Chapter 1
Mechanical Characteristics of the Lung and the Chest Wall

Ashok P. Sarnaik

Mechanical properties of the respiratory system govern the principles of air movement between the alveoli and the atmosphere. For air to move from one end to another, a pressure gradient is required. As long as there is some communication between the alveoli and the atmosphere, gas will flow (volume/time) from a higher pressure to a lower pressure until the pressures at both ends equilibrate resulting in cessation of flow. Pressure equilibration is not instantaneous; it requires time. Insufficient time will prevent pressure equilibration, and therefore the potential change in volume. Resistive properties of the respiratory system oppose generation of flow whereas the elastance characteristics oppose change in volume.

1.1 Lung Volumes and Capacities

Along with pressure, knowledge of lung volumes and capacities is crucial to understanding normal lung function as well as many pathological conditions (Fig. 1.1). *Tidal volume* (VT) is the volume of gas moved with each breath. In health, spontaneous VT is usually between 6-8 ml/kg. This volume refreshes alveolar gas with atmospheric air during inspiration and leads to removal of CO_2 during exhalation. The volume of gas remaining in the lung after tidal exhalation is termed *functional residual capacity* (FRC). FRC is measured either by measurement of

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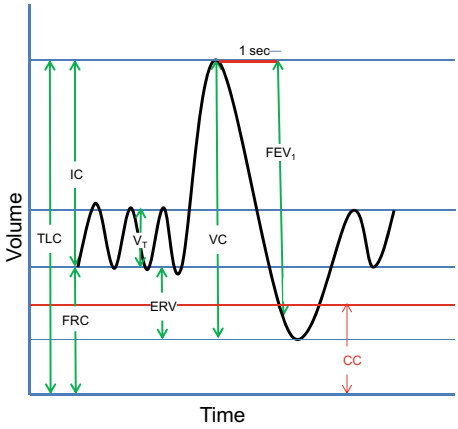


Fig. 1.1 Spirometry showing lung volumes and capacities. Total lung capacity = TLC, IC = Inspiratory capacity, FRC = Functional residual capacity, VT = Tidal volume, ERV = Expiratory reserve volume, VC = Vital capacity, FEV_1 = Forced expiratory volume in 1 s, CC = Closing capacity (measured only by gas dilution techniques). Reprinted with permission from Sarnaik AP, Heidemann S and Clark JA, Nelson Textbook of Pediatrics, 20th Edition, Kliegman, St. Geme et al. Editors, Elsevier 2016

thoracic gas volume by plethysmography or by helium dilution method. FRC acts as a reservoir for gas exchange between the alveoli and the pulmonary capillary blood throughout respiration. Diseases that decrease lung compliance and lower FRC can have profound effects on oxygenation. Positive end-expiratory pressure (PEEP) helps maintain end-expiratory volume and improve oxygenation. *Residual volume* (RV) is the volume of gas remaining in the lung after a maximal forced exhalation. The difference between FRC and RV is the *expiratory reserve volume*. *Inspiratory capacity* (IC) is the volume that can be inspired from FRC after a maximal inspiration from a normal exhalation and *total lung capacity* is the total volume of gas in the lung at maximum inspiration. Closing capacity (CC) is volume of gas in the lung during exhalation when the dependent airways start to close. Closing capacity can only be measured by specific gas dilution techniques and not by spirometry. In healthy children and adults, CC is well below the FRC, meaning that all the airways remain open during tidal respiration. In intrapulmonary obstructive diseases and even in healthy neonates, dependent airways start to close during tidal exhalation before reaching FRC. When CC is greater than FRC, the alveolar ventilation moves towards the nondependent, less perfused areas, away from dependent and better perfused areas, resulting in V/Q mismatch and a lower PaO_2. This also results in some amount of air trapping.

1.2 Pressure

In regards to pulmonary mechanics, pressure is generally described in relation to atmosphere which is considered to be 0 cm H_2O. During spontaneous breathing, inspiration results from negative pressure in the pleural space and alveoli drawing air from the atmosphere, while during positive pressure breathing air is pushed into the alveoli from a higher pressure source. There is a lack of consistency with terms and symbols used to describe reference pressures and pressure gradients. For the purpose of this discussion, we will use the following terminology (Fig. 1.2).

