Respiratory Mechanics

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Objectives

Introduction

Muscles of Respiration

Inspiratory and Expiratory forces

Compliance, Elastance, and Surface Tension

Airway Resistance

Flow-Volume loop

Time Constant

Work of Breathing

Conclusion

References

Introduction

Respiration is a vital process that enables gas exchange between the environment and the body. For this exchange to occur efficiently, it is essential to understand respiratory mechanics, which describe the physical principles that regulate the flow of air in and out of the lungs.

Respiratory mechanics are based on the interaction between pressure, gas flow, and lung volumes. Understanding it requires knowledge of the anatomy of the respiratory system, which consists of the airway, lungs, and thoracic wall. The latter includes the rib cage and the diaphragm. Between the lungs and the thoracic wall lies the intrapleural space, where pleural fluid lubricates the pleural surfaces and maintains lung adherence to the thoracic wall.

There are opposing static forces in the respiratory system: the lungs tend to collapse inward, while the thoracic wall exerts an outward force. These forces can be represented as two springs in parallel: the thoracic wall, compressed below its resting volume, and the lungs, stretched above their resting volume.

Muscles of Respiration

The respiratory muscles generate the force necessary for ventilation. The diaphragm is the main respiratory muscle, responsible for approximately 70% of the tidal volume under normal conditions. Its contraction increases thoracic volume, allowing air to enter. Other inspiratory muscles, such as the external intercostals, parasternal muscles, and scalenes, also assist in thoracic expansion.

The sternocleidomastoids act during situations of high ventilatory demand, such as exercise, elevating the first rib and the sternum to facilitate greater air intake. During expiration, the internal intercostals and abdominal muscles (external oblique, internal oblique, transversus abdominis, and rectus abdominis) aid in expelling air. Under normal conditions, expiration is passive and depends on the elastic recoil of the lungs, but in high-demand situations, it becomes an active process that requires energy expenditure (figure 1).

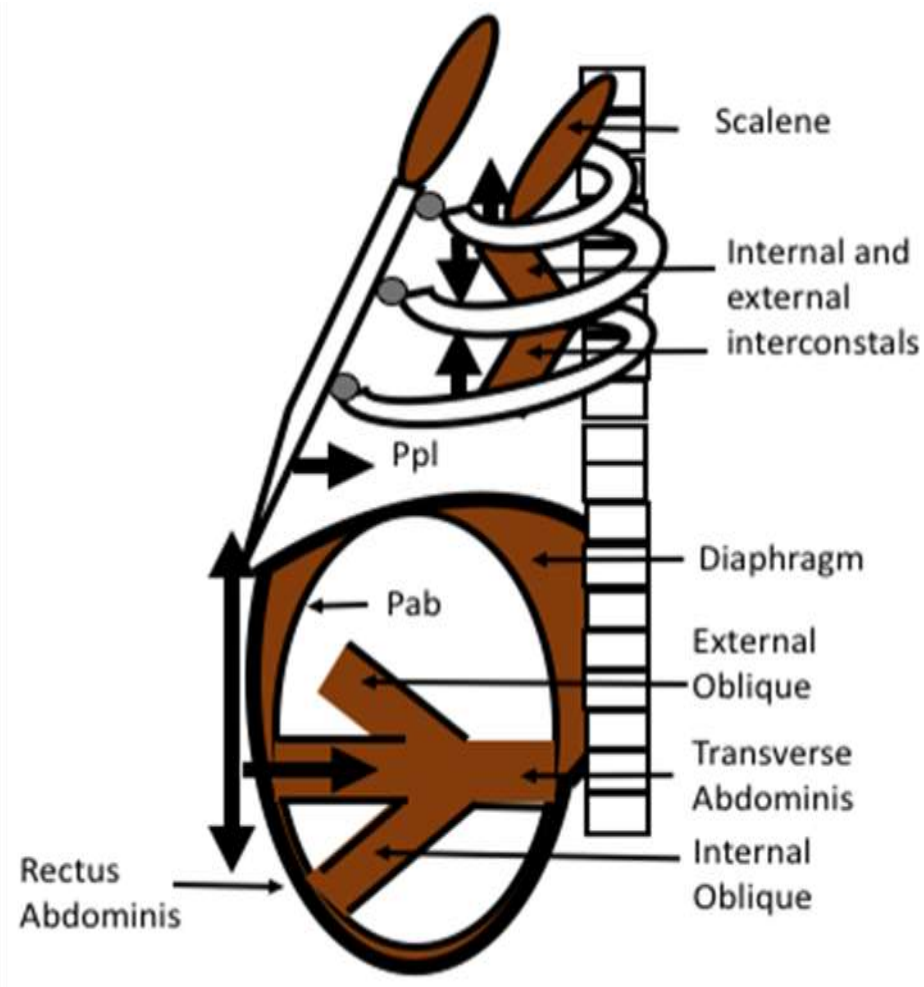

Figure 1: Respiratory muscles. Diagrammatic representation of the inspiratory and expiratory muscles; arrows indicate the direction of action. Pab, abdominal pressure; Ppl, intrapleural pressure. Figure created by the author based on reference 1.

