Respiratory Mechanics

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Inspiration

Diaphragm moves downward and Intercostal muscles and ribs move upward and outward. This produces an increase in the vertical length and circumference of the chest cavity.

In normal respiration a Tidal volume (TV) of about 500ml is produced.
The diaphragm moves about 1cm downward producing about 100ml of this TV.
The circumference of the chest increases by about 1.3cm over its entire length (about 30cm) producing the remaining 400 ml needed to create a total TV of 500ml.

In active inspiration the diaphragm will only move down by a maximum of 3cm (about 300ml) while the circumference increase (10-12cm) is responsible for the remaining 3 -4000ml change in TV.

Exhalation is essentially passive but may be supplemented the abdominal muscles increasing the intra-abdominal pressure, forcing air out of the lungs. (The passive nature of exhalation is shown on a pressure volume curve for a lung) (See Below)

Measurements of lung mechanics

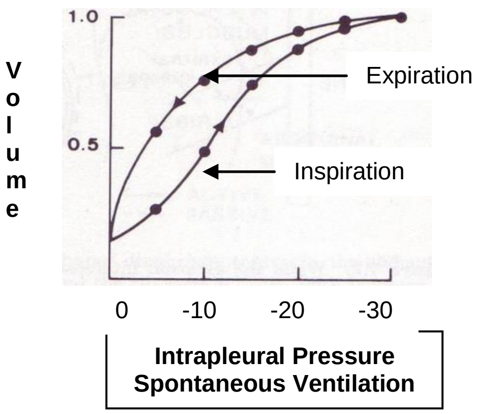
- **Static** - Measurements made when air is not moving (e.g. lung compliance)
- **Dynamic** - Measurements made determined when air is moving (e.g. FEV1)
- **Volume** – A measured number (e.g. Tidal volume)
- **Capacity** – A calculated number (e.g. Total lung Capacity = TV + RV +IRV + ERV)

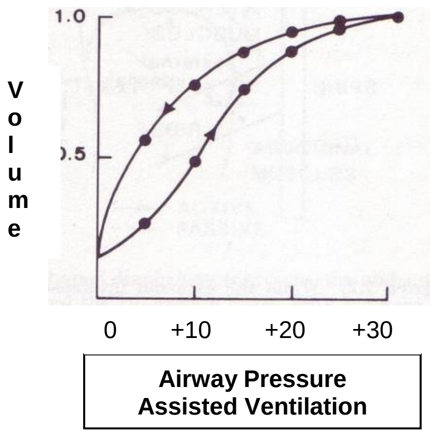
Pressure volume relations of the lung

Spontaneous ventilation (SV) depends on the generation of negative pressure in the pleural space. As the chest wall moves away from the lung a space is produced which results in a negative pressure between the chest wall and the lung. The greater the distance the chest wall moves the more negative this pressure will be. This negative pressure results in air being sucked inwards into the lung.

In assisted ventilation (AV) air is forced into the lung pushing out the chest wall. An increase in pressure pushing the lung outward produces a bigger volume in the lung.

Therefore as the pressures inside the lung changes so does the volume.





Important things to notice on these curves

- There is no difference between the change in volume for SV and AV.
- The pressure in SV is negative and reflects the intrapleural pressure while AV is positive and reflects the pressure in the airways.
- In exhalation the curve follows a different path to inspiration – **Hysteresis** - due to air exiting the lung passively.
- There is always some air left in the lung after exhalation so inspiration never starts from zero (residual volume).
- Zero pressure is atmospheric pressure.

Compliance
Is the ability of the lung to accept volume as the pressure changes.
It is calculated as:

$$\frac{\text{Change in volume}}{\text{Change in pressure}} \qquad \text{(Normal is 200ml/cm } H_2O\text{)}$$

