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Modeling the lung: A rheological question – Part 1

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Abstract

Rheology is a branch of physics that studies the deformation and flow of matter and provides the mathematical framework for analyzing pulmonary mechanics. Rheological theory views the lung as a viscoelastic body that exhibits both elastic and viscous properties, implying that its response to mechanical forces depends not only on the magnitude of the force but also on the speed and frequency with which it is applied. It provides an understanding of how elasticity, resistance, and viscosity contribute to respiratory mechanics and ventilator-induced lung injury

Monitoring respiratory mechanics in the Intensive Care Unit (ICU) has traditionally been based on the respiratory system equation of motion, which assumes first-order or linear behavior, characterized by constant static resistance and elastance (or its reciprocal, compliance). However, the physiology of the lung parenchyma is far from being a purely elastic system.

This publication represents the first installment of three about lung modeling.

Keywords: Lung modelling, Hysteresis, Viscoelasticity, Rheology

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Introduction

Rheology is a branch of physics that studies the deformation and flow of matter and provides the mathematical framework for analyzing pulmonary mechanics. Rheological theory views the lung as a viscoelastic body that exhibits both elastic and viscous properties, implying that its response to mechanical forces depends not only on the magnitude of the force but also on the speed and frequency with which it is applied. It provides an understanding of how elasticity, resistance, and viscosity contribute to respiratory mechanics and ventilator-induced lung injury (VILI). [1]

The lung parenchyma, composed of an intricate matrix of collagen fibers, elastin, and connective tissue, functions as a biological material exposed to repetitive mechanical loading. During ventilation, this tissue is subjected to cyclical stress and strain, reflecting its complex biomechanical response to respiratory forces. [2]

Monitoring respiratory mechanics in the Intensive Care Unit (ICU) has traditionally been based on the respiratory system equation of motion, which assumes first-order or linear behavior, characterized by constant static resistance and elastance (or its reciprocal, compliance). However, the physiology of the lung parenchyma is far from being a purely elastic system. [3]

Lung viscoelasticity refers to its ability to exhibit both elastic and viscous characteristics, which directly influences respiratory mechanics and gas exchange. This duality is fundamental to understanding phenomena such as pressure-volume (P-V) hysteresis and the dependence of pulmonary elastance and resistance on frequency. [4] Viscoelasticity in the lung involves the storage of energy (elasticity) and the dissipation of energy (viscosity) during the respiratory cycle. The macroscopic and clinical manifestation of this energy dissipation is lung hysteresis. [2]

Mathematical modeling of these properties has evolved from integer-order approaches, which use analogous electrical components such as springs and dashpots, [5] up to fractional order models, which offer a more accurate representation of the dynamic response of biological systems and system memory. [6]

We aim to provide a comprehensive review of this topic, which, despite its limited discussion in routine medical practice, is essential for the effective management of patients undergoing mechanical ventilation. Given the extensive scope of the subject, we have organized our analysis into three parts, with this publication representing the first installment.

Methods

A short but comprehensive review of the literature was carried out on the internet, looking for terms such as rheology, stress, strain and lung viscoelasticity. The authors selected those papers they believed to be more relevant.

Objectives

- To review the fundamental rheological concepts as applied to lung mechanics, including elasticity, viscosity, and viscoelasticity.
- To explain how viscoelastic properties of lung tissue influence respiratory mechanics and the risk of VILI.
- To describe key viscoelastic phenomena in the lung, such as creep, stress relaxation, and hysteresis, and their clinical relevance.
- To highlight the importance of understanding stress, strain, and strain rate in designing safer mechanical ventilation strategies and preventing irreversible lung damage.

