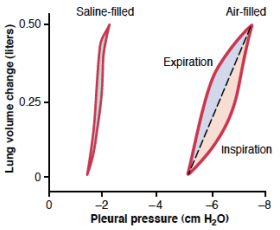
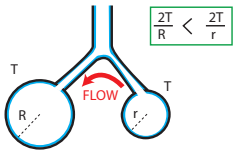
Elastic Recoil of the Lung For many years it was thought that the elastic recoil of the lungs was solely due to the elastin fibres present in the lung parenchyma. This notion was disproven by von Neergaard who showed a lung completely filled with saline had much greater compliance. This is shown in the adjacent diagram. Also note that the pressure is greater during inspiration rather than expiration, this is termed hysteresis and is a feature of all elastic bodies. Von Neergaard correctly concluded that this was due to the extensive surface tension acting throughout the vast air/water interface in the lungs. Thus the elastic recoil of the lungs is dependent on both the elastic fibres of the parenchyma and the surface tension of the alveoli. It is the later of these two which is considered more important.



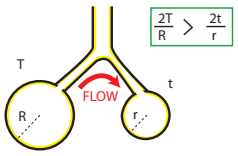
Lung compliance may be reduced by an increase of fibrous tissue in the lung, due to alveolar oedema, if a part of the lung is unventilated for a long period of time, and is somewhat reduced by increased pulmonary venous pressure and the lung becoming engorged with blood. Lung compliance is also reduced at the extremes of inflation. Lung compliance is increased in patients with emphysema and there is conflicting reports on the effects of age on compliance. By definition compliance is $\triangle V/\triangle P$, therefore larger lungs are likely to have a larger $\triangle V$ and subsequent compliance, hence the use of specific compliance to make conclusions about the intrinsic elastic properties of the lung tissue. The other main factor which influences lung compliance, namely surface tension/surfactant.

WITHOUT SURFACTANT (T is constant)



Surface Tension develops at air-water interfaces where the forces of attraction between the water molecules are much greater than those between the water and gas molecules. The result is that the liquid surface area becomes as small as possible as in a soap bubble where the shape of a sphere is assumed and pressure (P) is produced inside the bubble because of the surface tension. The pressure inside the bubble is determined by the radius of the bubble and the surface tension of the liquid. This was first characterised by Laplace. Pressure = 4(Surface tension)/Radius. If the bubble is a sphere and therefore has only one surface the formula is P = 2T/R. There is a very important consequence of this, if we assume that alveoli are spheres then smaller alveoli will have greater pressure and therefore will redistribute their contents to larger bubbles. This is not what is observed and it is believed that this is due to the presence of surfactant.

WITH SURFACTANT (T decreases with decreased radius)



Surfactant is formed from type II alveolar cells. It is mostly phospholipid with the principle component DPPC which equates to roughly 80% content. There are also four surfactant protiens "SP" A to D. These are believed to be important in the stabilisation of the surfactant, its activation and release from alveolar cells. Surfactant forms a mono layer due to its structure with a hydrophilic head adjacent to the cell wall and the hydrophobic tail facing inwards towards the air. Surfactant has a 15-30 hour half life and in addition to its primary role of reducing surface tension also prevents transudation into in the the alveolus (less pressure inside alveolus) and has an immunological role. The exact mechanism of how it reduces surface tension is unknown but the main hypothesis is that as it packs closer together when the alveolus reduces in size the action is accentuated, causing a greater decrease in surface tension with decreases in radius.

Time constant and Fast and Slow Alveolus The concept of the time constant is used to describe the exponential filling and emptying of a lung unit. One time constant is the time taken to achieve 63% of maximal inflation or deflation of the lung unit. After three time constants the lung unit will be 95% full. The time constant of the lung is a product of the resistance and the compliance. The resistance for normal lung tissue is 2 cm.H2O/L pre second and the total compliance is 100ml/cmH2O (assuming lung compliance and chest wall compliance of 200ml/cmH2O each), therefore the time constant for the whole lung is 0.2s. This is across the whole lung. We know however that the resistance and compliance of alveolar units are not always equal throughout the lung, and therefore by definition there will be different time constants for different areas. An alveolar unit with low resistance and low compliance will fill quickly - a so called "fast alveolus" and a unit with high resistance and high compliance will take a long time to fill, resulting in a "slow alveolus". This is not significant usually in non diseased lungs (as there is minimal differences in alveolar "speed") but may make regional ventilation dependent on the frequency of ventilation in disease states especially at high rates when the inspiratory time is decreased.

