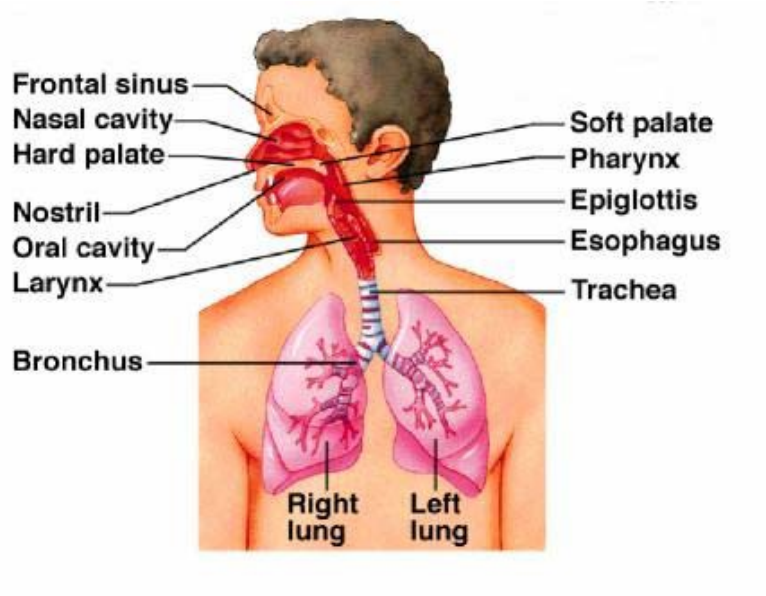
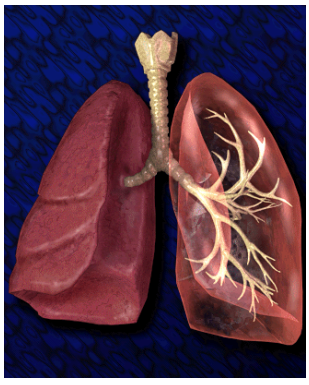
Anatomy and Physiology of Respiratory System

Overview

The primary function of the respiratory system is supplying oxygen to the blood and expelling waste gases, of which carbon dioxide is the main constituent, from the body. This is achieved through breathing: we inhale oxygen and exhale carbon dioxide. Respiration is achieved via inhalation through the mouth or nose as a result of the relaxation and contraction of the diaphragm. The air, in essence oxygen, then passes through the larynx and trachea to enter the chest cavity. The larynx, or voice box, is located at the head of the trachea, or windpipe. In the chest cavity, the trachea branches off into two smaller tubes called the bronchi which enter the hilus of the left and right lungs. The bronchi are then further subdivided into bronchioles. These, in turn, branch off to the alveolar ducts which lead to grape-like clusters called alveoli found in the alveolar sacs. The anatomy of the respiratory system is shown in Figure below. The walls of alveoli are extremely thin (less than 2 μm) but there are about 300 millions of alveoli (each with a diameter about 0.25 mm). If one flattens the alveoli, the resulted surface can cover about 100 m^2.

The lungs are the two sponge-like organs which expand with diaphragmatic contraction to admit air and house the alveoli where oxygen and carbon dioxide diffusion regenerates blood cells. The lungs are divided into right and left halves, which have three and two lobes, respectively. Each half is anchored by the mediastinum and rests on the diaphragm below. The medial surface of each half features an aperture, called a hilus, through which the bronchus, nerves and blood vessels pass.





Deoxygenated blood is pumped to the lungs from the heart through the pulmonary artery. This artery branches into both lungs, subdividing into arterioles and metarterioles deep within the lung tissue. These metarterioles lead to networks of smaller vessels, called capillaries, which pass through the alveolar surface. The blood diffuses waste carbon dioxide through the membranous walls of the alveoli and takes up oxygen from the air within. The re-oxygenised blood is then sent through metavenules and venules, which are tributaries to the pulmonary vein. This vein takes the re-oxygenised blood back to the heart to be pumped throughout the body for nourishment of its cells.

Without going through the pulmonary physiology in detail, it is necessary to introduce a few pulmonary parameters that will be referred to when we discuss the lung sound analysis.