Diaphragm Function

The diaphragm can be visualized as a piston with two sections: the costal portion, which has a zone of apposition with the ribs, and the crural portion, which forms the dome. Its stimulation by the phrenic nerve causes contraction and downward displacement, expanding the thoracic cavity and increasing intra-abdominal pressure.

In addition to its role in respiration, the diaphragm contributes to the generation of positive intra-abdominal pressure, facilitating functions such as urination, defecation, and childbirth. Its functional assessment is performed by measuring transdiaphragmatic pressure (Pdi), calculated by subtracting intrapleural pressure from abdominal pressure

$$Pdi = Pab - Ppl$$

Inspiratory and Expiratory force (MIP/MEP)

Inspiratory and expiratory strength reflects the ability of the respiratory muscles to generate the pressure gradients required to overcome the elastic and resistive properties of the respiratory system, thereby enabling effective ventilation.

Inspiratory strength is primarily generated by diaphragmatic contraction, supported by the external intercostal and accessory muscles, which reduces pleural pressure and facilitates air entry. Expiratory strength, under resting conditions, relies on the lung's elastic recoil; however, during active expiration—such as during exercise or in obstructive diseases, the abdominal and internal intercostal muscles contribute to forceful air expulsion.

Clinical assessment of respiratory muscle strength includes:

- Maximum Inspiratory Pressure (MIP or Pimax): Measured during a maximal inspiratory effort against occlusion from residual volume. It primarily reflects diaphragmatic strength, with normal values > -75 cmH_2O in men and > -50 cmH_2O in women.

- Maximum Expiratory Pressure (MEP or Pemax): Assessed during maximal expiratory effort from total lung capacity, reflecting the strength of expiratory muscles. Normal values are >100 cmH_2O in men and > 80 cmH_2O in women.

These measurements are essential in critically ill patients to detect respiratory muscle dysfunction, guide weaning strategies from mechanical ventilation, and assess the risk of respiratory complications.

Compliance, Elastance, and Surface Tension

Compliance is the ability of lung tissue to expand and is defined as the change in volume in relation to a change in airway pressure. Simply put, compliance describes how easily (or with how much difficulty) an elastic structure stretches, expands, or deforms (strain). This property is related to the elastic recoil of lung tissue, which reflects a tissue's tendency to return to its original shape after being deformed. Elastance, expressed as the pressure required for a volume change (ΔP/ΔV), is the reciprocal of compliance (ΔV/ΔP) and describes the lung's opposition to expansion.

This phenomenon can be observed in the pressure-volume curve of an air-filled lung during inspiration and expiration (figure 2). The curve shows a relatively steep slope at low to mid-range pressures, followed by flattening near the upper limit of pressure values. The reduction in compliance or the increase in elastic recoil at higher volumes is due to elastin and collagen fibers within the lung tissue. This greater elastic recoil at high volumes is an intrinsic property of the lung and represents one of the forces contributing to its inward pull.

