B. 3 Mechanics of breathing

a. Describe the inspiratory and expiratory process involving the chest wall, diaphragm, pleura and lung parenchyma.

Inspiration

In quiet respiration, inspiration is caused by contraction of the diaphragm, innervated by the phrenic nerve from cervical segments 3-5. The dome of the diaphragm moves about 1cm caudally, increasing the volume of the chest cavity and moving the abdominal contents down. This lowers intrathoracic pressure which is transmitted through the pleural space to the lungs, expanding the lungs and causing a pressure gradient between the mouth and small airways, resulting in gas flow into the lungs.

In rapid respiration other muscles have a role in increasing the rate of gas flow into the lung. The diaphragm contracts forcefully, moving as much as 10 cm, the external intercostals lift the ribs, increasing the lateral and anteroposterior dimensions of the chest and the scalenes and sternocleidomastoid lift the first two ribs and sternum.

Expiration

Expiration is normally a passive process, relying on the elasticity of the lung parenchyma and chest wall to return the lung to its resting volume (functional residual capacity). In active expiration, the rectus abdominis, internal and external obliques and transversus contract to increase intraabdominal pressure and force the diaphragm up. The internal intercostals bring the ribs together and stiffen the intercostal spaces. These effects markedly increase intrathoracic pressure resulting in more rapid exhalation (up to a limit imposed by airway collapse).

b. Define compliance (static and dynamic) and relate this to the elastic properties of the lung.

Compliance is defined as volume change per unit pressure change. This is the reciprocal of elastance. The isolated human lungs have a compliance of about 200 ml/cmH\textsubscript{2}O in the normal pressure range of –2 to –10 cmH\textsubscript{2}O. The lungs and chest wall as a unit have a compliance of 100 ml/cmH\textsubscript{2}O. At higher pressures (and volumes), compliance is reduced. Compliance is less on the inspiratory than the expiratory phase of respiration. This phenomenon is called hysteresis.

Static compliance is determined at zero flow and is usually quoted as the expiratory compliance over the litre above FRC. Dynamic compliance is determined over a range of respiratory frequencies as uneven time constants of adjacent alveoli in diseased lungs reduce compliance at high frequencies. Specific compliance equals compliance divided by FRC.

Compliance is a result of the elastic properties of the lung. This is partly a result of the arrangement of fibres of collagen and elastin in the alveoli and airways and substantially a result of the surface tension in the alveoli.

Surface tension in the alveoli is modified by surfactant secreted by type II pneumocytes. It displays non-linear properties, being much greater at high volumes than low volumes and thus produces hysteresis.

Specific compliance = static compliance ÷ FRC (normal 0.05 /cmH\textsubscript{2}O)

c. Explain the concept of time constants and relate these to “fast” and “slow”
alveoli.

Compliance equals change in volume per unit pressure. Resistance equals airway pressure per unit flow. The product of compliance and resistance is the “time constant” of a component of the lung; a measure of the rate of filling of that lung unit. A large time constant can result from airway obstruction or extreme distensibility of the unit. Areas of lung with a long time constant fill more slowly than the rest of the lung during inspiration and may still be filling (from the rest of the lung) when expiration has already begun. This is called “Pendelluft”.

Lungs with a wide range of time constants among their alveoli require slow respiration to be ventilated properly. As frequency of respiration increases, “slow” alveoli fill only partially before alveolar pressure rises high enough in expiration for them to start to empty again. This reduces the tidal volume and thus the apparent compliance of the lung as the frequency of respiration increases.

d. Describe the elastic properties of the chest wall and plot the pressure-volume relationships of the lung, chest wall and total respiratory system.

The chest wall is elastic. At resting volume, it pulls against the pleural space with a pressure of about -5 cmH₂O. This balances the elastic recoil of the lung, yielding a negative pleural pressure and zero pressure within the airways and outside the chest wall. Under normal circumstances the pleural space has a total volume of only a few ml. If it is opened, air is rapidly drawn in, the chest wall springs out and the lung collapses.

e. Describe the properties of surfactant and relate these to its role in determining respiratory mechanics.

Surfactant is secreted by type II pneumocytes. It lines the air-tissue interface of the alveoli and modifies the surface tension at the interface, preventing collapse and greatly increasing compliance. A major constituent of surfactant is dipalmitoyl phosphatidyl choline, synthesized from fatty acids. This is a long molecule with hydrophilic and hydrophobic ends.

When densely packed, DPPC molecules provide a strong repulsive force which opposes surface tension. As the surface area of an alveolus increases, the repulsion between DPPC molecules is reduced as they spread apart. At the same time, surface tension is less as it varies with the reciprocal of the radius of an alveolus.

The behaviour of surfactant is complex as it displays a greater effect as surface area is falling (during expiration) than rising. Normal detergents reduce surface tension but usually do so by a fixed amount regardless of area and do not display hysteresis.

The effect of surface tension is responsible for more than 80% of the work or inflating the lung, having a much greater effect at normal volumes than the tissue elasticity of the lung.
f. Explain the vertical gradient of pleural pressure and its significance.

In the erect position at resting volume, pleural pressure varies from –2.5 to –10 cmH₂O from base to top of the lung because of its weight. The resting volume of alveoli at the apex of the lung is much greater than at the base because of the more negative pleural pressure. However, the ventilation of the apex of the lung is proportionally less than the base during the respiratory cycle because the lung is more compliant at the lower volume and expanding pressure.