Ventilation Parameters

Lung Volumes

a. **Tidal Volume (TV):** Volume of gas exchanged each breath – can change as ventilation pattern changes and is about 0.5 liter.
b. **Inspiratory Reserve Volume (IRV):** maximum volume that can be inspired over and beyond the normal tidal volume and is about 3 liters in young male adult.
c. **Expiratory Reserve Volume (ERV):** maximum volume that can still be expired by forceful expiration after the end of a normal tidal expiration and is about 1.1 liter in young male adult.
d. **Residual Volume (RV)** is the volume remaining in the lungs and airways following a maximum expiratory effort and is about 1.2 liter in young male adult. Note that lungs cannot empty completely because of stiffness when compressed, and also airway collapse and gas trapping at low lung volumes.

Capacities: combined volumes

a. **Vital Capacity (VC):** maximum volume of gas that can be exchanged in a single breath. VC = TV + IRV + ERV
b. **Total Lung Capacity (TLC):** maximum volume of gas that the lungs (and airways) can contain. TLC = VC + RV
c. **Functional Residual Capacity (FRC):** volume of gas remaining in the lungs (and airways) at the end of expiratory position. FRC = RV + ERV. We normally breathe above FRC volume.
d. **Inspiratory Capacity (IC):** maximum volume of gas that can be inspired from the of expiratory position IC = TV + IRV

Minute Ventilation is the total flow of air volume in/out at the airway opening (mouth). Hence, Minute Ventilation = Tidal Volume × Respiratory Rate.

Dead Space is the volume of conducting airways that no gas diffusion occurs. Fresh air entering dead space does not reach alveoli, and hence doesn't mix with alveolar air. It is about 150 ml = 30% of resting tidal volume.

Fig. 4 shows a rough breakdown of these and other lung volumes. The vital capacity (VC) and its components, as shown in Fig. 4, may be measured using a means of pulmonary function testing known as spirometry, which involves inhalation of as much air as possible, i.e., to TLC, and maximally forcing the air out into a mouthpiece and flow sensor. Residual volume (RV) may be measured by whole-body plethysmography,

whereby a subject undergoing this test will be seated in an airtight chamber, and asked to breathe in a sequence of highly coordinated maneuvers, allowing for the measure of the difference between the known volume of the chamber and volumes breathed by the subject.

Lung Mechanic

The simplest and most common variables used to assess normal and altered mechanics of respiratory system are *airway resistance* and *lung compliance.* Both of these parameters change in various disease states; hence, they are important parameters to asses the lung and respiratory system.

Airway resistance is analogous to blood flow in the cardiovascular system and also analogous to resistance in an electrical circuit while pressure and air flow are analogous to voltage and current in that circuit, respectively. Hence, one can conclude that the airway resistance can be measured as the change of pressure (voltage) to the flow (current). This measurement and relationship is true regardless of the type of flow. Recall that there are two types of air flow: laminar and turbulent.

When flow is low in velocity and passes through narrow tubes, it tends to be orderly and move in one direction; this is called laminar flow. For laminar flow, resistance is quite low and cab be calculated by the Poiseuille's law, which is then directly proportional to the length of tube and inversely proportional to the forth power of radius of the tube. Hence, the radius has a huge effect on the resistance when the flow is laminar; if the diameter is doubled the resistance will drop by a factor of sixteen.

On the other hand, when flow is at high velocity, especially through an airway with irregular walls, the movement of flow is disorganized, perhaps even chaotic and makes eddies. In this case the pressure-flow relationship is not linear. Hence, there is no straight forward equation to compute airway resistance without knowing the pressure and flow velocity, and it can only be measured as the ratio of the change of pressure over the flow velocity. Airway resistance during turbulent flow is relatively much larger compared to laminar flow; a much greater pressure difference is required to produce the same flow rate as that of laminar flow.