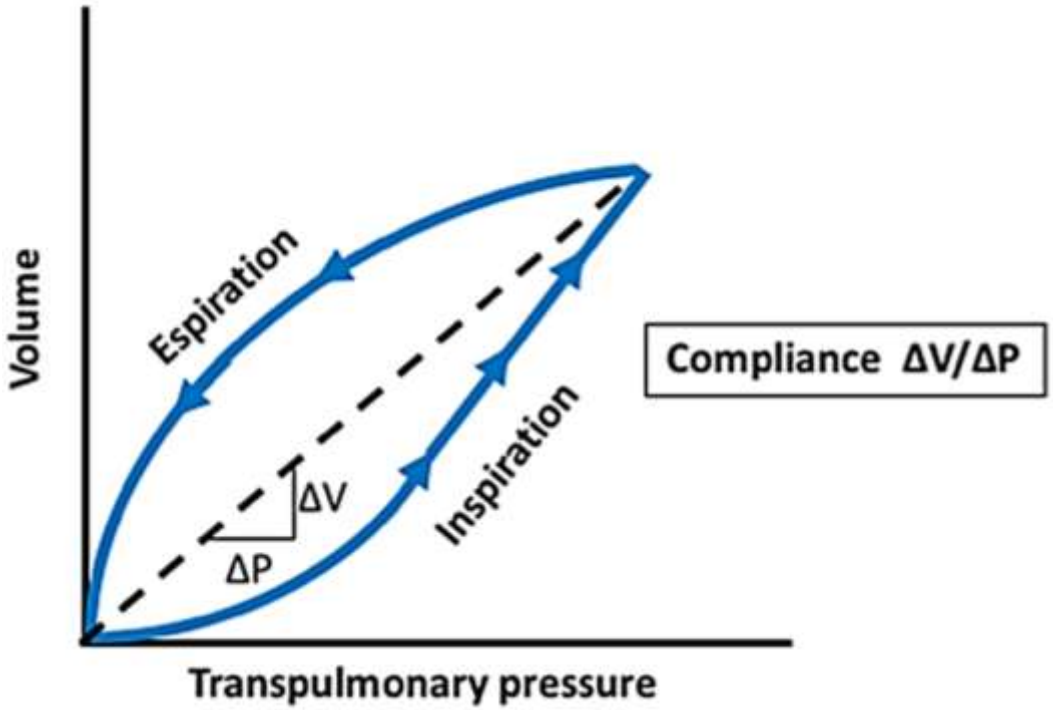

Figure 2: Compliance diagram in a healthy individual. This diagram shows the compliance of the lungs alone. Figure created by the author based on concepts from reference 2.

It is important to remember that the respiratory system is composed of two components arranged in series: the lung (L) and the chest wall (W). Variations in either component produce changes in the total compliance of the respiratory system. The interaction between the lung and the chest wall is fundamental

for maintaining respiratory mechanics and efficient gas exchange. The compliance of the entire respiratory system depends on the relationship between the increase in volume and the change in airway pressure. Thus, lung compliance reflects the ease with which the pulmonary parenchyma expands in response to a change in transpulmonary pressure (alveolar pressure – pleural pressure), while chest wall compliance indicates the ability of the thoracic cage to adapt to changes in pleural pressure.

Under physiological conditions, both lung and chest wall compliance contribute differently to the total compliance of the system. For example, in restrictive pulmonary diseases such as pulmonary fibrosis, reduced lung compliance increases the work of breathing due to greater stiffness of the parenchyma. In contrast, musculoskeletal or neuromuscular disorders affecting the chest wall may reduce its compliance, limiting lung expansion and compromising ventilation.

Additionally, surface tension at the alveolar air-liquid interface plays an important role in lung compliance. This tension tends to collapse the alveoli, which reduces lung compliance. Type II pneumocytes produce surfactant, a phospholipid that lowers surface tension and stabilizes the alveoli, preventing their collapse and facilitating homogeneous ventilation. Laplace's law describes the alveolar collapsing pressure as a function of surface tension and alveolar radius. In the absence of surfactant, smaller alveoli would collapse, transferring their volume to larger ones. Surfactant regulates this tension, preventing both alveolar collapse and overdistension.

The balance between the inward pull of the lungs and the outward pull of the chest wall is best illustrated through the pressure-volume curves of the relaxed lung, chest wall, and the entire respiratory system (figure 3).

These curves show the transmural pressure across each of the three structures at volumes ranging from residual volume (RV) to total lung capacity (TLC). The curve representing the lung shows that even at residual volume, the transpulmonary pressure (i.e., alveolar pressure minus pleural pressure) is positive, indicating that the lungs maintain an internal elastic recoil. In contrast, the chest wall curve shows that transthoracic pressure remains negative (i.e., pleural pressure is lower than atmospheric pressure) until volumes approach TLC.

The curve representing the respiratory system is a sum of the curves for the lung and chest wall and is generated by calculating the sum of transpulmonary pressure and transthoracic pressure at various volumes. The point where the respiratory system curve crosses the y-axis, known as functional residual

capacity (FRC), is where the system reaches its equilibrium volume—where the inward recoil of the lungs is balanced by the outward recoil of the chest wall.

At higher volumes, there is a positive intrapulmonary (alveolar) pressure, creating a gradient that drives air outward, returning the system toward FRC. The opposite occurs at volumes below FRC.

