RESPIRATORY VOLUMES AND RESPIRATORY DYNAMICS[1]

Summary: This section takes the last set of notes in active breathing. We deal in this case with changing pressures and flows instead of static situations as in the last packet. We start with a consideration of elastic and muscular processes in breathing and then move to an examination of the causes airway resistance and its effects on ventilation.

I. THE INTERACTION OF THE MECHANICAL (COMPLIANCE SYSTEMS) AND HOW THEY RELATE TO BREATHING

A. In the last packet, we learned that two separate compliance systems, the lung and the thorax, together determine the total compliance of the respiratory system. We also learned how to determine the compliance of all of these systems.

B. We will add muscles to what we have learned about elastic phenomena in breathing to gain an understanding of the actual dynamic mechanics of breathing.

C. A Brief Review of Ventilation:

1. **Quiet Breathing** (as would accompany rest or very light activity, such as slow walking):

a. **Inspiration**: air is added to the FRC already in the lungs. In quiet breathing the **main active component** is the downward movement of the **diaphragm** with a minor (if any) contribution from the chest wall muscles. The movement of these muscles **creates a subatmospheric (negative) pressure in the alveoli** and draws air inward.

1. **The lung and thorax elastic elements are moved to different positions**.

a) The lung in particular is elastically loaded to a greater extent than at rest. In the resting lung, there is already some stretch on the lung (due to the outward pull of the chest wall). However, as the lungs are pulled downward by the diaphragm and perhaps slightly outward by the chest wall, the elastic load increases and therefore, during inspiration, P_L increases.

(b) There is also an opposite movement of the thorax. The normal resting point for the chest wall is pulled (loaded) considerably inward from its equilibrium (unloaded point) and the diaphragm is slightly pulled upwards from its unloaded shape. Thus, the inspiration is actually aided somewhat by the elastic energy stored in the chest wall (P_{th}).

(c) an additional slight effect in quiet breathing is that the gut is slightly compressed and loaded elastically.

b. **Expiration**: the **diaphragm relaxes**, that is, it develops no more tension. Any tension from the chest wall musculature also is released.

1. Since **at the start of expiration the lungs are loaded much more than at rest, they rebound elastically to a smaller size** and thereby pull the diaphragm back to its normal position. This may be helped somewhat

2. In addition, **they also pull the chest wall inward** although in quiet breathing this is a minor effect. But the result is a slight increase in the elastic loading of the chest wall.

3. The diaphragm is also pulled slightly upward, loading it somewhat and contributing to an increased negative P_{th}.

Movement stops when the P_l and P_{th} are equal and opposite (If all muscles are relaxed, at the FRC -- see the last set of notes).

2. **Heavy breathing** (Not what those of you with erotic minds think!):

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a. **Inspiration**: **Same as quiet breathing except** that the chest wall musculature gets heavily involved. The **external intercostals contract and thereby elevate the ribs**, increasing the chest volume.

1. In very heavy breathing, the intercostals pull the elastic elements of the chest wall outward so far that these elements are loaded in a manner that favors their inward recoil.

2. In other words, they are pulled outward beyond their unloaded equilibrium point. Likewise, the bigger the breath, the even more the lungs are loaded elastically.

b. **Expiration** is the **same as for quiet breathing except** that the **musculature of the chest wall and the abdominal wall are actively involved.** In **addition, the elastic elements of both the lung and chest wall are involved in the expiration.**

1. **During a forced expiration, the internal intercostals contract, forcing the chest inward further than normal (storing elastic energy that will help ventilate the chest). It is not uncommon for the muscles to force the chest wall inward beyond the normal point where it would be found at the FRC**.

! Please note that the external and internal intercostals work best over different ranges of lung volumes -- externals are best at high volumes, internals are best at lower volumes. However, that does not prevent their use during high velocity forced breathing. We will see how both are affected by volume when we consider the effects of volume on air velocity at the end of this set of notes.

2. Also, **contractions of the abdominal wall help force the diaphragm back up into the thorax further than normal.**

! The contractions of both of these muscle groups also increase the rate of movement of the system over what it would be if elastic recoil were the only mechanism for expiration.

