### o\_two INSIGHTS Your Simple Path to Medical Findings

We simplify major findings into brief, straightforward summaries to keep you in the loop.

## Introcuction

Tidal volume is explained as the volume of air inhaled into or exhaled from the lungs during each respiratory cycle. A healthy adult male typically has a tidal volume of around 500 mL, while a healthy female's is about 400 mL. This parameter is crucial for ensuring efficient ventilation and gas exchange. As oxygen from the atmosphere is inhaled, it enters the lungs and diffuses across the alveolar-capillary interface to reach the arterial blood. This process is vital for supplying oxygen to the body while removing carbon dioxide produced by the body's metabolic processes, which is expelled during exhalation. This cycle of inhalation and exhalation ensures balanced levels of oxygen and carbon dioxide in the blood, highlighting the importance of tidal volume as an essential clinical parameter for maintaining proper respiratory function.



The Physiology of Tidal Volume Across Organ Systems.

# Insights into Tidal Volume

The primary responsibility of the lungs is to deliver a tidal volume that ensures sufficient ventilation. This process requires intricate coordination among the respiratory center in the brain, the respiratory muscles, and the respiratory pacemaker located in the brainstem, which controls the rate and depth of breathing. Central and peripheral chemoreceptors monitor blood levels of oxygen and carbon dioxide and adjust the pacemaker's activity accordingly. In turn, the diaphragm and other inspiratory muscles modify the tidal volume and the respiratory rate to maintain optimal levels of these gases in the blood. For instance, during physical activity, the body's demand for oxygen increases, leading to an accumulation of carbon dioxide. This prompts an increase in respiratory rate and tidal volume to accommodate the elevated demand.

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# Optimizing Tidal Volume in Mechanical Ventilation.

In mechanical ventilation, tidal volume delivery should be large enough to maintain adequate ventilation but small enough to prevent lung trauma. Initially, ventilating patients with 10 mL/kg of tidal volumes or more of the ideal body weight was used to prevent hypoxemia, avoid airway closure, and increase functional residual capacity. However, these high tidal volumes can cause volutrauma due to alveolar overdistension and the frequent reopening of collapsed alveoli, initiating an inflammatory cascade. This cascade is characterized by increased lung permeability, alteration of surfactant, pulmonary edema, and cytokine release that damages lung tissue. The adverse effects of volutrauma were first described in 1974 by Webb and Tierney, who demonstrated pulmonary edema in rats due to high inflation pressures. Additionally, such large tidal volumes may lead to barotrauma, where the rupture of alveoli allows air to accumulate in the pleural cavity or mediastinum.



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## Insights into Tidal Volume

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## Role of the Diaphragm in the Breathing Cycle.

Air is inhaled into and exhaled from the lungs due to the movement of the diaphragm and the chest wall. As the primary muscle of inspiration, the diaphragm plays a pivotal role in controlling tidal volumes. When the diaphragm contracts, it causes the thoracic cavity to expand vertically, decreasing intrapleural pressure from approximately -5 cm H2O to -8 cm H2O. This reduction in pressure allows the lungs to expand as the pleural connection to the chest wall creates negative intrapleural pressure, pulling the lungs outward and increasing lung volume. According to Boyle's law, the increase in lung volume results in decreased pressure inside the alveoli, creating sub-atmospheric intra-alveolar pressure that draws air into the lungs until the pressure stabilizes, typically delivering a tidal volume of about 500 mL. Exhalation primarily occurs as a passive process initiated when the diaphragm relaxes. This relaxation causes the thoracic cavity to decrease in size, which moves the rib cage closer to the lungs and increases intrapleural pressure back to approximately -5 cm H2O. As the lung volume decreases, the internal pressure exceeds atmospheric pressure, forcing air out of the lungs according to the pressure difference and returning them to their resting state.

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### Dynamic and Static Lung Volumes and Spirometry.

Lung volumes are classified as dynamic and static. The airflow rate influences Dynamic lung volumes, making them critical for diagnosing airflow-related pathologies. Static lung volumes remain unaffected by airflow velocity, providing a consistent measure of lung capacity. Changes in these volumes can indicate a range of lung pathologies, making pulmonary function tests a vital tool for diagnostic assessment. These tests, including spirometry, measure how air flows in and out of the lungs, measuring various lung volumes essential for diagnosing respiratory diseases.

Spirometry is valuable for diagnosing restrictive and obstructive pulmonary diseases. It involves the patient taking a normal breath, followed by a full inhalation and a maximal forced exhalation, before taking another normal breath. This process helps measure several lung volumes, including tidal volume, which is a static lung volume. In a typical healthy adult, the tidal volume is about 500 mL per breath, of which only 350 mL reaches the respiratory zone as dead space measures around 150 mL.

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## Respiratory Tract and Dead Space

The respiratory tract is divided into conducting airways, which range from the nose to the terminal bronchioles, and gas-exchanging airways from the respiratory bronchioles to the alveoli. Dead space refers to those areas of the lungs filled with air that do not engage in gas exchange. This includes anatomical dead space, which indicates air in the conducting airways, and alveolar dead space, referring to alveoli filled with air but not involved in gas exchange, though it contributes less to dead space. Anatomical and alveolar dead spaces form the physiological dead space, indicating the total volume of non-participated air in gas exchange within the lungs.



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### How Tidal Volume Influences Gas Exchange and Ventilation

Tidal volume impacts both minute ventilation and alveolar ventilation. Minute ventilation is the total air volume entering the lungs per minute, calculated by multiplying the respiratory rate by tidal volume. Alveolar ventilation indicates the air volume reaching the respiratory zone per minute.

• Minute Ventilation = Respiratory Rate X Tidal Volume.

#### • Alveolar Ventilation = Respiratory Rate X (Tidal Volume - Dead Space)

Alveolar ventilation, which deducts dead space, reflects the actual ventilation. Typically, tidal volume and respiratory rate contribute equally to minute ventilation. However, increasing tidal volume is generally more efficient for enhancing alveolar ventilation than increasing the respiratory rate. However, this concept is relevant for patients with hypercapnia. These patients often need to take slower, deeper breaths to reduce dead space ventilation effectively and optimize CO2 removal by increasing the tidal volume that reaches the respiratory zone.

# Insights into Tidal Volume

## 8 Monitoring Plateau pressure.

In mechanical ventilation, monitoring plateau pressure provides a reliable indicator of barotrauma risk. This pressure, exerted on the alveoli and small airways, is influenced by lung compliance and tidal volume. When lung compliance decreases, plateau pressure rises, increasing the risk of barotrauma. Therefore, if plateau pressure increases, it is often necessary to reduce tidal volume to lower the risk of alveolar rupture. The current standard practice is to use tidal volumes of 6 mL/kg of predicted body weight due to the ongoing research in lung-protective ventilation strategies.



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# Insights into Tidal Volume

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### Dynamic and Static Lung Volumes and Spirometry.

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## REFERENCES

The insights presented on this topic are derived from the following article. For a comprehensive review and more detailed information on this topic, please refer to the original text:

https://www.ncbi.nlm.nih.gov/books/NBK482502/

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