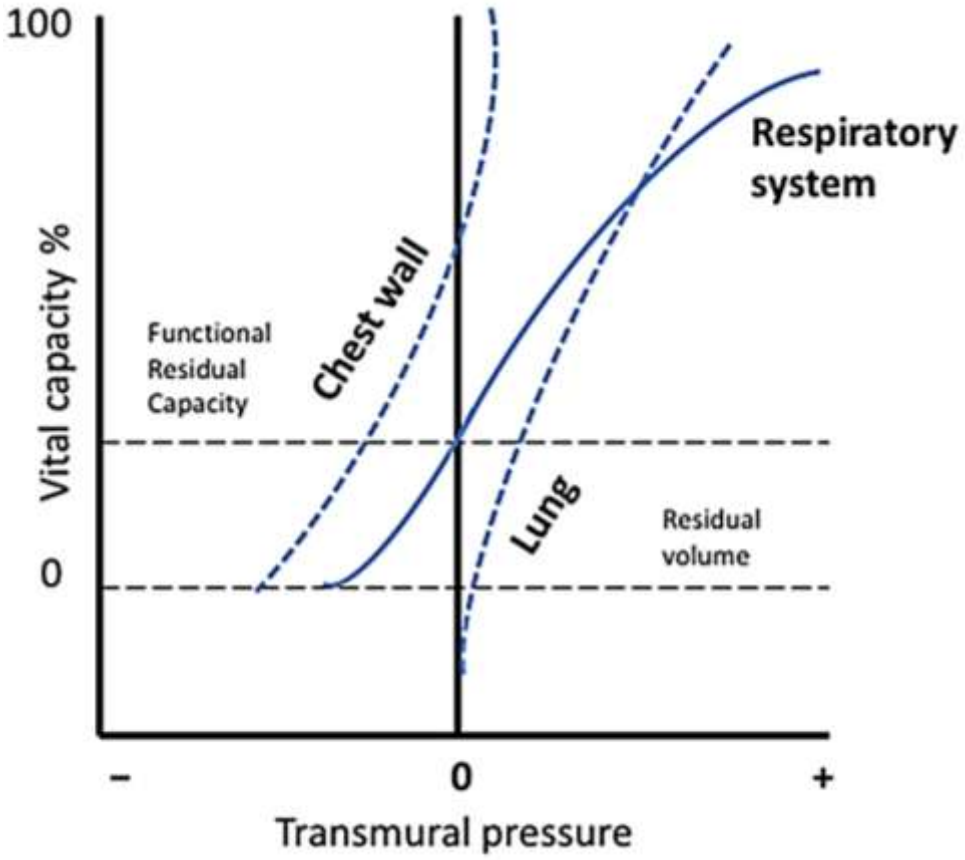

Figure 3: Relationship between transmural pressure and lung capacity for the respiratory system, chest wall, and lungs. The curves represent the individual elastic properties of the lungs (dashed line), chest wall (dashed line), and the combined respiratory system (solid line). The point where the elastic forces intersect (where transmural pressure is zero) corresponds to the functional residual capacity (FRC). Residual volume is also indicated for reference. Figure created by the author based on reference 2.

Measuring compliance in non-intubated and intubated patients

Compliance assessment is essential in the management of critically ill patients, both ventilated and non-ventilated, as it provides insight into respiratory mechanics and allows for individualized adjustment of ventilatory strategies.

For example, a decrease in compliance indicates a stiffer respiratory system, as observed in acute respiratory distress syndrome (ARDS), pulmonary fibrosis, or alveolar edema.

Measuring compliance in intubated patients

In intubated patients on mechanical ventilation, compliance measurement is more precise due to controlled tidal volumes and airway pressures.
Two types are distinguished:

- **Static Compliance (Cstat):** Measured under no-flow conditions, typically during an end-inspiratory pause maneuver.

  It is calculated as:

$$Cstat = \frac{Vt}{Pplat - PEEP}$$

  Where V_t is the tidal volume, Pplat is the plateau pressure measured after a 0.5–2 second inspiratory pause, and PEEP is the positive end-expiratory pressure.

  Cstat reflects the combined compliance of the lung parenchyma and chest wall.

- **Dynamic Compliance (Cdyn):** Measured during continuous inspiratory flow.

  It is calculated as:

$$Cdyn = \frac{Vt}{PIP - PEEP}$$

  Where PIP is the peak inspiratory pressure.

  Cdyn is influenced by both the elastic properties and airway resistance.

Measurement in Non-Intubated Patients

In non-intubated patients, direct measurement of compliance is more challenging, requiring simultaneous, noninvasive quantification of volume and pressure. Strategies include:

- Spontaneous breathing efforts with spirometry:

Portable spirometers measure inspiratory and expiratory volumes. Mouth pressure measurements (e.g., P0.1) can estimate initial respiratory effort and approximate respiratory compliance.