Proximal airway pressure (P_{AW}) is measured at the mouth during spontaneous breathing, inside the ventilator, at the patient-support device interface during non-invasive breathing support or in the hub of the endotracheal tube during invasive mechanical ventilation. When measured at the mouth during spontaneous respiration, it is same as the atmospheric pressure (P_{ATM}) or body surface pressure (P_B) which is referred to as 0 cm H_2O. During mechanical ventilation, P_{AW} is usually measured in the ventilator via a pressure transducer in various phases of respiratory cycle. Alveolar pressure (P_{ALV}) is inferred by inspiratory and expiratory occlusion techniques, allowing the P_{ALV} to equilibrate with the P_{AW} by accomplishing a "no flow" state and measuring pressure at the proximal airway. A no flow state assumes equalization of pressure at both ends of the system. Intrapleural pressure (P_{PL}) is not directly measured in clinical practice. Instead, it is inferred by measuring esophageal pressure (P_{ES}) by a balloon placed in distal esophagus.

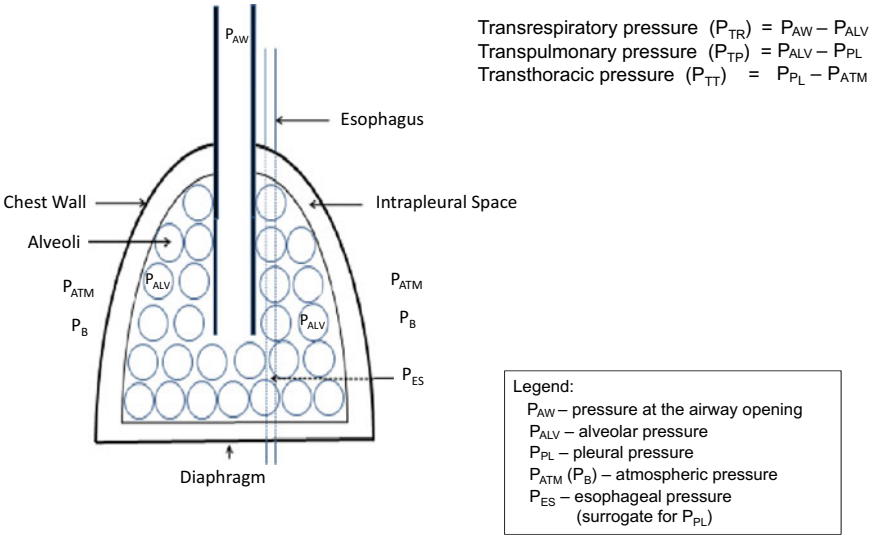


Fig. 1.2 Schematic presentation of various sites at which reference pressures are measured. P_{ATM} or P_B = atmospheric or body surface pressure, P_{AW} = proximal airway pressure, P_{ALV} = alveolar pressure, P_{PL} = intrapleural pressure, P_{ES} = esophageal pressure (used as a surrogate for P_{PL})

1.3 Pressure Gradients

Transrespiratory pressure is the pressure difference between P_{AW} and P_{ALV}. Transthoracic pressure ($P_{ATM} - P_{PL}$) is the pressure difference that thoracic cage is subjected to throughout the respiratory cycle. P_{ES} is used as a surrogate measure of P_{PL}. Transpulmonary pressure ($P_{ALV} - P_{PL}$) is the pressure difference between the alveolar pressure and the pleural pressure and is indicative of the pressure responsible for maintaining alveolar inflation. It reflects the stress the alveoli are exposed to during inflation and deflation with mechanical ventilation. Measurement of transpulmonary and transthoracic pressures allow the partitioning of the combined lung-thorax mechanics into separate chest wall and alveolar components. Trans-airway pressure ($P_{AW} - P_{ALV}$) denotes the pressure difference that influences air movement (flow) across the airways. It is used to calculate airway resistance (pressure/flow).

