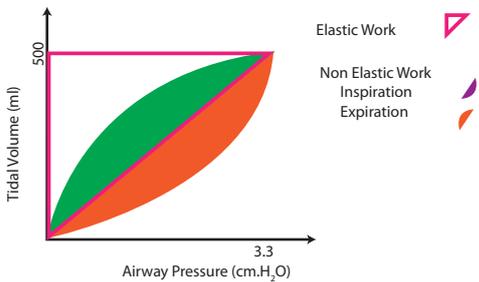


MECHANICS OF BREATHING

Work of breathing work is equal to the product of force applied and the distance moved. This is exactly analogous in the respiratory system, however in this setting it is the **pressure applied and the resultant volume attained**. The units are the same, 1 joule = 1 newton metre (1 newton moves its point of application 1 metre) = 1 litre kilopascal (when 1 litre moves in response to a pressure gradient of 1 kilopascal). The work done by the muscles of respiration result in an **efficiency of around 10% during quiet breathing** i.e. for every 10 joules of effort to result in the movement of 1 litre kilopascal, the extra work is lost overcoming the impedance in the system. Luckily **at rest the respiratory system uses only about 2% of the metabolic rate**, therefore this inefficiency is tolerated. When increased demands are placed on the system or the impedance is increased in disease states however, the efficiency decreases further and patients may become overwhelmed as any increase O₂ from ventilation is entirely consumed by the muscles of breathing.



The work of breathing overcomes **two main sources of impedance**, the **elastic recoil** of the lungs and the chest wall, whose energy is stored for further use (expiration) and **non elastic resistance** of the lung tissues, of the forces of inertia and of the resistance to airflow whose energy is lost as heat. This is shown graphically adjacent. Note that during normal breathing expiration does not incur a cost because the stored potential energy from the elastic work accounts for the non elastic force. For a constant minute volume the work done against elastic forces is increased when breathing slow and deep, conversely the work performed when there is high non elastic forces (mainly airways resistance) is increased when breathing. People with high resistance in respiratory system (asthmatics and emphysema) will tend to take slow and deep breaths and those with high elastic requirements such as those with pulmonary fibrosis will preferentially take shallow quick breaths. In both cases the patient is **minimising the impedance cost and therefore the work of breathing**. Each of these forms of impedance will be discussed in greater detail below.

Compliance The slope of the pressure-volume curve, or the **volume change per unit pressure change, is known as the compliance**. For lungs in a normal patient the compliance is 200ml.cmH₂O. If the chest wall is equally compliant then it is possible to calculate the total compliance. This is because compliance is analogous to electrical capacitance. Addition of the reciprocals of lung and chest wall equals the reciprocal of total compliance. $1/200 + 1/200 = 1/100$. Therefore TC = 100ml.cmH₂O

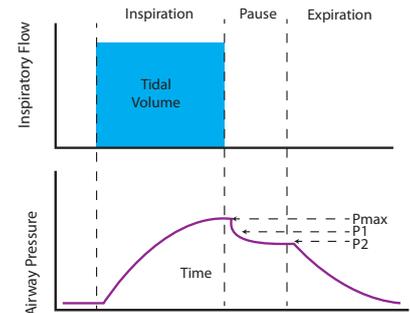
$$\frac{1}{\text{Total compliance}} = \frac{1}{\text{Lung compliance}} + \frac{1}{\text{Chest compliance}}$$

DYNAMIC COMPLIANCE

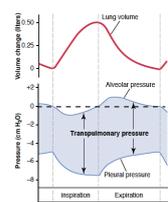
Is the slope of the volume/pressure curve (see above left) $500/3.3 = 150\text{ml/cm.H}_2\text{O}$

Compliance is measured as the change in lung volume divided by the the corresponding change in appropriate pressure gradient, there being **no gas flow when the two measurements** are made. For lung compliance the appropriate pressure gradient is alveolar-intrapleural. For chest wall compliance the gradient is intrapleural-ambient and for total compliance is alveolar-ambient. **Dynamic compliance** is measured during regular breathing **at the end of inspiration and expiration** (when there is no flow). **Static compliance** is measured by **interrupting the breathing cycle** at different times, then waiting for stabilisation before plotting the change in pressure and volume. This stabilisation period represents the tissue resistance and the airway resistance being removed from the calculation of compliance. Generally it is total compliance which is measured as it is the comparison between the ambient and alveolar pressures (pressure at the mouth equals alveolar pressure when there is no flow). It is possible however to measure lung compliance by estimating the intrapleural pressures with an oesophageal balloon. **Specific compliance** is used to compensate for the variation seen with different lung sizes and is the compliance per unit volume of the lung and is usually calculated by **compliance/FRC**. It is almost constant for both sexes and all ages down to the neonate.