- Body Plethysmography:

Considered the gold standard for noninvasive lung function testing. It allows measurement of total lung capacity (TLC) and airway resistance, providing indirect calculations of lung compliance.

- Esophageal Manometry:

The use of esophageal balloon catheters enables estimation of pleural pressure, offering a precise assessment of transpulmonary pressure and lung compliance, similar to measurements in intubated patients.

Clinical Relevance

Compliance measurement facilitates optimal adjustment of tidal volume in lung-protective ventilation, early identification of conditions like ARDS, individualized PEEP titration to improve oxygenation while avoiding overdistention, and monitoring of pulmonary recovery or restrictive disease progression.
In critically ill patients, it is essential to standardize measurement conditions: controlled tidal volume, constant flow, and proper patient–ventilator synchrony. Compliance interpretation must also consider factors such as obesity, ascites, or chest wall abnormalities, which can alter measured pressures.

Airway Resistance

Gas flow through the airways follows Poiseuille's law, which states that airway radius significantly influences flow resistance ($R = 8\eta l / \pi r^4$). This law applies when the flow is laminar, meaning at low velocities, and it establishes that resistance is directly proportional to the gas viscosity (η) and the length of the airway (l), and inversely proportional to the fourth power of the radius (r^4).

Factors such as lung volume, bronchial smooth muscle tone, and the viscosity of the inhaled gas affect airway resistance. Under normal conditions, the total cross-sectional area of the tracheobronchial tree increases exponentially with each successive generation.

The airway radius is the primary determinant of resistance, as neither the length of the airway nor the gas viscosity varies significantly under initial physiological conditions.

Measuring resistance in non-intubated and intubated patients

Airway resistance (Raw) is defined as the ratio between the pressure gradient across the respiratory system and the resulting airflow. Its measurement is fundamental in the management of critically ill patients, allowing for the identification of ventilatory disturbances and optimization of respiratory support strategies.

$$Raw = \Delta P / \dot{V}$$

Where: ΔP is the pressure difference (in cmH_2O), $\dot{V}$ is the airflow (in L/s)

Normal airway resistance is low (< 2 $cmH_2O/L/s$); however, it may increase significantly in conditions such as asthma, chronic obstructive pulmonary disease (COPD), or prolonged intubation.

Measurement in intubated patients

In intubated patients under mechanical ventilation, airway resistance can be measured directly using ventilator-derived pressure–time and volume–time curves.

- Inspiratory Pause Method:
  - During volume-controlled ventilation, an end-inspiratory pause is performed to measure plateau pressure (Pplat) and peak inspiratory pressure (PIP).
  - Resistance is calculated as:

$$Raw = PIP - Pplat / \dot{V}$$

  - PIP reflects the pressure needed to overcome both airway resistance and lung elastance, while Pplat isolates the elastic component.
- Considerations:
  - A constant inspiratory flow (square waveform) is required for valid measurement.
  - Factors such as secretions, endotracheal tube size, or suctioning techniques can alter measurements.

Measurement in non-intubated patients

In non-intubated patients, resistance is measured indirectly through specialized techniques:

- Body Plethysmography:
  - The gold standard for resistance measurement in spontaneously breathing individuals.
  - The patient breathes within a sealed chamber, allowing for thoracic volume changes and mouth pressures to be recorded during specific maneuvers.
  - Total airway resistance (Raw) is calculated using Boyle's Law, correlating pressure and volume changes.

- Impulse Oscillometry (IOS):
  - A noninvasive technique that evaluates airway resistance through sound waves superimposed on normal breathing.
  - It allows for assessment of central and peripheral resistance across multiple frequencies.

- Voluntary Respiratory Effort:
  - Techniques such as maximal inspiratory and expiratory pressure (MIP, MEP) measurements can help infer increased airway resistance, particularly in cases of flow limitation.

Clinical Relevance

Measuring respiratory resistance has multiple applications in critical care. It aids in the differential diagnosis of dynamic obstructions (e.g., bronchospasm vs. atelectasis), guides ventilator settings such as inspiratory time and trigger sensitivity, monitors disease progression in acute and chronic obstructive diseases, and helps assess the risk of weaning failure due to elevated resistive load.

High resistance may require tailored interventions such as prolonged expiratory time, increased inspiratory flow, or adjustments in bronchodilator strategies.

