Basic Respiratory Physiology

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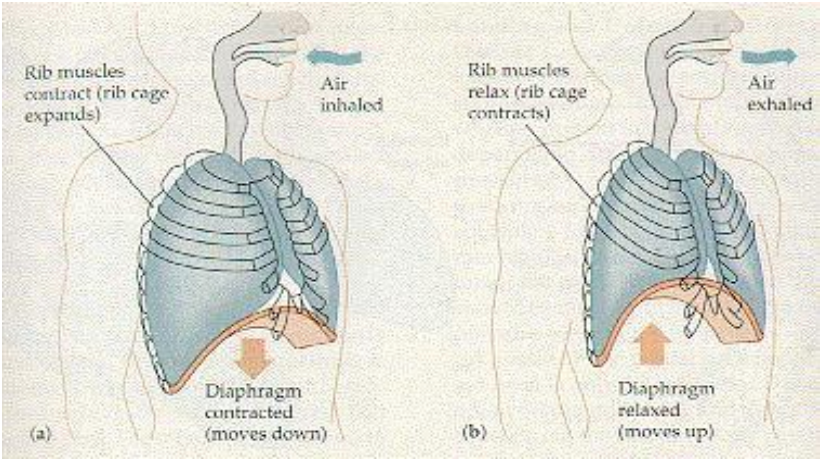
NORMAL RESPIRATORY PHYSIOLOGY

During inspiration, active contraction of respiratory muscles (intercostals, accessory muscle and diaphragm) causes the thoracic cage to move and expand in volume thereby creating negative intrathoracic pressure. As a result, atmospheric air rushes through air passages into the lung parenchyma. The force expanded is used to overcome the elastic properties of the thoracic cage and surface tension within the alveoli surfaces of the lung as well as the resistance of air passages. (Fig. 1)

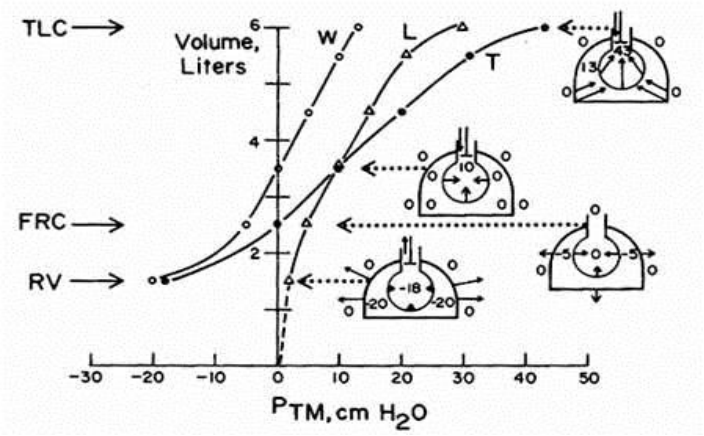
Fig 1. External Respiration



http://www.biology.eku.edu/RITCHISO/301notes6.htm

Expiration is a passive process. During expiration, various forces such as stability of thoracic cage and laryngeal cords prevent the lung from total collapse. When equilibrium is achieved between elastic recoil of the lung (tending to collapse) and thoracic cage wall (tending to expand), functional residual capacity (FRC) is reached. (Fig. 2)

Fig 2. Normal lung pressure-volume relationship



http://physioweb.med.uvm.edu/pulmonary_physiology

Compliance and *resistance* of the respiratory system will determine the work of breathing required for respiration. Mechanical ventilation greatly reduces or abolishes the work of breathing. This reduces oxygen needs and carbon dioxide production, which in turn reduces ventilatory requirements.

ELASTIC PROPERTIES OF THE LUNG

Compliance of the lung is measured by change in lung volume against a change in airway pressure. For alveoli to expand, the airway pressure must overcome strong elastic recoil forces of the alveoli. Once the initial inertia in alveoli opening is overcome (lower inflection point), the airways forces needed to expand the alveoli is near linear as depicted on the pressure-volume curve. Further expansion is possible until the maximal elastic property of the alveoli is reached and the pressure-volume relationship becomes non-linear (upper infection point). (Fig. 3) Attempts to distend the alveoli further will yield little change in volume but may cause damaging disruption to tissues and lung injury (zone of over-distension). Similarly, below the lower infection point, atelectatic segments experience shears stress as they pop open and close and this can also result in lung injury (zone of collapse). (Fig. 4)