1.4 Surface Tension

Alveolar surface is lined with a liquid film creating an air-fluid interface for gas exchange. Alveoli are connected to each other and the atmosphere via airways. The pressure required to keep an alveolus open is governed by Laplace's law which is expressed as:

$$P = \frac{2T}{r}$$

where P is the pressure required to inflate the alveolus, T is the surface tension at the air-fluid interface, and r is the radius. Pressure needed to keep the alveolus open is greater, with higher surface tension and smaller radius. If surface tension remains the same, smaller alveoli will tend to collapse and empty into larger alveoli resulting in atelectasis. Pulmonary surfactant is produced by type II alveolar cells and forms the lining of alveolar air interface. There are two major functions of surfactant. Surfactant is necessary to decrease the surface tension thereby requiring less pressure (critical opening pressure) for alveolar inflation. Surfactant also decreases the surface tension of smaller alveoli to a greater extent compared to the larger ones. Because the concentration of surfactant is greater in smaller alveoli compared to larger alveoli, the surface tension is decreased to a greater extent thus equalizing the pressure in alveoli of different sizes (Fig. 1.3).

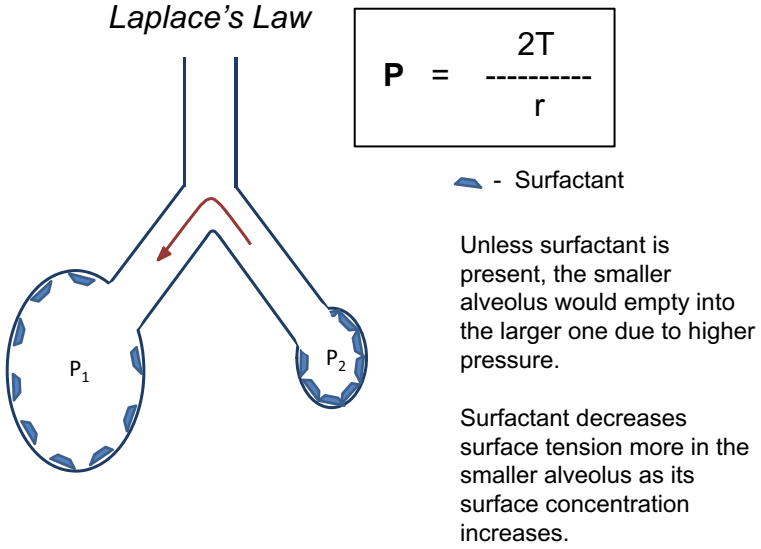


Fig. 1.3 Pressure generated in a smaller alveolus (P_2) is greater than that in a larger alveolus (P_1) with a tendency of the smaller alveolus to empty into the larger one. Surfactant, in general, lowers the surface tension. Being more concentrated in a smaller alveolus, surfactant lowers the surface tension to a greater extent allowing alveoli of different sizes to remain open and in communication with each other.

1.5 Elastance and Compliance

Elastance is the property of a substance to return to its original state when the deforming stress (e.g. pressure) is removed. Pulmonary elastance is determined by two factors: elastin in elastic fibers in the connective tissue of the lungs including the airways and surface tension in the alveoli. Elastance is decreased with loss of elastin and increased with increased surface tension or accumulation of fluid and inflammatory material. Elastance of the chest wall is determined by the stiffness and the integrity of its skeletal components and, strength and tone of the musculature.

$$E = \frac{\Delta P}{\Delta V}$$

where E = Elastance, P = Pressure, and V = Volume. Elastic recoil refers to the rapidity and force (pressure) with which a substance returns to its original state when the deforming stress is removed. Compliance, which is the inverse of elastance, refers to the distensibility or stretch-ability of a substance when subjected to a deforming stress.

$$C = \frac{1}{E} = \frac{\Delta V}{\Delta P}$$

Specific compliance Since small lungs will have a smaller volume change when subjected to the same amount of pressure change, compliance is sometimes corrected to lung volume (usually functional residual capacity) to more accurately describe the structural properties of tissues. As a matter of convenience, compliance is sometimes corrected for weight or height of the patient to reflect the effect of size of the lung. This is referred to as specific compliance.

During normal spontaneous breathing, lungs and chest wall recoil in different directions during tidal respiration. Recoil pressures of the lungs and the chest wall refer to their respective pressures generated (in opposite directions) as they tend to return to their passive volume at 0 cm H_2O atmospheric pressure. Chest wall tends to recoil to a higher volume during tidal respiration while the lungs tend to recoil towards the lowest volume.