Review

A material is considered viscoelastic when its response to an applied stress or strain exhibits characteristics intermediate between an ideal elastic solid (whose deformation depends solely on stress) and an ideal viscous fluid (whose stress depends on the strain rate). The lung classifies as a viscoelastic material. This dynamic behavior implies that part of the energy applied during inspiration is stored (elastic component), while another part is dissipated (viscous component) in the form of heat or through structural reorganization of the tissue. [7] The macroscopic and clinical manifestation of this energy dissipation is pulmonary hysteresis, the difference between the P-V curve during inspiration and expiration, a phenomenon that has been studied in the context of PEEP and recruitability in patients with acute respiratory distress syndrome (ARDS). [8]

This phenomenon is fundamental in biological tissues such as the lung, where the stress-strain relationship plays a pivotal role in the development of atelectrauma. Clinically, pulmonary viscoelasticity becomes evident when inspiratory flow is abruptly interrupted (inspiratory pause), resulting in a rapid initial decline in airway pressure attributable to airway resistance followed by a gradual, exponential decrease that reflects stress relaxation within the viscoelastic lung tissue. [7]

The lung parenchyma, consisting of a complex matrix of collagen fibers, elastin, and connective tissue, exhibits the behavior of a biological material subjected to cyclic mechanical loading. During ventilation, this tissue experiences stress (force per unit area, analogous to pressure)

and strain (relative deformation, analogous to normalized volume), reflecting its intricate biomechanical response to respiratory forces. [8] Unlike an ideally elastic material, which responds instantaneously to stress, or a viscous material, which flows upon application of stress, viscoelastic materials exhibit a dependence of strain on time under load. [9]

We will define some of the properties of viscoelastic materials applied to the lungs.

1. Fundamental components

Elasticity: ability of lung tissue and alveolar structure to return to their original shape after inspiration. [7] It describes the ability of lung tissue to store mechanical energy reversibly. It is characterized by:

- Instantaneous response: deformation occurs simultaneously with the application of stress.
- Complete reversibility: upon removal of stress, the tissue completely returns to its initial configuration.
- Energy storage: applied energy is conserved in the form of elastic energy. [10]

In the lung, elasticity is primarily derived from elastin fibers (up to 200% elongation) and collagen fibers (dominant at larger deformations), as well as from surfactant-mediated alveolar surface tension. [10]

Viscosity: internal friction between lung tissue and moving air, generating resistance to volume change. [7] Viscous components dissipate energy in the form of heat during cyclic deformation. [11] This energy dissipation contributes significantly to the hysteresis observed in the P-V curves, reflecting the energy loss during the respiratory cycle. [12]
Viscosity represents the tissue's resistance to strain rates and is associated with irreversible energy dissipation. Its characteristics include: [13]

- Strain rate dependence: stress is proportional to deformation in relation to time.
- Energy dissipation: mechanical energy is converted into heat.
- Delayed response: there is a time lag between stress and strain. [14]

The distinction between the instantaneous elastic response and the time-dependent viscous response is fundamental for understanding lung mechanics and for evaluating various respiratory pathologies. Both lung resistance and elastance are frequency-dependent, underscoring the nonlinear nature of viscoelasticity, a property that is more pronounced in the parenchyma than in the chest wall. [15]

In the lung parenchyma, viscosity originates from the relative movement between fibers, the flow of interstitial fluid through the extracellular matrix, and the continuous breaking and reformation of weak molecular bonds. [16]

Plasticity: permanent deformation that occurs once stress exceeds the elastic limit of the material. In the lung, this may be associated with tissue remodeling or the breakage of fibers that define structural rigidity. [3] It can manifest in:

- Residual deformation: the tissue does not completely return to its initial configuration.
- Irreversible flow: permanent reorganization of the extracellular matrix.
- Structural remodeling: particularly relevant in pathological processes such as fibrosis. [17]

2. Viscoelastic phenomena

Creep: progressive deformation of a material subjected to a constant load over time (Figure 1). If a constant force is applied to a viscoelastic tissue, it will continue to deform over time, even if the force remains unchanged. In ARDS, the lung with altered matrix may show exaggerated creep, favoring overdistension. In sustained recruitment maneuvers, creep can be useful to slowly open units. This phenomenon is crucial in prolonged ventilation, since the application of constant pressure can result in a progressive increase in tissue volume or strain, with the potential risk of damage. [18]