At low volumes, the pleural pressure at the base of the lung may become greater than atmospheric, resulting in airway closure and poor ventilation, while the apex benefits from a greater compliance and better ventilation. Transpulmonary pressure is defined as alveolar minus pleural pressure.

g. Explain the physics of gas flow and the significance of the relationship between resistance and pressure in the respiratory tract.

At low speeds and in smooth, small tubes, laminar flow is present, with a steady increase in flow speed from the edge to the centre of the tube. Under these circumstances, flow is described by the Poiseuille equation:

\[ \dot{V} = \frac{\pi Pr^4}{8\eta l} \]

As speed and tube size increase, flow may become turbulent after a transitional phase. Turbulent flow exists when Reynolds number exceeds 2000 and laminar flow below 200. The transition from laminar to turbulent flow is not predictable. Reynolds number is given by the equation:

\[ RN = \frac{\nu \rho d}{\eta} \]

where \( \rho \) is density, \( d \) diameter, \( \nu \) velocity and \( \eta \) viscosity. Where flow is turbulent, its relationship to the radius of the tube is not simple, being proportional to greater than the fourth power. The relationship to pressure, density and length of tube is:

\[ \dot{V} \propto \frac{P}{\sqrt{\rho l}} \]

flow being unrelated to viscosity.

Resistance is equal to pressure gradient per unit flow, so each of these flow equations can be transformed into an expression of resistance.

In the respiratory tract, air flow is turbulent in the trachea, transitional in all the large airways down to the level of terminal bronchioles and probably laminar in the very small airways. The relationship between driving pressure and air flow is complex and of the form:

\[ P = K_1\dot{V} + K_2\dot{V}^2 \]

The first term reflecting laminar flow and the second turbulent flow.

h. Describe the factors affecting resistance and how to measure airway resistance.

Most of the resistance to air flow in healthy lungs is in the medium sized bronchi, though diameter falls with each generation of airways, the total number of airways rises exponentially, reducing resistance to very low levels by the tenth generation.

Density and viscosity of inspired gas are related to resistance as described in the above equations.

Lung volume has a major effect on airway resistance.
At low lung volumes there is little support of small airways by the surrounding parenchyma and consequently high resistance, varying with the reciprocal of lung volume. Smooth muscle tone in bronchioles can markedly increase airway resistance. The bronchioles are innervated by the vagus. Contraction can be from parasympathetic outflow, local irritant factors or systemic factors such as histamine release. Bronchiolar smooth muscle also relaxes in response to a rise in $\text{PCO}_2$.

In the special case of maximal expiratory flow, resistance rises as the gradient from intrapleural pressure to airway pressure exceeds the force supporting the airways and collapse occurs. This limits flow regardless of the resistance downstream. The force supporting the airways depends on lung volume. This produces an effort-independent maximal flow envelope related only to lung volume in any individual.

Airway resistance is measured using a plethysmograph. By measuring airflow at the mouth and pressure change in the plethysmograph, having first measured lung volume, it is possible to calculate the pressure gradient between alveoli and mouth which when divided by flow equals total airway resistance.

Less satisfactorily, intrapleural pressure can be measured with an oesophageal manometer in the lower $\frac{1}{3}$ of the oesophagus when erect. If the recoil due to chest wall elasticity is reliably calculated, the pressure gradient due to both airway and tissue resistance can be determined.

Information about resistance can also be obtained using a single forced expiration. The conventional measures are $\text{FEV}_1/\text{FVC}$ (normal $>$80%) and $\text{FEF}_{25-75\%}$ which is an average flow over the middle 50% of expiration (by volume). A simpler measure is $\text{PEFR}$, commonly used to assess the severity of obstruction in asthma.

More detailed information can be obtained from a flow-volume curve, in which the expiratory flow rates are much lower in airway obstruction and the mean volume may be increased in chronic disease.

i. Define closing capacity and its relationship to airway closure and explain its clinical significance and measurement.

In a single breath $\text{N}_2$ washout test, (used to measure anatomical deadspace), the exhaled nitrogen concentration rises above its alveolar plateau at a low lung volume (in healthy individuals). This represents the exhalation of alveolar gas from less well-ventilated alveoli, usually in the upper part of the lung, following closure of airways in the bottom of the lung.

The volume at which this rise starts is that at which airway closure first occurs in the more dependent part of the lung. This is about 10% of $\text{VC}$ in healthy young adults and rises with age (45 ml/year) and airway disease to equal $\text{FRC}$ at 45 years in the supine position and 65 years when erect. Closing capacity is also higher in infants, falling to $\text{FRC}$ at 8 years.

j. Describe the work of breathing and its components.

Work is equal to pressure times volume. The work of breathing may be described using a pressure-volume graph. The area of the graph represents the work of breathing; the pale part being work against elastic forces (on inspiration) and the dark area work against viscous forces such as airway resistance. On expiration, the viscous work required is less than the energy stored in elastic forces during quiet breathing.

In exercise or during rapid breathing, the work against viscous forces increases.
dramatically and on expiration will exceed the work done against elastic forces on inspiration. Expiration then becomes an active process and the expiratory limb of the curve falls outside the pale area.

Work of breathing at rest is about 2% of BMR (2 W or 7.2 kJ/hr) with 10% efficiency. There is a characteristic frequency of minimal work of breathing for a given minute volume and elastic properties and airway resistance, with high elastic work at low frequencies and high airway resistance work at high frequencies. Conversely the frequency of minimum work is reduced by an increase in airway resistance and increased by a rise in elastic resistance.

**k. Describe altered lung mechanics in disease states.**