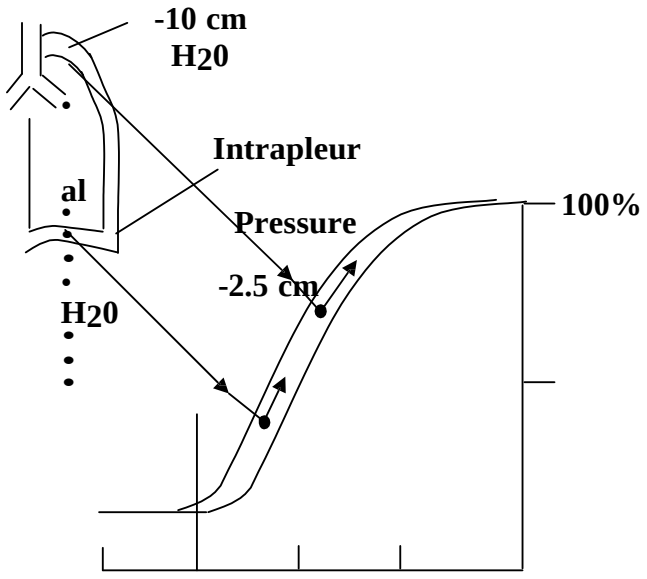
Regardless of the type of flow, the airway resistance increases when the radius of the airway decreases. Therefore, in first glance at the respiratory system, it is expected that the larger airways, i.e. trachea, should have less resistance compared to that of smaller airways such as alveoli. However, it is the opposite. Recall that the bronchi tree has many branches in parallel with each other (i.e., parallel resistors), hence the net effective resistance of the alveoli is much less than that of the larger airways, i.e., trachea. In fact, approximately 90% of the total airway resistance belongs to the airways larger than 2 mm.

Airway resistance is a very useful parameter as it can quantify the degree of obstruction to airflow in the airways. However, since the smallest airways get affected first by the development of obstructive lung disease and also that the majority of airway resistance appears in larger airways, therefore obstructive lung

disease may exist without symptoms of obstructive airways at least at early stages of the disease.

Compliance is a measure of lung stiffness or elasticity. Because of this inflatable property, the lung has often been compared to a balloon. For example, in fibrosis the lungs become stiff, making a large pressure necessary to maintain a moderate volume. Such lungs would be considered poorly compliant. On the other hand, in emphysema, where many alveolar walls are lost, the lungs would be considered highly compliant, i.e., only a small pressure difference inflates the lung.

Compliance is measured as the ratio of the change of volume over the change of pressure. However, volume-pressure relationship is not the same during inflation (inspiration) and deflation (expiration); it forms a hysteresis loop (Figure below). The dependence of a property on past history is called hysteresis. As it can be observed in Figure below the volume at a given pressure during deflation is always larger than that during inflation. Another important observation from the volume-pressure hysteresis curve is that the compliance changes with volume and actually it has a shape like an inverted bell with peaks at FRC; implying the highest compliance of lung is when we breathe at tidal flow (which is above the FRC volume), hence the effort (pressure) is required for tidal breathing. One can correctly expect and experience that at higher volumes than FRC (higher flow rates) the lung becomes stiffer (less compliant) and breathing requires more effort (pressure).



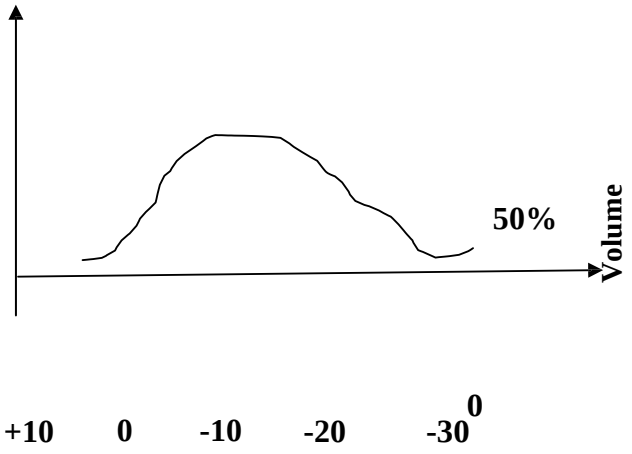


Figure An explanation of the regional differences of ventilation down the lung. Because of the weight of the lung, the intrapleural pressure is less negative at the base than at the apex. As a consequence, the basal lung is relatively compressed in its resting state but expands better on inspiration than the apex.