3. The elastic elements get involved just as before. However, be aware of the fact that the chest wall may, in very extreme breathing, be moved inward much more than normal and so may become more elastically loaded than normal. The lungs end up smaller and therefore less loaded than normal.

? Draw a graph that shows normal tidal volumes in relation to the FRC. Show how these tidal volumes change at the expense of the IRV and ERV as the workload progressively increases; i.e., plot steady-state tidal volume vs. increasing workload.
? What antagonizes the diaphragm during quite breathing? What are the passive and active antagonists of the inspiratory muscles during heavy breathing?

II. RESPIRATORY DYNAMICS: refers to situations where there is airflow.

A. The movement of air in the lungs is a bit more complicated than the study of static volumes due to the presence of resistance. Therefore, let's consider resistance for a while:

1. For any fluid in motion:

2. $\dot{V} \propto \Delta P$

where $\dot{V}$ is the flow rate (vol./time) and ΔP is the pressure difference between two points where flow is occurring.

! NOTE: this is not strictly true -- **fluids actually flow down total energy gradients**, not pressure gradients. However, in most cases pressure will suffice and we will use this approximation in this class.

Changing this relationship to an equation, we have:

3. $\dot{V} = G * \Delta P$

where *G* is a constant called the conductance. It is related inversely to another constant, resistance, *R*:

4. $R = \frac{1}{G}$

and by substitution:

5a,b. $\dot{V} = \frac{\Delta P}{R}$ -- or-- $r = \frac{\Delta P}{\dot{V}}$

Note that this equation is essentially identical to Ohm's Law.

2. We also know from **Poiseuilles' equation** that:

6. $\dot{V} = \frac{(\Delta P * \Pi * r^4)}{(8 * \eta * L)}$

7. $\dot{V} = k * \frac{(\Delta P * r^4)}{L}$

where $\dot{V}$ is flow (vol./time), ΔP is the pressure difference, r is the radius of the airway through which the air passes, η is the viscosity of the fluid (air), L is the length of the airway, and k in eq. 7 is a constant for air at sea level and is equal to $\Pi/(8 * \eta)$.

? Under what physical conditions would η vary significantly for a human? (hint -- it would be an unusual activity.)