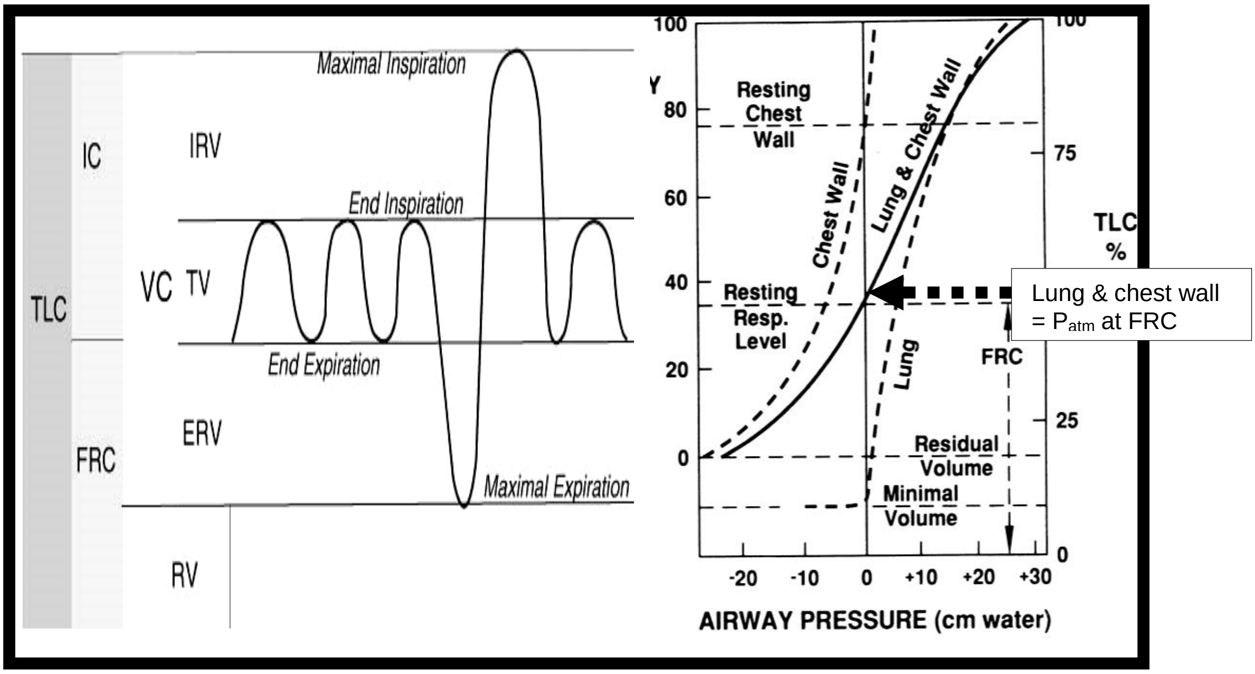
It is dependent on
- The elastic stretch of the alveoli. (Static compliance). This is determined by collagen, elastin & air fluid interfaces (surfactant).
- The chest wall and pleural cavity rigidity. (Static compliance)
- The airway resistance to gas flow. (Dynamic compliance) Which is affected by the diameter of the conducting airway, and the work needed to open that airway.

Compliance is constantly changing, varying in different parts of the lung and with different pressures. It is also affected by oedema fluid in the alveolus and interstitium, atelectasis, loss of surfactant, fibrosis, age, COPD and chest wall disease.

Compliance and recall of the lung and chest wall in combination

Fundamentals
In a pneumothorax the lung collapses and the chest wall moves outwards!

1. The lung is always at a positive pressure (above P_{atm}) and wants to collapse.
2. The chest wall is always kept at a pressure less than P_{atm} and wants to spring outwards.
3. At FRC (functional residual capacity) the positive pressure in the lung balances the negative pressure in the chest wall and normal spontaneous respiration starts from here i.e. we breathe our tidal volume from atmospheric pressure.
4. With inspiration the chest wall is pulled outwards and it draws the lung out at the same time increasing the pressure in both
5. The chest wall pressure only reaches P_{atm} with deep active inspiration.



Airway resistance

Airflow
3 types of airflow are seen in the airways
1. Turbulent flow (In trachea)
2. Laminar flow (In very small airways)
3. Transitional (A mixture of Laminar and turbulent - in the majority of the respiratory tree)

Flow characteristics are determined by Reynolds number (Re) with radius and velocity of flow being the principal determinants.
(>2000 turbulent & <10 laminar)

$$Re = \frac{2rvd}{\eta}$$

r = radius
v = average velocity
d = density
η = viscosity
l = length
R = resistance
V = flow
P = pressure

From *Ohms* law we know that
Pressure (P) = Flow (V) x Resistance (R)
Or $R = \frac{P}{V}$

Poiseuille determined flow in a tube to be:

$$V = \frac{P \pi r^4}{8\eta l} \rightarrow \frac{V}{P} = \frac{\pi r^4}{8\eta l} \rightarrow \text{and} \quad \frac{V}{P} = \frac{1}{R}$$

therefore $R = \frac{8\eta l}{\pi r^4}$

Halving the radius increases resistance 16 times while doubling the length only doubles it.