Fig 3. Zones of De recruitment and Over distension

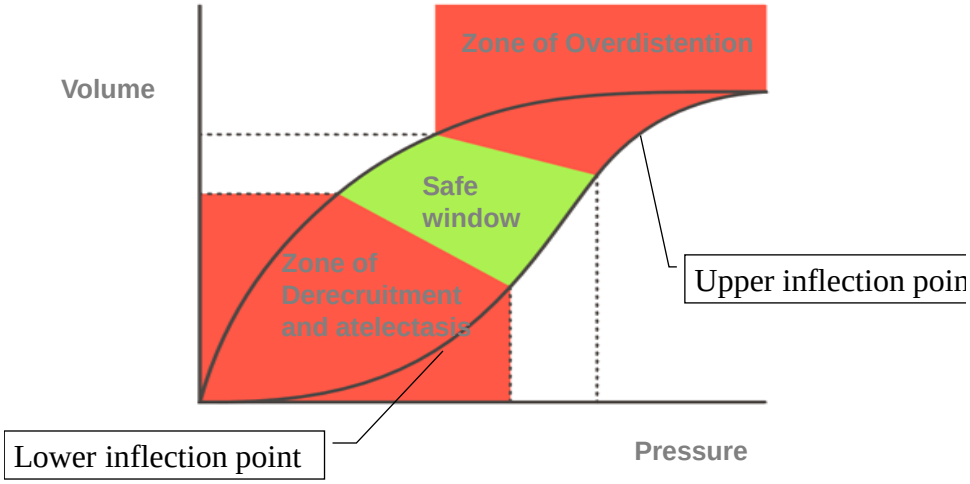
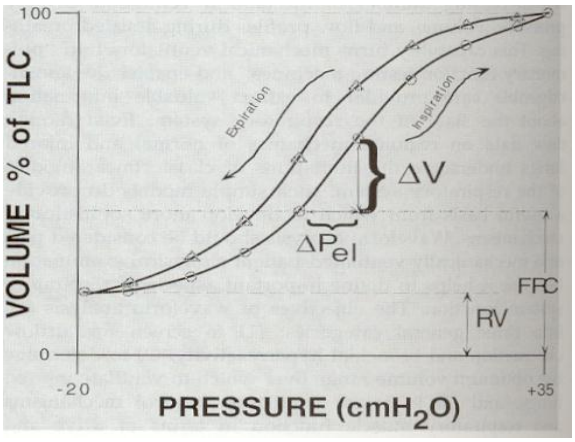


Fig 4. Compliance



Burton SL & Hubmayr RD: Determinants of Patient-Ventilator Interactions: Bedside Waveform Analysis, in Tobin MJ (ed): *Principles & Practice of Intensive Care Monitoring*

Fortunately in healthy lungs, mechanical ventilation usually occurs between these 2 zones of potential harm. When the lung deflates from total lung capacity, the pressure-volume relationship is not similar to that experienced during inflation. This is the result of the property of surfactant that reduces surface tension and the effect changes depending whether the alveoli is fully expanded or closed. This effect is known as *hysteresis* and is an

important consideration while manipulating ventilatory strategies to recruit alveoli that are collapsed due to disease.

In severe lung disease states like acute respiratory distress syndrome, pulmonary edema and interstitial lung disease, the elastic property of the lung is decreased and it is possible for the pressure-volume relationship to be so altered that the lung segments may transverse these 2 zones during a tidal breath.

In order to prevent ventilator induced lung injury (VILI), it is essential to pay close attention to the airway pressures during mechanical ventilation. However, measured airway pressure may not necessarily reflect the pressure that the alveoli are exposed to. This is because of the smaller endotracheal tubes used in paediatrics, discrepancy between alveoli-pleural pressure gradient with measured airway pressure and in situations when the thoracic cage is stiff.

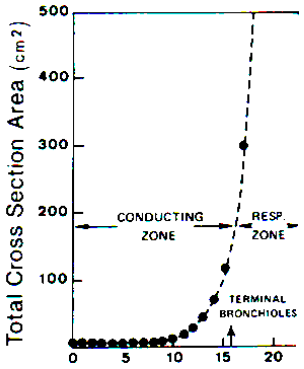
Results from adult lung injury trials have shown that controlling delivered tidal volume is a good way to limit VILI. Tidal volume around 6ml/kg is considered lung protective. However, the total dead space in paediatrics (including endotracheal tube) may be 2-3ml/kg. Therefore, providing too low a tidal volume may cause unanticipated lung collapse and excessive dead space effect.

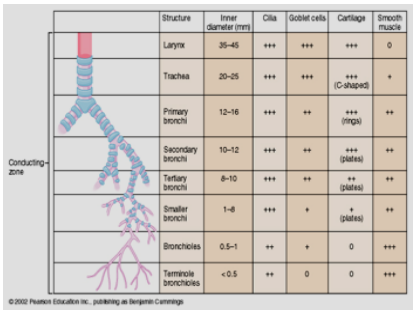
Another lung protective measure is to prevent lung closure during expiration, especially during mechanical ventilation when the auto air-trapping mechanism of the laryngeal cords is bypassed by the endotracheal tube. It is therefore prudent to administer positive end expiratory pressure (PEEP) to prevent end expiratory lung collapse. PEEP also helps to keep diseased atelectatic lung units that have been opened during inspiration to remain patent (recruitment).

RESISTIVE PROPERTIES OF THE LUNG

Airflow resistance increases as the diameter of the air passages narrows. However, with each succeeding generations, there is an exponential increase in the number of airways. Therefore the combined cross section surface area increases dramatically and the *total* airway resistance falls as a result. (Fig 5)

Fig 5. Cross sectional area of airways





Nevertheless, the paediatric airway is extremely vulnerable to pathology that reduces airway diameter like secretions (infections) and edema (asthma). Resistive forces of the airways obey the Poiseuille's law, which states that airflow resistance is related to the viscosity of flow and length and diameter of the passage within which air flows take place.