3. We can now substitute eq. 7 into eq. 5b:

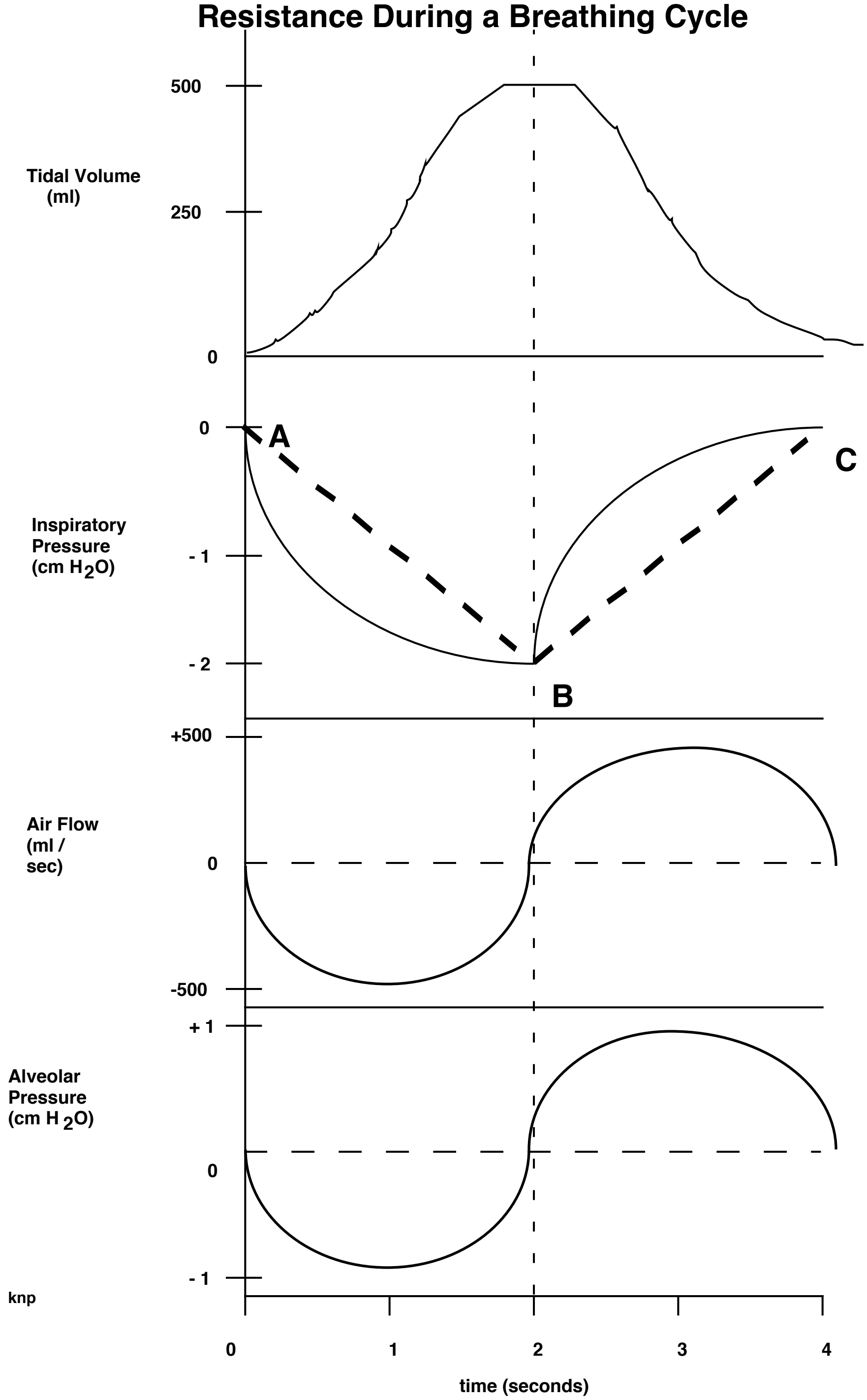
8. $r = \frac{\Delta P}{k * \Delta P * r^{4*} L^{-1}} = \frac{length}{k * r^4}$

Thus the longer the tube the more resistance, the smaller the radius, the more resistance.

B. What is the significance of resistance in breathing?

The Best Way to Understand Resistance and Dynamic Aspects of Breathing is to look at a diagram that shows pressures, volumes and flows. This diagram is an excellent summary of breathing:

The Relationships Between Flow, Pressure, and Resistance During a Breathing Cycle



At the top is an idealized tidal inspiration and expiration -- it more or less approximates a sine wave (within n ability to draw). Real breathing is not exactly like this. The bottom two panels depict the velocity of air flow (3) and P_{alv}. Notice that Palv becomes most negative or positive at the time of greatest flow rate (check the flow equation to verify that this should be true). Panel #2 is the one that actually shows the <u>effect of resistance</u>.

<u>The Dotted straight lines</u> (at 4 angles) represent the pressures that would be found if a series of <u>static</u> measurements were made by taking a series of different tidal inspirations and then relaxing with a closed glottis (<u>not</u> -- in this case the air flow diagram (pane #3 would be a horizontal line of no flow) This particular curve would be for inhaling 500 ml. The sign is used to indicate direction of air movement, a negative sign measn air has entered the lung; the more negative the more air. ((This convention is used since negative alveolar pressures are needed to inspir air). Thus, as lung volume increased, th graph shows that the inspiratory pressure becomes more negative.

<u>The Curved Lines</u> represent th actual inspiratory pressures that must b produced in order to get a flow rate like that depicted in panels #1 and #3. The <u>additional pressure</u> (*think about this pressure in terms of its absolute value relative to the static pressure line*) <u>is th additional pressure needed to overcom the effects of resistance to flow</u>.

? How would you calculate the work needed over 1/2 and a full cycle? How would you calculate the work needed to expand the lungs to some volume if resistance was ignored? How about if resistance was included? How would you calculate the work of overcoming resistance? How about power for all of these? Be sure you can do these calculations. Check your units to be sure that your calculation is dimensionally correct. (hint -- this should be easy -- pressure times volume gives what type of unit?)