Although lungs and chest wall have their individual elastance and compliance properties, they need to be considered together since they are connected by pleural space which transmits recoil forces generated by one to the other. Individual elastic recoil pressures of chest wall and lung are represented schematically at various lung volumes corresponding to a percentage of total lung capacity (Fig. 1.4). At any

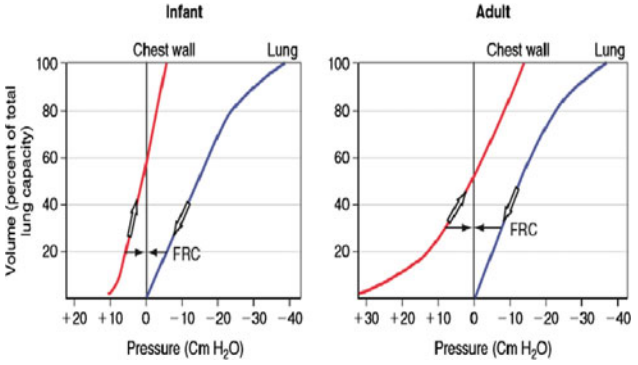


Fig. 1.4 Interaction between chest wall and lung recoil pressures in infants compared to adults. The lower recoil pressure of the chest wall in infants favors a lower FRC in infants. **FRC** – Functional Residual Capacity (Reprinted with permission from Sarnaik AP, Heidemann S and Clark JA, *Nelson Textbook of Pediatrics*, 20th Edition, Kliegman, St. Geme et al. Editors, Elsevier 2016)

volume, both the lung and the chest wall recoil to their passive volumes where their recoil pressure will be 0 cm H_2O. When corrected for volume, an infant's lungs exhibit remarkably similar elastance (and compliance) for an equivalent % change in volume compared to a healthy adult. The major difference between a neonate and an adult lies in the elastance of the chest wall. Neonatal chest wall has much less elastic recoil compared to an adult chest wall. It generates/requires less pressure (i.e. is more compliant) for a given change in volume. It can be easily understood that the elastic recoil pressure generated by the lung will bring about a greater change in the neonatal chest wall compared to the adult chest wall. The amount of air left in the lung at the end of tidal exhalation is referred to the functional residual capacity (FRC) which serves an important function as a reservoir for gas exchange during exhalation. At FRC the recoil pressures of the chest wall and the lungs are equal and opposite. FRC is also therefore termed as the "rest volume" which is achieved by equal and opposite recoil forces of the lung and the chest wall with no energy expenditure. The actual FRC determined in a spontaneously breathing neonate is considerably higher than what can be expected on the sole basis of respective lung and chest wall recoils. It is closer to what is observed in older children and adults corrected for lung volume. This is because (1) a neonate holds its chest wall in inspiratory position at the end of expiration by sustained tonic activity of the diaphragm and intercostal muscles, (2) increased respiratory rate (decreased time for exhalation) does not allow for complete lung deflation and (3) higher closing capacity which exceeds the volume at which tidal ventilation is occurring.

There are several implications of the lung-chest wall interaction in infants compared to older children. In newborns, the chest wall compliance is 3 to 6 times greater than the lung compliance. By 1 year of age, the chest wall elastance increases sufficiently to maintain FRC at a higher level solely on the basis the respective elastic recoils of the lung and the thoracic cage. In younger infants however, a marked decrease in FRC can occur in certain states: (1) conditions where inspiratory muscle tone is decreased, the chest wall becomes increasingly compliant such as during REM sleep, or with neuromuscular diseases (e.g. myopathies, neuropathies), use of sedatives/anesthesia and muscle relaxants, and CNS depression; (2) increased lung elastance (decreased lung compliance) such as with ARDS, pneumonia, pulmonary edema; and (3) extrathoracic airway obstruction which worsens during inspiration necessitating higher negative intrapleural pressure. In all these instances, the increasingly deformable chest wall retracts inward to a greater extent with a loss of FRC at end expiration and impeding air entry during inspiration. Under general anesthesia, because of the relaxed chest wall muscles, FRC declines by 10–25% in healthy adults, 35–45% in 6–18 year olds and greater than 50% in younger children. Application of PEEP is necessary in such instances to prevent atelectasis and hypoxemia.

1.5.1 Static and Dynamic Compliance

The pressure needed to overcome elastic recoil and move air, is measured once pressure has equilibrated and airflow has stopped. When compliance (ΔV/ΔP) is measured in this manner it is termed static compliance (C_{STAT}). Additional pressure is necessary to overcome resistance when air is flowing. The effect of resistance on compliance can be demonstrated using a pressure–volume plot (Fig. 1.5). The static relationship between pressure and volume is represented by the line A. At any given change in pressure, a corresponding change in volume is achieved, once airflow has stopped. The additional pressure necessary to overcome resistance is represented by the curves B and C when flow is occurring. During air flow, the same change in pressure results in less change in volume depending on the amount of resistance.