The Burgers model is an attempt to capture this phenomenon of delayed creep. [13] The Burgers model is an attempt to capture this phenomenon of delayed creep, [13] but studies in cardiovascular tissues have shown that creep exhibits power law behavior. [19]

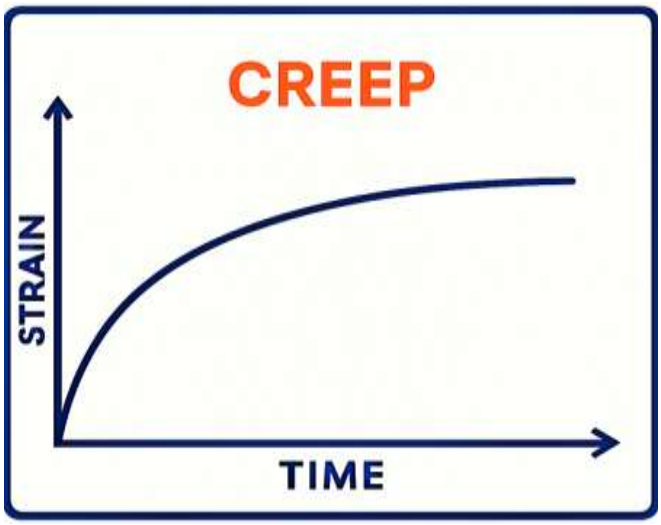

Figure 1: Creep. Progressive deformation (strain) over time under a constant load.

Stress relaxation: this is the reverse phenomenon. When a tissue is subjected to a constant strain, the force required to maintain that strain decreases over time (Figures 2 and 3). The tissue adapts and requires less pressure to remain stretched, due to the microstructural reorganization and redistribution of stress in the viscoelastic tissue. In mechanical ventilation with an inspiratory pause, pressure may drop during the pause: a sign of relaxation. It can be used to estimate the viscoelastic properties of the lung. In inflamed or fibrosed tissues, relaxation may be impaired, so pressure remains elevated. [7]

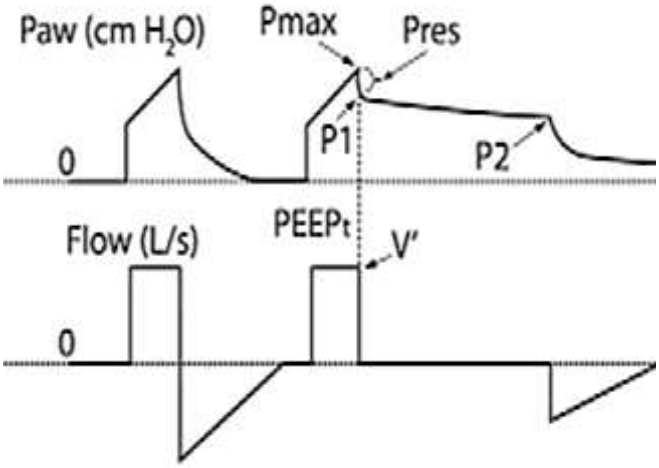

Figure 2: Airway pressure-time curve (top) and flow-time curve (bottom) in volume-controlled mode with constant inspiratory flow using an inspiratory holding maneuver. Pmax: peak inspiratory pressure, Pres: is the airway resistance (Pmax – P1), P1: is the initial drop of pressure at the beginning of the hold maneuver, P2: is the true plateau pressure at the end of the maneuver. The difference between P1 and P2 is due to the stress-relaxation phenomena. From reference 20

Figure 3: Stress relaxation. The force (stress) required over time to maintain a constant deformation (strain) progressively decreases.