! Be certain that you can explain the relationships between flow, P_{alv}, and volume. After reviewing the material below, you should thoroughly understand breathing.

1. If a subject inhales air and then stops, in a series of steps and we measure the alveolar pressure after relaxation against a closed glottis, we see that this pressure becomes larger as the inspired volume increases. We normally indicate this as a negative pressure since negative pressures are required to draw air into the lungs **(Thus, the inspiratory pressure is actually the $P_{surface}$ - P_{rs} since the alveolar pressure measured after relaxation with a closed air passage is the same as the total recoil pressure)**The curve for such a series of static measures is linear and was shown earlier.

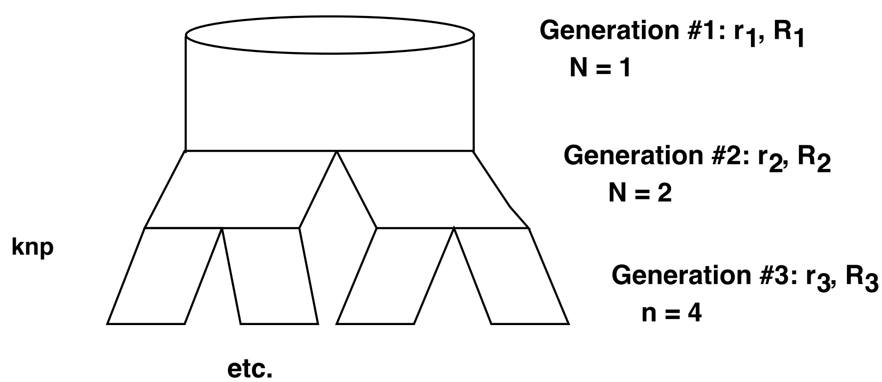
2. If instead we continuously measure volume, flow, and alveolar pressure (in other words, if we make a dynamic measurement), then we get a curve different from the static curve.

a. We know that flow increases and then decreases during inspiration.

b. If we assume that R is constant (it is determined mainly by airway diameter and this is not a big enough breath to greatly expand the airways), then inspiratory pressure at any given volume must be more negative than when measured under static conditions since an "extra" increment in pressure is required to draw a certain volume of air against the resistance that exists when there is flow. The degree of difference between static and dynamic inspiratory pressures is a measure of resistance.

3. Next, as anyone with asthma knows, airway diameter is very important in determining resistance to airflow. Let's look at a number of important phenomena:

a. Does resistance to airflow change as the bronchi become smaller and smaller? The cross-sectional area per bronchus decreases with each increasing **GENERATION** of the **RESPIRATORY TREE:**



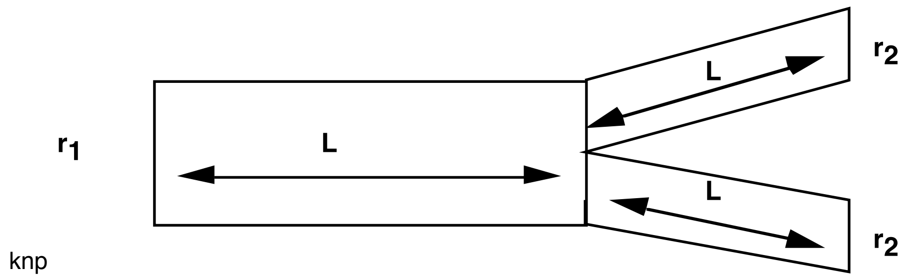
Thus, we would expect the total airway resistance to increase as we move to smaller and smaller bronchi. However, as is shown above, we can see that generally each bronchus divides into at least two others of smaller size. Since total resistance of such parallel elements is determined by:

9. $$\frac{1}{R_t} = \frac{1}{R_1} + \frac{1}{R_2} + \ldots\ldots + \frac{1}{R_n}$$

then we can calculate the maximum that the airways can decrease in diameter and still maintain constant resistance each time that they divide. To do this, remember that:

10. $R = \frac{k_1}{r^4}$

Let's assume a single airway always divides into two airways of equal diameter to each other but of smaller diameter than the single airway they came from. Assume that the length of all of these is the same and the viscosity of air is constant. Then:
(diagram of assumptions)



And let r_1 be the radius of the larger tube and r_2 of the second-generation tubes.