The ΔV/ΔP relationship during flow is termed dynamic compliance (C_{DYN}). The C_{DYN} for curve C is lower than for curve B because of increased resistance. Therefore, the difference between C_{DYN} and C_{STAT} represents the degree of resistance. Clinically, the difference between static and dynamic compliances can be measured during mechanical ventilation. When patients are receiving a set tidal volume using constant flow (volume control mode), the difference between peak

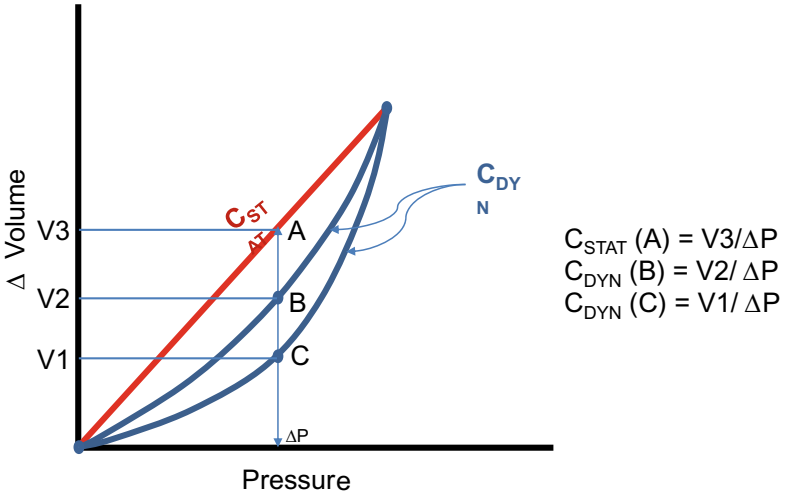


Fig. 1.5 Inspiratory pressure–volume (PV) curves. The red line (A) represents PV relationship during no-flow (static) state. The blue lines (B and C) represent PV curves while flow (dynamic) is occurring. Static compliance (C_{STAT}) and dynamic compliance (C_{DYN}) are calculated at a given ΔP. C_{STAT} (A) > C_{DYN} (B) > C_{DYN} (C)

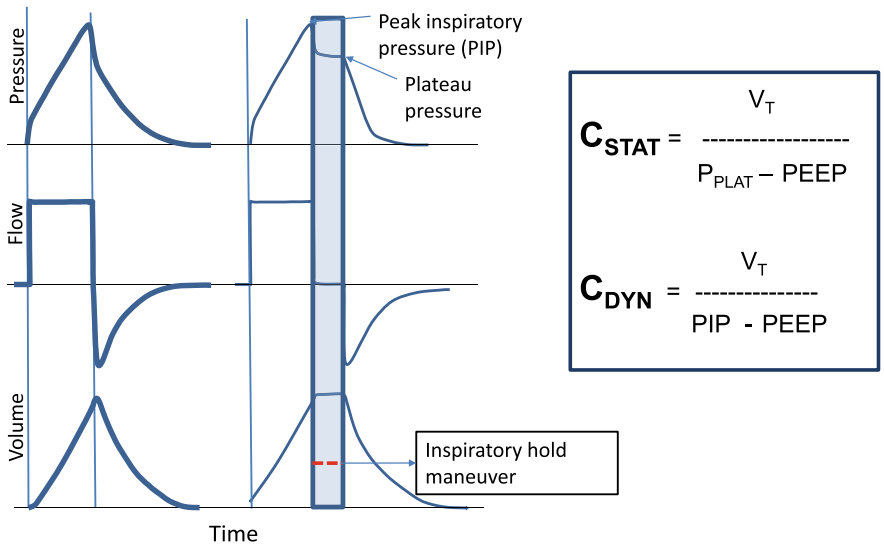


Fig. 1.6 Time relationships are shown for pressure, flow and volume in a volume-controlled ventilation with constant flow. Inspiratory hold is applied after peak pressure (PIP) is reached to allow pressure equilibration to occur between P_{AW} and P_{ALV} resulting in a plateau pressure (P_{PLAT}) which is lower than PIP as resistive forces are overcome

pressure and the plateau pressure obtained using an inspiratory hold maneuver can give an estimate of the airflow resistance (Fig. 1.6).