Factors such as endotracheal tube diameter, airway secretions, measurement technique (e.g., constant flow), and patient cooperation in noninvasive settings can significantly influence resistance values.

Flow-Volume loop

The Flow-Volume Loop graphically represents the relationship between airflow and lung volume during a complete respiratory cycle. It is a key tool for assessing ventilatory mechanics, particularly airway patency and overall pulmonary function.

During the maneuver, the patient performs a forced expiration from total lung capacity (TLC) to residual volume (RV), followed by a forced inspiration. The resulting curve enables identification of characteristic patterns of obstruction or restriction.

A normal loop displays a sharp expiratory peak followed by a progressive decline in flow, while the inspiratory phase is more symmetric. Morphologic alterations in the loop allow for diagnostic suggestions such as:

- Variable intrathoracic obstruction: Flattening of the expiratory phase
- Variable extrathoracic obstruction: Flattening of the inspiratory phase
- Fixed obstruction: Global flattening of both phases
- Lower airway obstruction (e.g., asthma, COPD): Increased expiratory concavity
- Restrictive disease (e.g., pulmonary fibrosis): Globally reduced loop with preserved shape

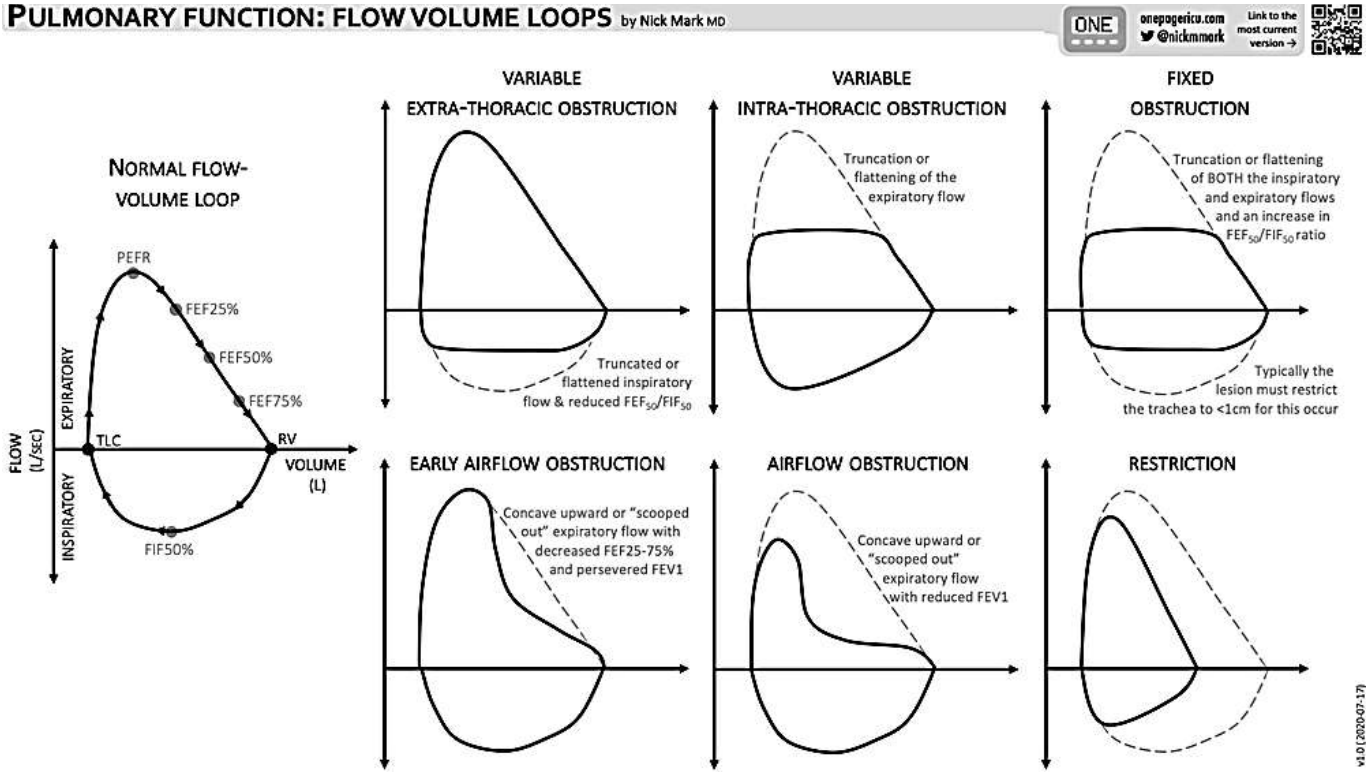

Figure 4: Different Flow-Volume loop shapes and their etiologies. From one page ICU with permission.