In diseases such as fibrosis, the compliance is reduced and the lung becomes stiff. On the other hand, in chronic obstructive pulmonary disease, i.e. emphysema, the alveolar walls degenerate; hence, increasing the lung compliance. In emphysema, the airways might be normal but because the surrounding lung tissue is progressively destroyed, the airways lose their normal support. Therefore, during inspiration they do not enlarge and on expiration they tend to collapse. Lung fibrosis on the other hand may have opposite effect. By stiffening the lung tissue, airways in a fibrotic lung may be larger and more stable than normal. However, this does not mean that ventilation is easier in fibrosis. Even though the airway resistance may be smaller, the increased lung stiffness inhibits normal lung expansion making breathing very hard work. Asthmatic people tend to breathe at high lung volume in order to counteract the increase in smooth muscle tension, which is the primary defect in an asthmatic attack.

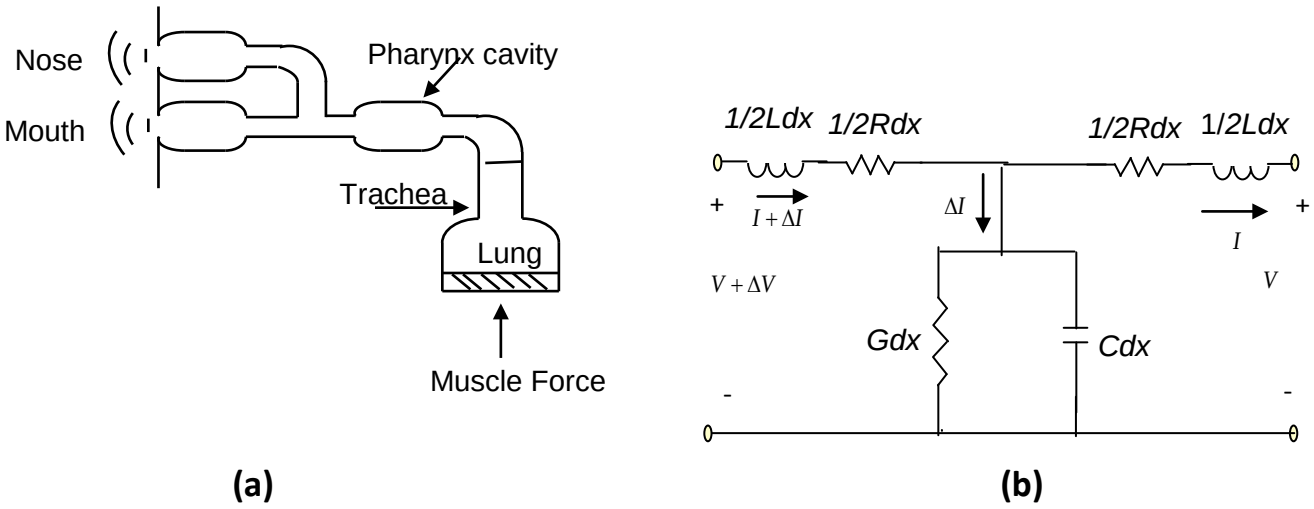


Figure 1. (a) Schematic diagram of functional components of the vocal tract; (b) Electrical equivalent for a one-dimensional wave flowing through a lossy cylindrical pipe.

The length of the vocal tract (about 17 cm in adult men) is fully comparable to the wavelength of sound in air at audible frequencies. It is therefore not possible to obtain a precise analysis of the tract operation from a lumped-constant approximation of the major acoustic components. Wave motion in the system must be considered for frequencies above several hundred cps. The vocal and nasal tracts constitute lossy tubes of non-uniform cross- sectional area. Wave motion in such tubes is difficult to describe, even for lossless propagation. Practically, the vocal tract is modeled as a series of uniform, lossy cylindrical pipes. Consider the length *dx* of such pipe of area A. Assume plane wave transmission so that the sound pressure and volume velocity are spatially dependent only upon *x*. Because of its mass, the air in the pipe exhibits an interance, which opposes acceleration. Because of its compressibility the volume of air exhibits compliance. Assuming that the tube is smooth and hard-walled, energy losses can occur at the wall through viscous friction and heat conduction. Viscous losses are proportional to the square of the particle velocity, and heat conduction losses are proportional to the square of the sound pressure. The characteristics of sound propagation in such a tube are easily described by drawing upon electrical lossy transmission line theory. Consider sound pressure analogous to the voltage and volume velocity analogous to the current in an electrical line. Sound pressure and volume velocity for plane wave propagation in the uniform tube satisfy the save wave equation as do voltage and current on a uniform transmission line. A *dx* length of lossy electrical line is illustrated in Figure 1b. Let *x* be the distance measured from the receiving end of the line and then *Zdx* is the series impedance of the *dx* length of the line and *Ydx* is its shunt admittance. The voltage at the end of the end *dx* line is V and is the complex expression of the RMS voltage, whose magnitude and phase vary with distance along the line. The voltage at the other side of the *dx* line is *V+dV*. By writing a KVL, we get:

$$V + dV = \frac{1}{2}(I + dI)Zdx + \frac{1}{2}ZdxI + V \Rightarrow \frac{dV}{dx} = IZ + \frac{1}{2}ZdI$$