In pig lungs, experimental evidence has demonstrated that this relaxation process follows a power-law behavior, rather than the exponential decay predicted by classical models. 13

Hysteresis: defined as the difference in the P-V loop between the inspiratory and expiratory phases (Figure 4), is a clear manifestation of viscoelasticity, wherein energy is dissipated during the loading and unloading cycle due to the viscous properties of lung tissue. [7,13] The greater the energy dissipation, the more pronounced the viscoelastic behavior, which is manifested as an increased difference between the inspiratory and expiratory branches of the pressure-volume (P-V) curve. [21] Quantification of this dissipated energy is vital, as it is the portion of mechanical work that is not recovered and is therefore considered a direct contributor to biotrauma and atelectrauma in the setting of VILI. [13] It describes the dependence of the state of the system not only on current conditions, but also on its previous history. [22]

In the context of ARDS, P-V loop hysteresis provides crucial information about:

- Lung recruitability: a larger area of hysteresis correlates with a greater recruitable volume. [23]
- Tissue viscoelastic properties: the shape of the loop reflects relaxation and creep phenomena. [24]
- Regional heterogeneity: hysteresis increases with the non-uniform distribution of mechanical properties. [7]

Experimentally, it has been shown that hysteresis does not depend solely on simple elastic or viscous properties, but on the complex interaction between surface tension, alveolar recruitment and viscoelastic properties of the extracellular matrix. [24]

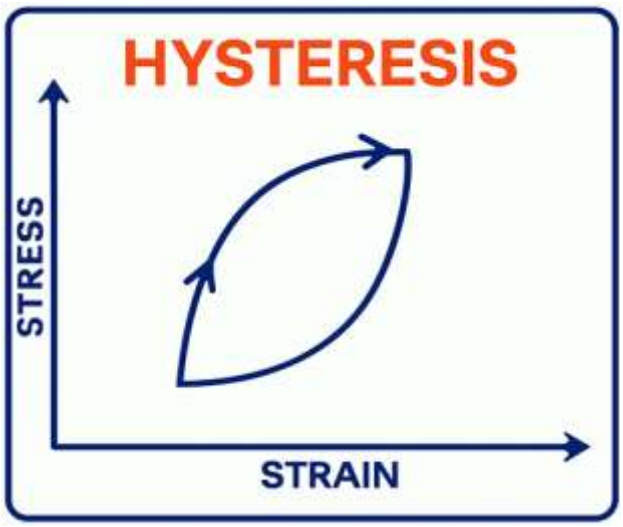

Figure 4: Hysteresis. At the same volume, inspiratory pressure exceeds expiratory pressure, resulting in the formation of a loop in the P-V curve, the area of which represents energy dissipated by the tissue.

3. Other related concepts

Resilience: maximum amount of energy that the lung can store elastically, without suffering permanent structural damage. It defines a safety threshold energy for the lung up to which lung deformation is reversible and the tissue can return to its original state without microfractures or injury. If the applied energy exceeds the resilience threshold, irreversible plastic deformations occur, resulting in microfractures within the lung parenchyma, inflammation, and progression of VILI. Injury occurs when ventilation pushes the lung beyond its "elastic region" of the stress-strain curve and enters the territory of "plastic deformation," where structural damage is permanent. [1] Rheological theory predicts that there will be no VILI if the amount of applied energy remains within this safe zone. [25]

Anisotropy: the mechanical response of the lung varies depending on the direction of the applied force due to the arrangement of its collagen and elastin fibers. This variability varies throughout the lung tissue and increases with higher inflation volumes. [26]

Stress (σ): internal force per unit of area applied to a material (Figures 5 and 6). It represents the pressure or tension generated in lung tissue or airways in response to ventilation or distension. It is not measured directly, but we can infer it through transpulmonary pressure i.e. (the pressure difference between the alveoli and the pleura, (PTP) during inspiration and expiration. Due to the difficulty of routinely measuring PTP, driving pressure (DP) is used as a clinical surrogate. PTP represents 73%–85% of DP in patients with ARDS. [1,7,26]

The principle of materials engineering states that increased stress increases the likelihood of creating internal fractures in the material. Similarly, high DP in the lung, even when the tidal volume considered safe, can generate excessive stress capable of causing microfractures in lung tissue. [25]

Strain (ε): fractional change in the shape or volume of a material in response to stress compared to its original state, due to an external load (Figures 5 and 6). In the lung, it refers to how much the alveolar tissue stretches relative to its resting volume. Clinically, global strain relates to the ratio of tidal volume (VT) to the lung's functional residual capacity (FRC). An alveolus that doubles its volume during inspiration experiences significant deformation and contributes to the risk of VILI. Stretching as little as 20% can initiate the production of inflammatory mediators and reactive oxygen species. [1,27]