A small decrease in diameter can lead to a large increase in airway resistance. When the resistance is high, expiration may become an active process in order for air to be forced out.

In severe airway obstruction, the airways may even collapse resulting in excessive air trapping (*intrinsic PEEP*). Forced expiration may also cause dynamic airway collapse, and effect which is exaggerated in children with broncho-malacia.

During spontaneous respiration in children with obstructive airway disease, the chest is hyperinflated due to increased FRC. This serves to keep the airways open due to greater negative intra-thoracic pressures as a result of increased thoracic volume. The intrinsic elastic tone of respiratory muscles also increases and this may help with expiration.

In children with bronchiolitis and status asthmaticus who require mechanical ventilation, it is ideal to allow the patient to breathe spontaneously as negative intra-thoracic pressures encourages airway opening. It is also important to monitor for evidence of air trapping and airway collapse. A high airway pressure is usually needed to open during inspiration but it is also important to keep airways open during expiration when it is most likely to collapse using PEEP. The PEEP applied with the ventilator should match intrinsic PEEP and the resultant increase in FRC mimics the physiological benefits effect seen during spontaneous breathing.

GAS EXCHANGE

Airflow in the proximal airways is mainly laminar but by the fifth generation of airways, majority of the airflows to the alveoli is conducted by diffusion.

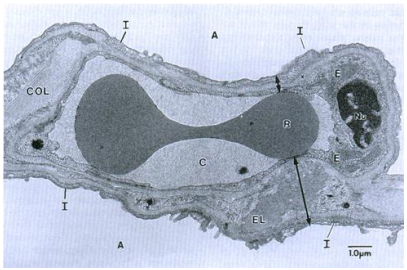
At the alveolo-capillary barrier, determinants of gas exchange are:

- cross sectional area available for diffusion
- partial pressures of the gas between the barrier

- thickness of the barrier. (Fig 6)

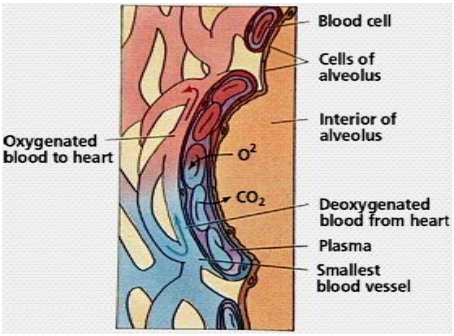
In situations where there is ventilation-perfusion imbalance, venous admixture can result. This can arise when pulmonary blood flows into non-ventilated alveoli or when ventilated alveoli are poorly perfused. The former arises when lung units are 'lost' due to disease or collapse and ventilatory strategies can help to open or recruit these 'lost' units.

Fig 6. Blood Gas Barrier



$$V = \frac{A.\ D_{gas}\ \Delta p_{gas}(alv\text{-}cap)}{T}$$

- Fick's Diffusion Law
  - Pressure gradient **ΔP**
  - Thickness **T**
  - Surface area **A**
  - Gas Properties **D**



Murray, et al. Textbook of Respiratory Medicine 3rd ed. Saunders 2000, p.16.

Increasing mean airway pressures through increasing peak inspiratory pressure (PIP), positive end expiratory pressure (PEEP), inspiratory time (Ti) can facilitate gas exchange by recruiting non-ventilated lung units.

NEONATAL CONSIDERATIONS

The respiratory reserves in the neonate are poor as a result of the following physiological and anatomical differences:

- the neonatal chest wall is much more compliant than adult, the end expiratory lung volume maybe very small and prone to airway closure and collapse
- horizontal profile of the ribs and diaphragm results in poor respiratory mechanics.
- the intercostals and diaphragmatic muscles are not well developed.
- Central respiratory control is immature

As a result, respiratory decompensation can occur quickly.

REFERENCES

1. Nicolai T. The physiological basis of respiratory support. Paediatric Respiratory Reviews 2006: 7; 97-102.
2. Lands LC. Applying physiology to conventional mechanical ventilation. Paediatric Respiratory Reviews 2006: 7S; S33-S36.
3. Acosta P et al. The use of PEEP in Mechanical Ventilation. Critical Care Clinics 2007; 23: 251-261.

4. Greenough A et al. Matching Ventilatory Support Strategies to Respiratory Pathophysiology. Clinics in Perinatology 2007; 34: 35-53.
5. Eskandar N et al. Weaning from Mechanical Ventilation. Critical Care Clinics 2007; 23: 263-274.
6. Haitsma J J. Physiology of Mechanical Ventilation. Critical Care Clinics 2007; 23: 117-134.
7. Magnay A R. Recent Advances in intubation and mechanical ventilation. Paediatric Respiratory Reviews 2001; 2: 184-194.
8. Koh S O. Mode of Mechanical Ventilation: Volume Controlled Mode. Critical Care Clinics 2007; 23: 161-167.
9. Nichols D, Haranath S. Mode of Mechanical Ventilation: Pressure Controlled Mode. Critical Care Clinics 2007; 23:183-199.