Respiratory system resistance unlike elastic resistance, respiratory system resistance, the non elastic components of impedence do not store potential energy, it is dissapated as heat. There are three main types of resistance; tissues resistance, inertance and airway resistance. The later is the most important and is considered seperately below. Tissue resistance originates from both lung and chest wall tissues, with a significant proportion originating from the chest wall. Tissue resistance can be measured in anaesthetised patients using the end inspiratory interruption method similar to the measurement for static compliance adjacent. In this instance however, it is the decrease from P1-P2 that represents tissue resistance. Inertance is analogous to electrical inductance. It represents the system resistance caused during the change in direction of gas flow. Its contribution to total respiratory system resistance is likely to be negligable, however it may become a factor during high respiratory frequencies.

Physics of gas flow Gas flows from a region of high pressure to one of lower pressure. The rate at which it does so is a function of the pressure difference and the resistance to gas flow thus being analogous to an electrical current. The precise relationship between pressure difference and flow rate depends on the nature of the flow which may be laminar, turbulent or transitional.

Laminar flow; the gas flows along a straight unbranched tube of a minimum length as a series of cocentric cyclinders that slide over each other with the cental cyclinder moving the fastest. This is particularly significant in high frequency ventilation, it means that some of the alveolar will be ventilated even when the tidal volume is less than the anatomical dead space (by the high velocity central cylinder). With laminar flow the gas flow rate is directly proportional to the pressure gradient along the tube, and it is the viscosity of the gas which influences flow rate according to the Poiseuille equation. Because it is to the fourth power, changes in radius have significant consequences.

$$\text{resistance} = \triangle P \ / \text{flow rate}$$

Poiseuille Equation

$$\text{flow rate} = \frac{\triangle P \times \pi \times \text{radius}^4}{8 \times \text{length} \times \text{viscosity}}$$

Turbulent flow high flow rates, particularly through branched or irregular tubes results in the breakdown of orderly gas flow seen in laminar flow models. The result is turbulent flow, which has a square front (rather than cone shaped). It means that no fresh gas can reach the end of the tube until the amount of gas entering the tube is almost equal to the amount of gas in the tube, thus has important consequences with respect to anatomical dead space. Flow is no longer directly proportional to the pressure drop but rather its square root. As the relationship is no longer linear, the resistance is not constant but varies with flow. Turbulent flow is not affected by gas viscosity but is inversely proportional to the square root of density. This is why divers find it difficult to breath at depth, helium has a low density and is therefore easier to breath at higher pressures if the flow is turbulent (larger airways). It has a similar viscosity to air and therefore at laminar flows it makes little difference.

Whether flow is turbulent or laminar is dependent on the Reynolds no. Re = 2(radius)(ave velocity)(density)/viscocity. The higher the number the more likely the flow is turbulent (usually >2000). Due to the complex nature of the bronchial tree it is difficult to apply the above principles. It is unlikely that flow in the respiratory system is laminar until the very small airways (probably the 11th generation) where the Reynolds numbers are low. In general, the driving pressure is determined by both the flow rate and its square $P=K(FR) + K(FR)^2$

Airways Resistance the main site of airways resistance is the medium sized bronchi. This was determined experimentally. One consequence of this is that measurements of airways resistance may not detect airways disease in the peripheries leading to a delay in diagnosis until the condition has progressed further. There are a range of factors which determine airways resistance, and these relate to the physics of resistance. Anything that reduces the calibre of airways will lead to an increase in resistance, examples of this are decreased lung volume, if the bronchi become narrowed due to bronchospasm, mucous oedema, mucus plugging, or epithelial desquamation. There are four main pathways in controlling the muscle tone in bronchioles and bronchi. Neural pathways are primarily parasympathetic activation but also reduced levels of consciousness there may be loss of pharyngeal reflexes and subsequent increase in resistance. Humoral pathways are adrenaline via the blood. Physcial and chemical effects include laryngeal spasm or subglottic oedema due to anaesthetic procedures, inhalation of an irritant such as an aerosol of water or cold air. Local cellular effects include immuno-logical activation and subsequent inflammation. Airways may become obstructed due to tumour, gastric contents or blood. In addition to changes in the calibre of the airway (and length) the other important factor in airways resistance is the composition of the gas, which dense gases increasing resistance during turbulent flow and viscous gases increasing resistance during laminar flow.

Airways resistance can be measured in anaesthetised patitents using end inspiratory interruption and is represented by Pmax-P1/flow rate. The other method involves using a body plethysmograph and is based on boyle's law to calculate the alveolar pressure accurately and therefore the using the pressure at the mouth the defference can be calculated, divide by flow and the result is the airways resistance.