? What is the largest the radius can be in the secondary tubes and have the resistance remain constant?:

First, we set the total resistance of each generation of airways to an equal value:

11. $R_1 = R_2$

where R_1 is the total resistance of airway generation 1 and
R_2 is the total resistance of airway generation 2. Now, using eq. #9:

12. $\frac{1}{R_1} = \frac{1}{R_{small}} + \frac{1}{R_{small}}$

-OR--

12b. $\frac{1}{R_1} = \frac{2}{R_{small}}$

and since $R_1 = R_2$:

13. $R_2 = \frac{R_{small}}{2}$

since we stated that each of the two airways in the next generation have the same radius and length. Next, since:

14. $R_{small} = \frac{k_1}{(r_2)^4}$ -- then --

15. $\frac{k_1}{(r_1)^4} = k_1 \frac{(\frac{1}{r_2})^4}{2}$

16. $\frac{1}{(r_1)^4} = \frac{1}{2} * \frac{1}{(r_2)^4}$

17. $r_2^4 = 0.5 * r_1^4$

Now, if we take the 4th root of both sides of equation #17:

18. $r_2 = \sqrt[4]{0.5 * r_1}$

and finally:

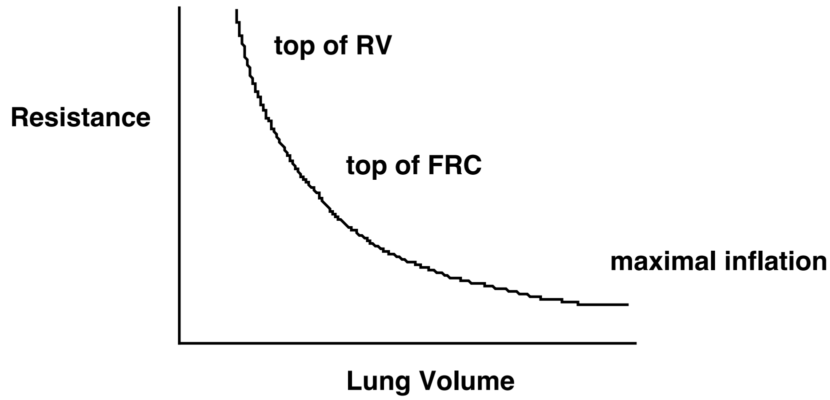
19. $r_2 = 0.84 * r_1$

If r_2 is smaller than this limit (0.84 * r_1), the resistance will increase in the next generation of the tree. In most generations of bronchi, the airway resistance actually decreases slightly, although there are some cases where resistance increases.

b. What are the factors that affect airway diameter?:

stimulus	contraction	relaxation
nervous and endocrine	cholinergic acetylcholine histamine prostaglandin F-2a	adrenergic NE and E prostaglandin E
chemical	smoke, dust, SO_2	

c. The lung volume itself affects the airway resistance:



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EXPLANATION: The greater the lung volume the more distended the conducting system is.