In mechanically ventilated patients, interpretation of Flow-Volume Loops helps detect phenomena such as air trapping (auto-PEEP) or pulmonary overdistension.

Proper execution of the maneuver requires active patient cooperation and adherence to technical quality criteria established by ATS/ERS guidelines.

Time Constant

A fundamental aspect to consider in respiratory mechanics is the time constant (τ) of the respiratory system. To illustrate this concept, we can use a simple model composed of a balloon and a straw. Imagine inflating the balloon and covering the end of the straw with your thumb. Once you remove your thumb, the air begins to escape.

The speed at which the balloon deflates depends on two main factors: its compliance and the resistance of the straw. If the balloon has high elastic recoil (low compliance) or the straw is wide (low resistance), the air exits quickly and the balloon empties in a short time. In contrast, if the balloon has low elastic recoil (high compliance) or the straw is narrow (high resistance), the flow is slower, and emptying takes longer.

This same principle applies to the respiratory system during passive exhalation. The speed of exhalation is determined by the product of respiratory system compliance (C) and airway resistance (Raw), which is known as the time constant (τ), measured in seconds ($\tau = C \times Raw$).

During passive exhalation, the volume of gas remaining in the lungs (V) at a specific time (t) is determined by the initial tidal volume (Vi) and the time constant. While memorizing the equation isn't necessary, it is useful to recall that after 1-, 2-, and 3-time constants, approximately 37%, 14%, and 5% of the tidal volume, respectively, remain in the lungs. Since expiratory flow also decreases exponentially, there is a similar relationship between the initial (peak) flow and the flow at any given moment during exhalation (figure 4).

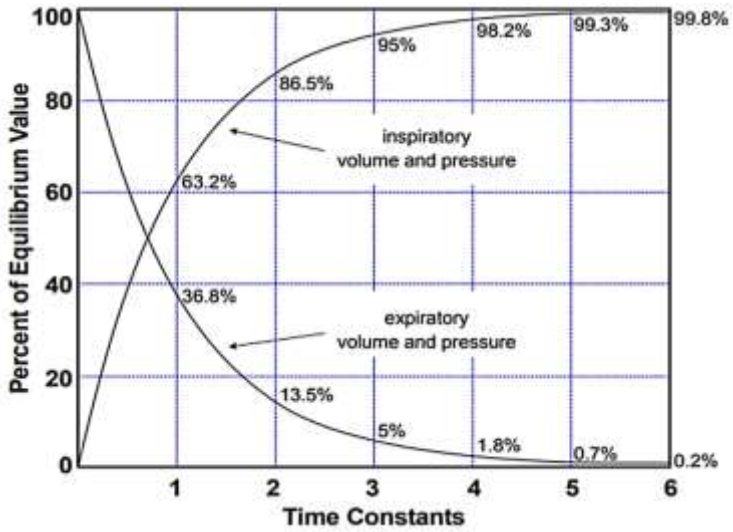

Figure 5; Distribution of time constants in the lung. The graph illustrates how time constants (the product of airway resistance and lung compliance) vary across different pulmonary units. Units with higher resistance or increased compliance exhibit prolonged time constants, resulting in slower filling and emptying. This heterogeneity impacts gas distribution during mechanical ventilation.

Work of Breathing (WOB)

In the field of physics, the concept of work is defined as the result of applying a force to an object, causing displacement in the same direction as the force. Mathematically, this relationship is expressed as the product of force and the distance traveled ($W = F \times d$). In the context of the respiratory system, the work of breathing (WOB) is determined as the product of the integral of pressure and volume, which is equivalent to the area under the curve in a pressure–volume diagram:

$$WOB = \int \text{pressure} \times \text{volume}$$

From a physiological perspective, the work of breathing represents the effort required to overcome the load imposed by the respiratory system during ventilation. This work can be performed entirely by the respiratory muscles during spontaneous breathing, exclusively by a mechanical ventilator in controlled ventilation, or shared between both in assisted ventilation.