A critical finding is the heterogeneity of strain in the lung. Animal studies suggest that central and basal regions exhibit greater deformation, while in humans, this is greatest at the apices. Even a minimal increase in strain above the tolerance limit can alter the tissue and its nonlinear behavior. The risk of VILI increases when strain approaches a value of 1.5, which marks the elastic limit of lung tissue. [1,26]

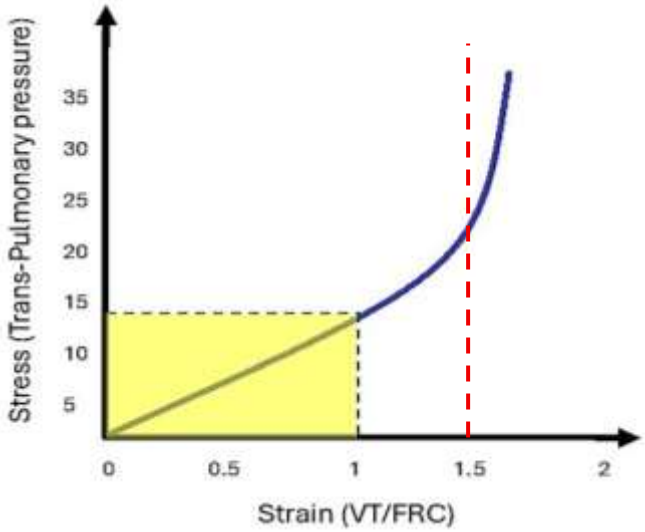

Figure 5: Stress – Strain relationship. The yellow shaded area represents the safe zone of ventilation.

The interaction between collagen and elastin fibers determines the stress-strain curve, which is initially soft and then becomes stiffer as more collagen fibers are recruited (27,28). Healthy lungs have a nearly linear stress-strain relationship, meaning that as stress increases, deformation increases proportionally. However, in ARDS, the lungs are heterogeneous, with stiff areas and normal areas. [1]

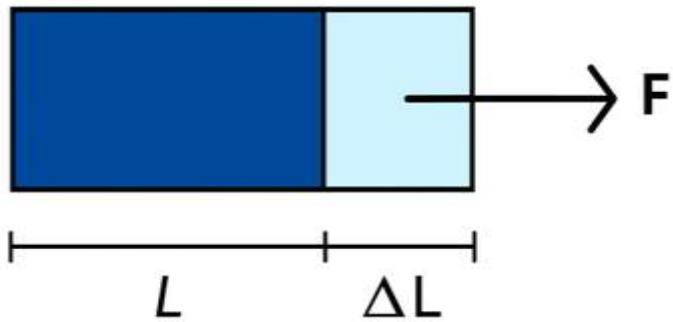

Figure 6: Stress and strain. F: Force = Stress = PTP, L: Length = Volume). Strain = ΔL/L.

Shear stress: it is a type of tangential stress that acts parallel to the surface of a material, resulting in lateral deformation or “shearing” of the structure. This is different from normal stress (such as pressure) which acts perpendicularly. It is the stress that occurs when forces attempt to slide one layer of tissue relative to another in a parallel direction. [30]

In the lung, shear stress is particularly relevant in ARDS. Due to the heterogeneity of the lung: there are open alveolar units alongside collapsed ones. The movement of these two adjacent areas during ventilation (one expanding and the other not) generates shear stress at the interface, which can cause tearing and damage. Shear stress is linked to mechanotransduction, that is, how endothelial cells and other cell types detect and respond to these mechanical stresses. [9]

It is also related to atelectrauma: the damage that occurs due to the cyclical collapse and reopening of the alveoli during mechanical ventilation, especially when an adequate residual volume is not maintained (insufficient PEEP). During the repetitive cycle of alveolar collapse and reopening, the adjacent alveolar surfaces experience intense shear forces, generating elevated shear stress in these regions of abrupt re-expansion. This increase in shear stress contributes to the rupture of the alveolar membrane, local inflammation, and structural damage to the lung parenchyma, which characterize atelectrauma. The more opening-closing cycles, the greater the exposure to these tangential stresses, which worsens VILI. [9]