C_{DYN} can be calculated as the tidal volume divided by the difference between the peak inspiratory pressure and the end-expiratory pressure (Fig. 1.6). C_{STAT} is calculated as the tidal volume divided by the difference between the plateau pressure and end-expiratory pressure (Fig. 1.6). During volume controlled ventilation, P_{PLAT} is always lower than PIP and therefore C_{STAT} is always lower than C_{DYN}, and the degree of difference is dependent of the degree of airway obstruction.

1.5.2 Frequency Dependence of Compliance

Dynamic compliance (C_{DYN}) takes into account both the resistance (when flow is maximum) and the compliance (when flow is zero) of the respiratory system. Unlike C_{STAT}, which is relatively constant and a reflection of structural properties of the lung, C_{DYN} takes into account the flow resistive properties of the airways as well as the structural properties. Since the pressure required is a product of flow and resistance, an increase in either the flow or the resistance will require a greater pressure when considering the dynamic compliance. An increase in respiratory frequency will decrease the amount of time provided for inflation and deflation to

occur and necessitate an increase in flow. The resultant increase in flow resistive property will require a greater pressure to deliver the tidal volume and thus a decrease in C_{DYN}. This is termed frequency dependence of compliance; C_{DYN} decreases with increase in respiratory frequency. In diseases of increased resistance (prolonged time constant), C_{DYN} decreases markedly as respiratory frequency is increased.

1.6 Resistance

For air to flow across the airways, some force (pressure) is required to overcome opposing forces (resistance) such as inertia and friction. In the context of air movement from the mouth into the alveoli, the responsible pressure is termed the transrespiratory pressure gradient ($P_{AW} - P_{ALV}$). In a spontaneously breathing patient the pressure at mouth or proximal airway is same as the atmospheric pressure whereas with mechanically ventilated patient the proximal airway pressure is pressure at the patient-machine interface. Air flows from a higher pressure to a lower pressure both during inspiration and expiration. When the pressures at two ends are equal, flow ceases. By the same token, when airway is occluded stopping the flow, pressure is presumed to have equilibrated after a period of time. These airway occlusion techniques are utilized at various phases of respiration to estimate alveolar pressure measured at the proximal airway which is readily available for pressure measurement. Resistance is calculated as transrespiratory pressure gradient required generating a given amount of flow (volume per time) and expressed conventionally as cm H_2O/L/sec. Two of the important determinants of resistance to airflow are: (a) airway diameter and (b) nature of the flow, laminar or turbulent.

Laminar versus Turbulent flow: When gas molecules travel in a straight direction, the flow is referred to as being laminar. At higher velocities (distance/time), such as would occur when same amount of flow (volume/time) is pushed through a narrowed airway, the movement of gas molecules becomes chaotic thus resulting in turbulence.

Laminar flow **Turbulent flow**

Whether the flow is laminar or turbulent depends upon density, viscosity and velocity of gas and the diameter of the airway. When airflow is laminar, resistance is governed by Poiseuille's law:

$$R = \frac{8nL}{\pi r^4}$$

R is resistance, l is length, η is viscosity, and r is the radius. The practical implication of pressure-flow relationship is that airway resistance is inversely proportional to its radius raised to the 4th power. If the airway lumen is decreased in half (1/2), the resistance increases 16-fold. A flow change from laminar to turbulent occurs when Reynold's number exceeds 2000. Reynold's number (Re) is a dimensionless entity represented as:

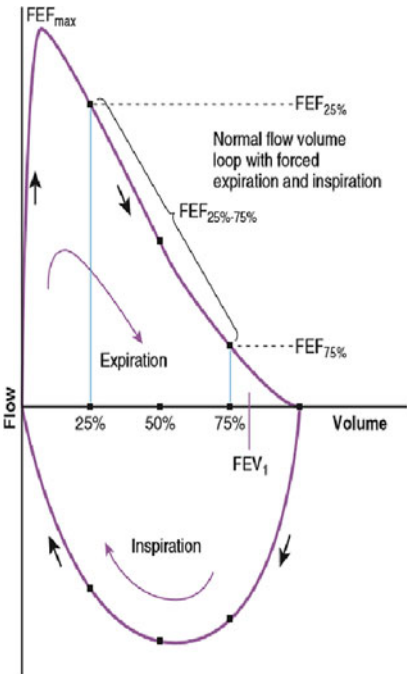
$$\text{Re} = (\text{Diameter} \times \text{Velocity} \times \text{Density}) \div \text{Viscosity}$$