Determinants of airway resistance (Inspiratory & expiratory)

1. Bronchial divisions
- Major site of resistance is the first 7 divisions.
- Small airways produce less than 20% of airway resistance

2. Lung volume
- Increased volume decreases resistance
- Additional volume opens alveoli and terminal bronchioles
- Small tidal volumes may produce increased closing capacity increasing resistance

3. Pulmonary vasodilation
- Distended blood vessels obstruct terminal bronchioles

4. Bronchial smooth muscle contraction
- Worsens resistance and may be reversed by bronchodilators

5. Density and viscosity of gases
- Helium-O_2 admixtures decrease density and resistance, improving flow

6. Dynamic airway compression
- Expiratory flow is effort independent at low flow rates due to terminal bronchial compression by intra-thoracic pressure.
- Terminal bronchi which are not cartilage supported collapse when external pressure reaches the pressure within the bronchus
- This is worsened by forced expiration, loss of elastic tissue in COPD, increased airway resistance and the equal pressure point approaching the alveolus as flow decreases.
- The inability of alveoli to empty increases the intra thoracic pressure further (stacking)

Work of breathing

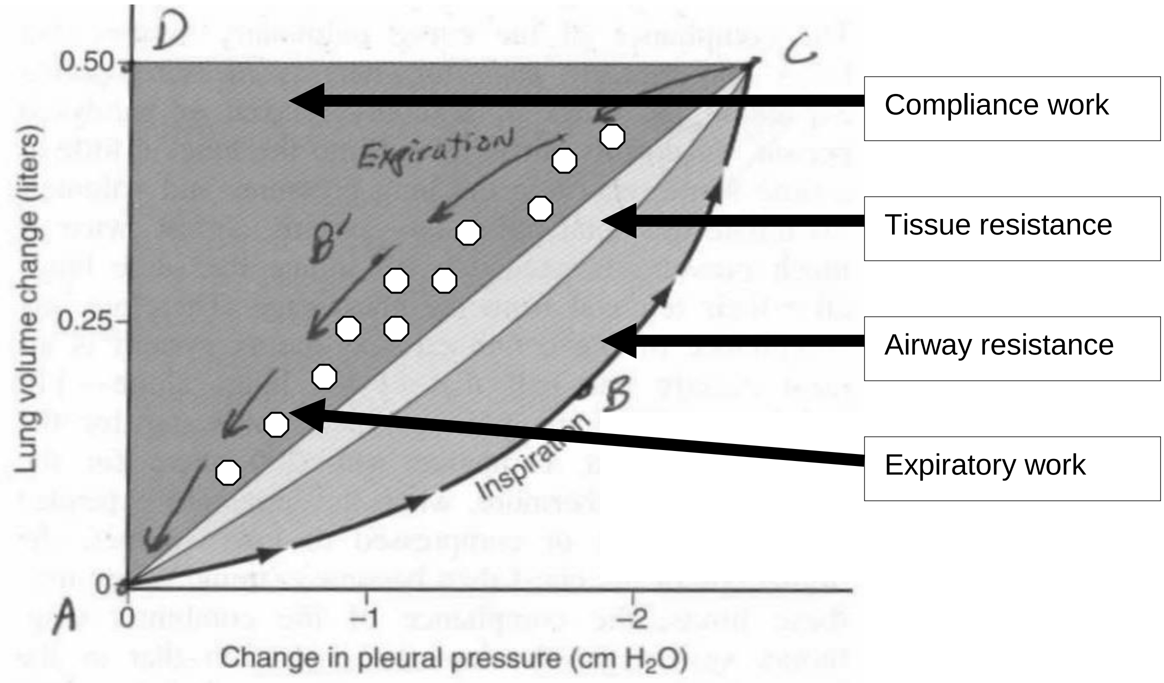
Work = force x distance
Force = pressure x area
Distance = volume/area
Work = (pressure x area) (volume/area)

Work = Pressure x Volume

The total work of is the sum of work needed to overcome:

- Compliance - essentially the work needed to overcome resistance from elastic forces of the lung and chest wall.
- Airway resistance
- Tissue resistance is the resistance provided by the lung pleura moving against the chest wall pleura. It is often added to airway resistance and called viscous resistance.

Work = Compliance + Airway resistance + Tissue resistance



Important things to notice from this curve:

1. Total work is all the areas added together
2. Exhalation work falls within the total work area and is done using the stored elastic energy from the compliance work
3. The compliance work less the expiratory work is dissipated as heat
4. Compliance work increases with higher tidal volumes
5. Viscous work (tissue & airway) increases with faster breathing and flow rates.
6. In COPD slow breaths with big tidal volumes takes advantage of increased compliance (less compliance work needed) while in restrictive airways disease fast and shallow breathing means less work against decreased compliance.

It is very difficult to measure work of breathing.
Work of breathing can be estimated from the percentage of O_2 consumption required for breathing.
Normal resting respiration requires less than 5%.
In COPD this can climb above 30% limiting effort tolerance.

References

- Respiratory physiology, John B West 6th edition
- Textbook of Medical Physiology, Guyton & Hall, 10th edition
- Anesthesia, Ronald D Miller 6th edition