As we let *dx* approach zero, *dI* also approaches zero, and the second term on the right vanishes and therefore in the limit

$$\frac{dV}{dx} = IZ \qquad (1)$$

Similarly by writing KCL and neglecting the second order effects

$$\frac{dI}{dx} = VY \qquad (2)$$

By differentiating the Equations 1 and 2, we obtain

$$\frac{d^2V}{dx^2} = ZYV \quad (3) \text{ and } \quad \frac{d^2I}{dx^2} = ZYI \qquad (4)$$

The above two equations must hold together. Assume that the solution of Equation 3 is

$$V = A_1 e^{(\sqrt{YZ})x} + A_2 e^{(-\sqrt{YZ})x} \qquad (5)$$

Taking the second derivative of V with respect to x in above equation yields Equation 3. Therefore, this is indeed a solution for Equation 3. Using this solution and Equation 1, we obtain the solution for I:

$$I = \frac{1}{\sqrt{Z/Y}} A_1 e^{(\sqrt{YZ})x} - \frac{1}{\sqrt{Z/Y}} A_2 e^{(-\sqrt{YZ})x} \qquad (6)$$

The constants A1 and A2 can be evaluated by using the conditions at the receiving end of the line when $x=0$, $V=V_R$ and $I=I_R$. Substituting of these values in Equations 5 and 6 yields

$$V = \frac{V_R + I_R Z_c}{2} e^{\gamma x} + \frac{V_R - I_R Z_c}{2} e^{-\gamma x}$$
$$I = \frac{V_R / Z_c + I_R}{2} e^{\gamma x} + \frac{V_R / Z_c - I_R}{2} e^{-\gamma x}$$

where $\gamma = \sqrt{ZY}$ and is called the propagation constant, and $Z_c = \sqrt{Z/Y}$ which is called characteristic impedance of the line.

Having recalled the relations for the uniform, lossy electrical line, we want to interpret plane wave propagation in a uniform, lossy pipe in analogous terms. If sound pressure, p, is considered analogous to voltage and acoustic volume velocity, U, analogous to current, the lossy, one-dimensional, sinusoidal sound propagation constant is complex and the wave attenuates as it travels. In a smooth hard-walled tube the viscous and heat conduction losses can be represented, in effect by an I^2R loss and an V^2G loss, respectively. The interance of the air mass is analogous to the electrical inductance and the compliance of the air volume is analogous to the electrical capacity.

The Acoustic "*L*"

The mass of air contained in the pipe with length *l*, is ρAl, where ρ is the air density. The differential pressure drop in accelerating this mass by Newton's Law is:

$$F = ma \quad \Rightarrow \quad PA = \rho Al \frac{du}{dt} = \rho l \frac{dU}{dt} \quad \Rightarrow \quad P = \rho \frac{l}{A} \frac{dU}{dt}$$

comparing with $V = L \frac{dI}{dt} \quad \Rightarrow \quad \mathrm{L_a} = \frac{\rho l}{A}$

Note that *u* is particle velocity and $U=Au$ is volume velocity.

The Acoustic "C"

The analogous acoustic capacitance, or compliance arises from the compressibility of the volume of air contained in the *dx* length of tube. Most of the elemental air volume *Adx* experiences compression and expansions that follow the adiabatic gas law:

PV^{η} = contant, where V and P are the total pressure and volume of the gas, and η is the adiabatic constant. Differentiating with respect to time gives :

$$p\eta V^{\eta-1}\frac{dV}{dt}+V^{\eta}\frac{dP}{dt}=0$$

$$\frac{1}{P}\frac{dP}{dt}=-\frac{\eta}{V}\frac{dV}{dt}=\frac{\eta}{V}U$$

$$\Rightarrow U=\frac{V}{P\eta}\frac{dP}{dt}=C\frac{dP}{dt}$$

$$\therefore C=\frac{V}{P\eta}$$

However, in pulmonary mechanics C is measured as $\frac{\Delta V}{\Delta \mathrm{P}}$