Resistance to turbulent flow is much greater than to laminar flow. In clinical situations, the most effective way of decreasing the Reynold's number is to decrease the density of inspired gas. For this purpose, helium is used to replace nitrogen to promote laminar flow. Helium is about 7 times less dense and slightly more (1.1X) viscous than nitrogen. For helium to be effective for this purpose, it needs to be present to a sufficient degree. It is generally believed that for Helium–oxygen mixture (Heliox) to effectively reduce resistance, at least 50–60% of the mixture needs to be comprised of helium. This means patients requiring more than 50% oxygen may not benefit from Heliox.

Resistance to airflow is different during inspiration and expiration. In normal circumstance, expiratory resistance is higher than inspiratory resistance. This is because the intrathoracic airways expand during inspiration as they are subjected to more negative pleural pressure from the outside in spontaneous breathing and positive pressure from the inside in mechanical ventilation. In intrathoracic airway obstruction (asthma, bronchiolitis, vascular ring etc.) this discrepancy increases as expiratory resistance increases exponentially due to airway compression by excessively positive pleural pressure. In extrathoracic airway obstruction (subglottic stenosis, vocal cord paralysis etc.), the inspiratory resistance can exceed the expiratory resistance as the airway outside the thorax collapses because of excessively increased intraluminal negative pressure.

1.7 Flow/Volume Relationships

Flow/volume relationships curves are clinically useful tools for demonstrating the effect changes in pulmonary mechanics have on volumes and gas flow (Fig. 1.7). These curves are generated with spirometry machines and can be used in both the outpatient setting well as the bedside. Typically, a maximal inspiration is followed by maximal forced exhalation, generating a flow/volume loop. Forced vital capacity (FVC) is the total volume exhaled during this maneuver. Forced expiratory volume during the first second of exhalation is termed FEV1. Decreases in FVC and FEV1 result from decreased lung compliance, increased airway resistance and decreased



A normal flow/volume loop. Performed by maximal inspiration followed by maximal forced exhalation to completion.
FEF$_{MAX}$ is synonymous with peak expiratory flow rate (PEFR) and represents the maximum expiratory flow rate during exhalation. This occurs soon after the start of forced exhalation.
FEF$_{25\%-75\%}$ represents the mean flow between 25% and 75% total expiratory volume. Also referred to as mid maximum expiratory flow rate (MMEFR).
FEV$_1$ is the volume of expired gas in the first second of expiration.

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Fig. 1.7 Flow-volume loop created by maximum inhalation followed by forced maximum exhalation

respiratory muscle strength. The maximum flow occurs in the first phase of forced exhalation and is referred to as FEF_{max} (also referred to as peak flow). It is effort dependent but also a marker of airway obstruction. The volume exhaled between 25 and 75% of the expiratory volume (FEF 25–75%) is relatively effort independent. The reason for this is that a higher intrathoracic pressure results in narrowing of intrathoracic airway preventing further increase in flow.

A decrease in $FEF_{25–75\%}$ is indicative of intrathoracic airway obstruction such as in asthma (Fig. 1.8). The shape of the expiratory curve gives clues to disease pathophysiology, as in obstructive disease, where the mid-expiratory curve may be increasingly concave. In restrictive lung and chest wall diseases, all the lung volumes and capacities are decreased without appreciable decreases in flow rates.

Intrapulmonary Airway Obstruction (A): note the decrease in **FEF_{MAX}** and **$FEF_{25\%-75\%}$** and the concavity of the mid-expiratory curve.

Restrictive disorder (B): Note the more vertical and narrow shape. **FVC** is decreased but flow rates are relatively less affected.