To better understand the different components of the work of breathing, it is essential to analyze the equation of motion of alveolar gas. This equation describes how, at every moment in the respiratory cycle, the airway pressure (Paw) is determined by the interaction of three main factors:

$$Paw = Ers \times V + Raw \times \dot{V} + PEEPtot$$

Where: Paw: airway pressure, Ers: elastance of the respiratory system, V: lung volume, Raw: airway resistance, $\dot{V}$: airflow through the airways, PEEPtot: total positive end-expiratory pressure

From this equation, it can be deduced that the work of breathing must overcome three main components (figure 5):

1. Elastic component: Related to the elastance of the lung parenchyma and chest wall. It represents the effort required to expand the lungs and thoracic cage against their natural tendency to recoil.
2. Resistive component: Determined by the airway resistance to airflow. The greater the resistance, the more effort is required to maintain effective ventilation.
3. Component associated with total positive end-expiratory pressure (PEEPtot): In pathological conditions, air trapping may occur, forcing the respiratory muscles to generate additional pressure to overcome this load before a new inspiration can begin.

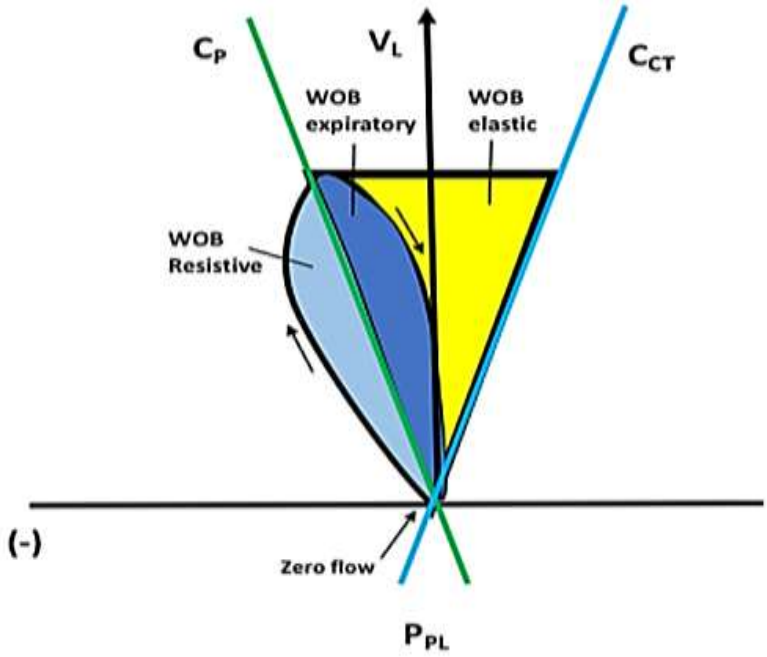

Figure 6: Campbell's diagram. Work of breathing (WOB) measured by the esophageal pressure: resistive WOB, elastic WOB, WOB related to active expiration (WOB expiratory). CCT chest wall compliance; CP, compliance pulmonar, VL, lung volume; PPL, pleural pressure. Figure created by the author based on concepts from Steinberg E. Elaboración e interpretación del diagrama de Cnampbell. Argentinian Journal of Respiratory & Physical Therapy (2023).

Although the concept of respiratory work is based on the classical definition of mechanical work, there is an important nuance in respiratory physiology. Traditionally, it is assumed that for mechanical work to occur, there must be a displacement associated with the application of a force. However, in respiration, both in healthy subjects and in those with respiratory pathology, there is an isometric component in which the respiratory muscles can generate effort without producing a change in volume. This effort, although it does not translate into measurable mechanical work in terms of pressure-volume, involves significant energy consumption by the respiratory muscles and contributes to the sensation of dyspnea in pulmonary diseases.

Therefore, in the clinical assessment of respiratory work, it is not only important to measure changes in pressure and volume but also to consider the metabolic cost of respiratory effort, especially in conditions where the load placed on the respiratory muscles can lead to fatigue and eventual ventilatory failure.

Conclusion

Understanding respiratory mechanics is essential for the proper management of patients with respiratory pathologies and for optimizing mechanical ventilation. The interaction between anatomical components, such as respiratory muscles, pulmonary compliance, and the properties of the chest wall, defines the efficiency of gas exchange. Additionally, factors such as airway resistance, surface tension, and time constant play critical roles in normal ventilation and respiratory disorders.

The work of breathing, in particular, is a key aspect in clinical evaluation, as it reflects the effort required to overcome the resistances of the respiratory system. This work is not only mechanical but also metabolically costly, especially in pathological conditions. Integrating the principles of respiratory mechanics into clinical practice allows for a better understanding of respiratory alterations, facilitating appropriate therapeutic intervention and improving outcomes in patients with respiratory failure or chronic lung diseases.

In summary, a comprehensive and multidimensional approach to respiratory mechanics is crucial for clinical decision-making, from optimizing ventilation to preventing respiratory muscle fatigue in critical situations.

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