The Acoustic "R"

The acoustic R is defined as $R_a=\frac{lS}{A^2}\sqrt{\frac{\omega\rho\mu}{2}}$, where *A* and *S* are the tube area and circumference, respectively. ρ is air density and μ is viscosity coefficient. In practice of pulmonary mechanics however, R is measured as $R=\frac{\Delta P}{\Delta U}$.

The Acoustic "G"

The acoustic G is defined as $G_a=Sl\frac{\eta-1}{\rho c^2}\sqrt{\frac{\lambda\omega}{2c\rho}}$, where *c* is sound velocity, λ is coefficient of heat conduction, η is the adiabatic constant and c_p is the specific heat of air at constant pressure.

Respiratory Sounds

Breath Sounds Recording

Since the invention of stethoscope by the French physician, Laennec, in 1821, auscultation (listening to the sounds at body surface) has been the primary assessment technique for physicians. Despite the high cost of many modern stethoscope including digital stethoscopes, their use is limited to auscultation only as they are not usually tested, calibrated or compared. Furthermore, they do not represent the full frequency spectrum of the sounds as they selectively amplify or attenuate sounds within the spectrum of clinical interest.

Digital data recording, on the other hand, provide a faithful representation of sounds. Respiratory sounds are usually recorded either by electret microphones or sensitive contact accelerometers, amplified, filtered in the bandwidth of 50-2500 Hz and digitized by a sampling rate higher than 5 kHz. Respiratory flow is also commonly measured by face mask or pnuemotachgraph attached to a pressure transducer as shown in lecture slides, and is digitized simultaneously with respiratory sounds.

Breath Sound Characteristics

Lung sounds' amplitude is different between persons and different locations on the chest surface and varies with flow. The peak of lung sound is in frequencies below 100 Hz. The lung sound energy drops off sharply between 100-200 Hz but it can still be detected at or above 800 Hz with sensitive microphones. The left top graph shows a typical airflow signal measured by a mouth-piece pneumotach. The positive values refer to inspiration and negative values refer to expiration airflow. The left bottom graph on Slide #42 shows the sonogram of the lung sound recorded simultaneously with that airflow signal. The sonogram is a representation of the power spectrum for each time segment of the signal. The horizontal axis is the duration of the recording in seconds and the vertical axis is the frequency range. The magnitude of the power spectrum is therefore shown by color, where the pink color represents above 40 dB whereas the dark gray represents less than 4 dB of the power. As you see, the inspiration segments of the lung sound have much higher frequency components than expiration segments. In another word, inspiration sounds are louder than expiration sounds over the chest wall and this observation is fairly consistent between the subjects. The right graph shows the average spectrum of all inspiration segments compared to that of expiration segments. Again, as you see there is about 6-10 dB difference between inspiration and expiration power spectrum over a fairly large frequency range.

On the other hand, tracheal sound is strong and covers a wider frequency range than lung sound. Tracheal sound has a direct relationship with airflow and covers a frequency range up to 1500 Hz at normal flow rate. Similar to the last slide, the left graphs on Slide #43 show a typical airflow signal on the top and the associated sonogram of the tracheal signal on the bottom. As you see, the tracheal sound signal is a much louder signal than lung sound signal. However, the difference in inspiration and expiration power of the tracheal sound signal, varies between the subjects greatly.

In some, there is not much difference, while in some such as the subject in this example the expiration sound is louder than inspiration.

The relationship of flow with power density of tracheal and lung sounds leads to the idea that at least the breath phases, i.e., inspiration/expiration and the onset of breaths can be determined acoustically without the actual flow measurement, which indeed was done a few year ago. The actual flow estimation by acoustical means however, requires much more signal processing techniques and investigations. We will discuss this issue in more details in the following sections.

Like all other biological signals, respiratory sounds also differ between the subjects as their chest size and body mass are different. However, using digital signal processing techniques researchers have sought methods to extract some characteristic features of the respiratory sounds that can be used for diagnostic purpose between healthy individuals and patients with various respiratory diseases.