STATIC COMPLIANCE

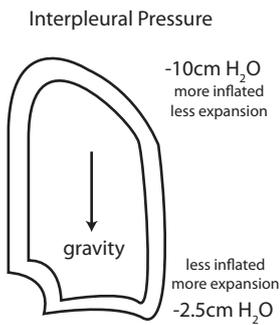


Elastic properties of the chest wall When air is introduced into the pleural space the lung collapses inwards and the chest wall springs outwards. The elastic forces of the lung and the chest wall are in equilibrium; the tendency of the lung is to contract down, and the tendency of the chest wall is to expand resulting in negative intrapleural pressure. At the **end of normal expiration the two forces balance, the lung volume is at functional residual capacity (FRC)**. The transpulmonary pressure is the difference between the alveolar and intrapleural pressures. **Chest wall compliance may be reduced by obesity, by loss of skin elasticity (eg burns patients) or by skeletal problems such as fusion of the costochondral joints**. **Posture is also very important** in terms of chest wall compliance, compared with the supine position, the seated position results in an increase in thoracic cage compliance of 30%. Furthermore, total static compliance of respiratory system is **diminished 60% in the prone position**.



Pressures and flow during the breathing cycle At rest, the pressure in the alveoli of the lung is the same as at the mouth, ie zero with respect to atmospheric pressure. The intrapleural pressure is -5cmH₂O. The volume of gas within the lungs is the FRC, and there is no gas flow into or out of the airways. **During inspiration**, intrapleural pressure falls because of the activity of the inspiratory muscles expanding the chest wall. This is transmitted across the lung, and alveolar pressure falls towards -1cmH₂O. Since this is subatmospheric air flows from the mouth to the alveoli. At the end of inspiration the intrapleural pressure is -8 cmH₂O, alveolar pressure is again atmospheric and gas flow to the lungs has ceased, the result being 500ml of air has flowed into the lungs. **Expiration** commences when the activity of the inspiratory muscles stop. The lung and the chest wall decrease in volume as the equilibrium between the expanding chest wall and the elasticity of the lung move back down to resting volume.

Regional differences in pleural pressure. It has been shown that the intrapleural pressure is less negative at the bottom than the top of the lung. The reason for this is the **weight of the lung**. The alveoli in the **upper part of the lung have a larger volume** than those in the dependent part except at total lung capacity. The greater degree of expansion of the alveoli in the upper part results in a greater transmural pressure gradient, which decreases steadily down the lung at about **1cmH₂O per 3cm of vertical height**. There are important consequences of this regional differences in transmural pressure, in particular as it pertains to ventilation-perfusion relationships and the airway closing capacity.



Ventilation consequences Now when we talk of regional differences in ventilation, we mean the **change in volume per unit resting volume**. It is clear from that the **base of the lung has both a larger change in volume and smaller resting volume than the apex**. Thus, its **ventilation is greater**. Note the paradox that although the base of the lung is relatively poorly expanded compared with the apex, it is better ventilated. The same explanation can be given for the large ventilation of dependent lung in both the supine and lateral positions.

Closing capacity The airways and alveoli in the **dependent parts of the lung are always smaller than those at the apex** (except at TLC or zero gravity when they are equal). **As the lung volume is reduced towards residual volume, there is a point at which dependent airways begin to close, known as the closing capacity (CC)**. An alternative term is the closing volume (CV) which equals the CC-RV. **Closing capacity increases linearly with age** and is less than FRC in young adults but increases to become **equal to FRC at the mean age of 44 in the supine position and 75 years in the upright position**. The closing capacity seems to be independent of body position but the FRC varies markedly with position. When the FRC is less than the CC some of the pulmonary blood flow will be distributed to alveoli with closed airways, usually in dependent parts of the lungs. This will **constitute a shunt**. Measurement of closing capacity is made by inspiration of a **tracer gas** from RV when dependent airways are closed, preferentially distributing it to the upper airways. The patient then slowly exhales from TLC. When the dependent airways close again the tracer gas will spike in concentration, at this point is the closing capacity.