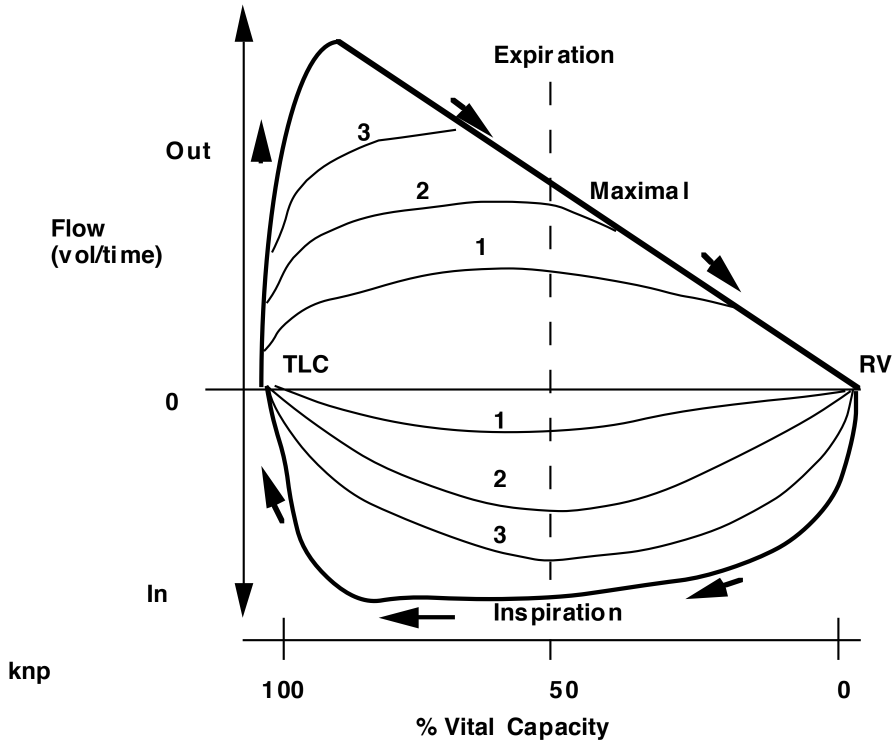
i) At large volumes the elastic recoil of the lungs are high and as a result the airways are pulled out and their diameter is increased.

ii) The opposite is true at small lung volumes.

! Note: Destruction of alveolar walls results, as in emphysema, in a larger compliance of the alveoli and less recoil pressure. As a result of the lower recoil pressure, the airway resistance is secondarily affected and it increases.

C. In light of the observations above that show that airway diameter is a function of volume of the lung, we may ask how FLOW varies with lung volume? On the next page is the graph of maximum airflow and several smaller volumes:

Flow Patterns During Expirations and Inspirations of the Same Volume but with Different Rates of Gas Movement



The outer curve (darkest line) represents the most forceful expiration and inspiration. The other graphs represent the flow volume relationships with progressively less forceful expiration or inspiration (3-1).
Note the difference in the shape and values of the flow volume curves for inspiration and expiration -- they are not mirror images of each other.

Explanation: During a maximal expiratory effort, **flow is initially very high** due to:

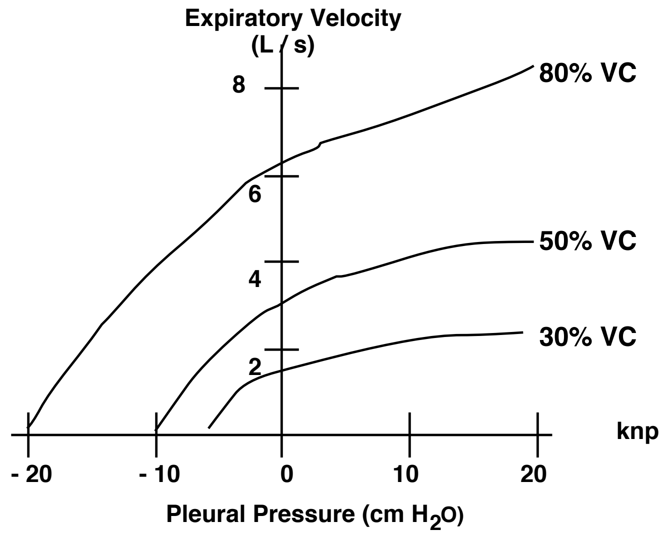
i) large volume means that the airways are fully opened and resistance is low (see previous graph).

ii) Elastic recoil for both the lungs and thorax is large and directed inwards (both are well beyond their resting volumes).

iii) Expiratory muscles (ex. intercostals and abdominals) are close to their L_O.

As expiration continues, resistance increases, recoil pressures become less favorable (less elastic energy available, especially from the chest wall which now is stretched inward and resists this movement -- energy must be put in the chest wall) and the muscles are shortening below L_O. As a result flow decreases.

? See if you can determine the reasons for the different shape of the maximal inspiration curve and the sub-maximal inspiration and expiration curves.



Each curve shows the flow rate at different pleural pressures when the lung is inflated to different volumes. This graph keeps a number of variables important to the flow volume graph given previously constant. The high flow rates associated with the large volumes are due to the low airway resistance at these values.

