR = (air flow per respiration cycle) \times (breathing cycles per minute)
  - MVR = (Tidal Volume) \times (cycles per minute)

During quiet respiration MVR = 500 ml times 12 breathing cycles in a minute = 6000 ml

\textit{Alveolar Ventilation Rate}

- AVR = rate of new air that reaches the alveoli
  - AVR = (Tidal volume – dead space) \times (breathing rate)

What occurs now when we increase metabolic rate (thus oxygen demand)?
- Levels of oxygen in lungs drop, those of CO\textsubscript{2} increase
- That reduces the efficiency of oxygen diffusion and carbon dioxide release
- Thus we end up with inefficient gas exchange

The solution?

Increase ventilation rate and this can be done by
- increasing Tidal volume
- Increasing respiratory cycles

Well trained athletes are known to increase tidal volume up to 3000 ml (by dipping into the IRV) and respiratory breathing cycles up to 30 and more, resulting in a ventilation rate well above 100 liters per minute.
An interesting aspect is if we take the ratio of AVR over MVR:

\[
\frac{AVR}{MVR} = \frac{(\text{Tidal volume} - \text{Dead space})}{(\text{Tidal volume})}
\]

This ratio is thus the fraction of the tidal volume that reaches the alveoli:

- Dead space is a pretty constant factor from person to person
- Increasing tidal volume increases the fraction of useful air
- Shallow faster breathing lowers the amount of air reaching the alveoli since dead space volume becomes a greater % of TV

<table>
<thead>
<tr>
<th>Activity</th>
<th>Breath. rate</th>
<th>Tidal Volume</th>
<th>Dead Space</th>
<th>MVR</th>
<th>AVR</th>
<th>ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>12/min</td>
<td>500 ml</td>
<td>150 ml</td>
<td>6000</td>
<td>4200</td>
<td>0.7</td>
</tr>
<tr>
<td>HypoVentil.</td>
<td>6/min</td>
<td>1000 ml</td>
<td>150 ml</td>
<td>6000</td>
<td>5100</td>
<td>0.85</td>
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<td>HyperVentil.</td>
<td>24/min</td>
<td>250 ml</td>
<td>150 ml</td>
<td>6000</td>
<td>2400</td>
<td>0.4</td>
</tr>
</tbody>
</table>

What are some of the consequences of this anatomical/physiological arrangement in the respiratory system?

- Increasing dead space lowers the amount of fresh air entering the respiratory areas of the alveoli (example: using snorkels and tubing to inhale)
- The disadvantage of dead space can be overcome by increasing the tidal volume (a necessary event in long-necked animals)
- Adjusting tidal volume is more efficient than adjusting respiratory rate

Increased activity usually sees an adjustment in those two parameters to provide the best possible efficiency in air intake.