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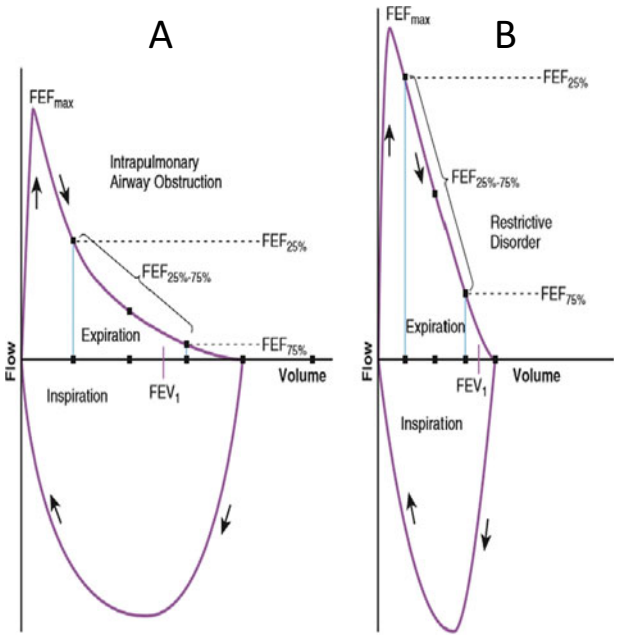

Fig. 1.8 Flow volume loops in intrapulmonary obstructive lung disease (**a**) and restrictive disorders (**b**)

1.8 Equal Pressure Point (EPP)

When intrathoracic airway pressure is increased during exhalation (either by forced voluntary exhalation or in intrathoracic airway obstruction), the pressure must dissipate along the airway to reach the reference atmospheric pressure of 0 cm H_2O or the positive end expiratory pressure set in the mechanical device. The site at which the intraluminal pressure equals pleural pressure is termed the equal pressure point (EPP).

The significance of EPP is that the pressure in the intrathoracic airway proximal to this point (downstream) is less than intrapleural pressure and therefore subject to collapse depending on the magnitude of the pressure difference and stiffness/softness of its wall. With intrathoracic airway obstruction, the EPP is shifted distally towards the alveolus, causing a greater length of airway to collapse above (Fig. 1.9). Softer infantile airways are more susceptible to change in diameter when subjected to increased pressure. Marked dynamic changes in intrathoracic airway diameter during inspiration and exhalation in young infants above EPP are often termed collapsible trachea. Tracheal collapse is often a result of airway obstruction and even contributes to its severity but it is rarely the primary abnormality.

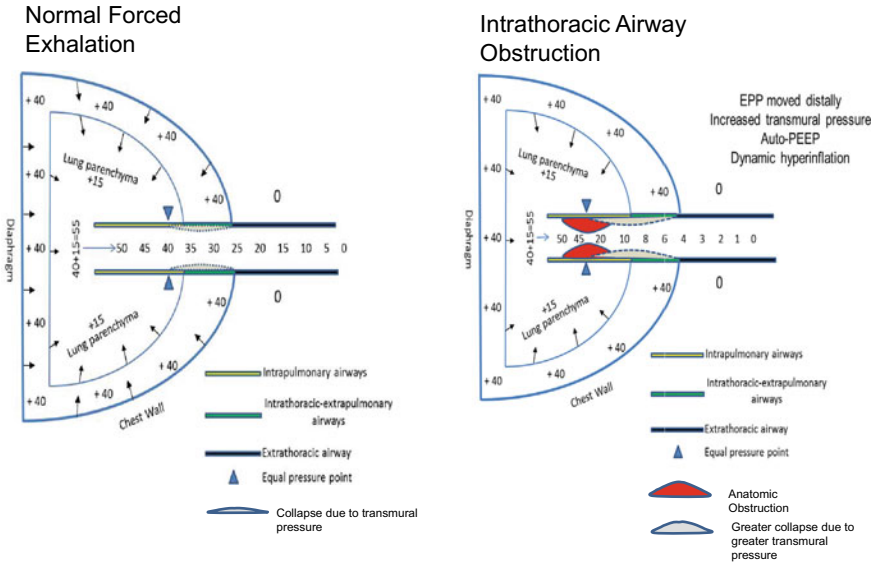

Fig. 1.9 Equal pressure point (EPP) is a site at which intrathoracic and intraluminal pressures during exhalation are equal. Proximal to EPP (downstream), intrathoracic pressure exceeds intraluminal pressure resulting in airway collapse. EPP is displaced distally in intrathoracic airway obstruction, and the magnitude of difference between intrathoracic and intraluminal pressures is increased resulting in greater airway collapse

Suggested Readings

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