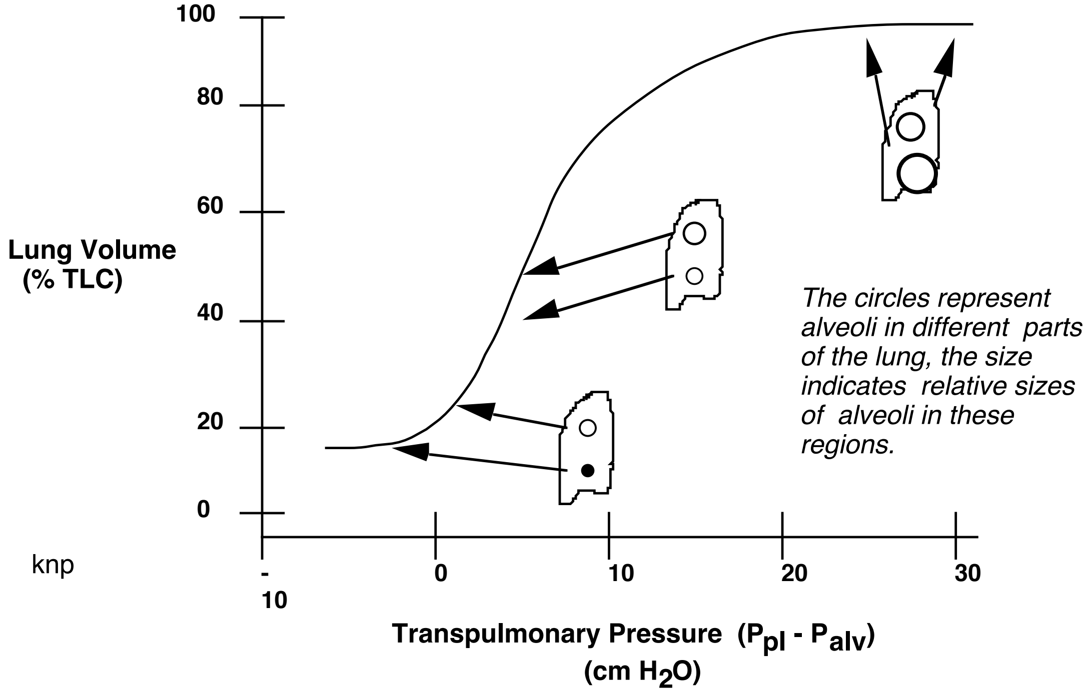
! Think about the graph above --the most important factor is that it shows that for a given range of expiratory pressures, the volume (%VC) and therefore resistance is crucial in determining the size of the flow rate. This clearly relates to eqs. #5 and 6.

D. Finally, a bit about of the **distribution of gases in the lungs**:

1. **At typical lung volumes such as the respiratory rest point (top of the FRC), the pleural pressure is more sub-atmospheric at the top of the lung than lower parts** since the weight of the lung pulls on the top. Thus, transpulmonary pressure (Palv - Ppl; i.e. lung recoil pressure) is greater at the top of the lung than at the bottom. **The result is that at small lung volumes the alveoli are larger at the top**. The same is true of the airway diameter. Therefore, the alveoli at the apex (top) of the lung are more ventilated than those that the base (bottom) except in cases where the lung is totally expanded and a high P_{pl} is felt everywhere on the lung:

2. On the other hand, **at large lung volumes the alveoli are largest at the bottom**. This has to do with the fact that the **chest wall moves (expands) a greater amount near the bottom** (think of how the ribs are hinged on the top vs. bottom and also the movement of the diaphragm).

Distribution of Air at Different Lung Volumes in Different Regions of the Lung



Notice how the relative ventilation of alveoli in different regions of the lung vary according to the degree of inflation of the entire lung. Further note that this is related to regional differences in transpulmonary pressure in different parts of the lung -- at low volumes the top of the lung is relatively stretched by the weight of the lung compared to the lower portions and as a result the pleural pressure is different in the different regions resulting in a different transpulmonary pressure (alveolar pressure is the same everywhere).