Strain rate: it is the speed at which the deformation (strain) occurs. That is, how quickly the lung is stretching. It corresponds to the gas flow divided by the FRC. It determines the dynamic response and resistance of the lung. A high inspiratory flow (which means a short inspiratory time) generates a high strain rate. This is more damaging to lung tissue, as it does not have time to adapt to the deformation. A high strain rate, even with a similar total strain, increases the risk of VILI, suggesting that the speed of energy delivery is a critical factor in the pathogenesis of injury. [1,7]

Although classical theories did not consider it as a central item, rheological theory assigns it a critical role. For a material that behaves like a liquid, a higher deformation rate (i.e., a rapid inspiratory flow) generates greater internal stress, which can be highly damaging. Because the viscoelastic lung exhibits both solid-like and liquid-like behavior, the total stress experienced is determined by the combined effects of pressure, volume, and flow velocity. Consequently, the same tidal volume can be more injurious if delivered rapidly rather than gradually and gently. [25]

Table 1: Integrated terminology: respiratory rheology and physiology.

Engineering material term	Rheological definition	Clinical/Physiological correlation	Application in lung-protective mechanical ventilation (LPMV)
Stress (σ)	Internal tension or force per unit area	Transpulmonary pressure (PTP)	Limit to prevent barotrauma/volutrauma
Strain (ϵ)	Relative volumetric deformation	Tidal volume (VT) normalized to EELV (functional lung)	Goal: maintain low VT (6 ml/kg ideal body weight)
Strain rate ($\dot{\epsilon}$)	Rate of change in deformation (deformation/time)	Flow ($\dot{V}$)	Control flow patterns (e.g., square vs. decelerating) to manage dynamic stress
Shear stress (τ)	Tangential stress or force parallel to a surface	Local forces at tissue interfaces (e.g., stress hotspots)	Minimize by optimizing PEEP to avoid cyclic opening/closing (atelectrauma)
Creep	Increase in deformation under constant stress	Delayed alveolar recruitment	Use prolonged inspiratory pauses to optimize EELV
Stress relaxation	Decrease in stress under constant deformation	Drop in plateau airway pressure during inspiratory pause	Determination of dynamic tissue viscosity and response time
Hysteresis	Deviation of the stress-strain curve during loading and unloading	Difference between inspiratory and expiratory P/V	Indicator of recruitability and energy dissipation

2. Rheological models

A viscoelastic model is a mathematical representation that describes the constitutive relationship between stress and strain of a biological material as a function of time. In the pulmonary context, this constitutive relationship must capture three fundamental aspects: [13]

- The instantaneous elastic response upon application of PTP.
- The viscous dissipation of energy during deformation.
- The time-dependent phenomena that characterize tissue structural reorganization. [10]

Early models of lung viscoelasticity were based on Mount's framework, which accounted for the inverse frequency dependence of both dynamic work and compliance observed in open-chest rats. [15] More complex models, such as the standard linear solid model or fractional order derivatives, have been developed to characterize this response, allowing a more reliable representation of the biomechanical behavior of the lung parenchyma and the prediction of stress relaxation. [4]

A thorough understanding of the underlying concepts is essential for elucidating the mechanisms and significance of the models employed in this context.

Spring: it represents elasticity (E), defined as the ability to resist deformation and store potential energy (Figure 7), and its behavior is governed by Hooke's Law. [31] Its constitutive equation involves the zeroth derivative (stress is proportional to strain). It is the behavior that allows a body to return to its original shape. If a spring is stretched and then released, it instantly returns to its original shape. In the lung, this property is referred to as elastance (the inverse of compliance). It pertains to the elastic components of lung tissue primarily elastin and collagen fibers which, when stretched, generate a recoil force that tends to collapse the lung. [32]

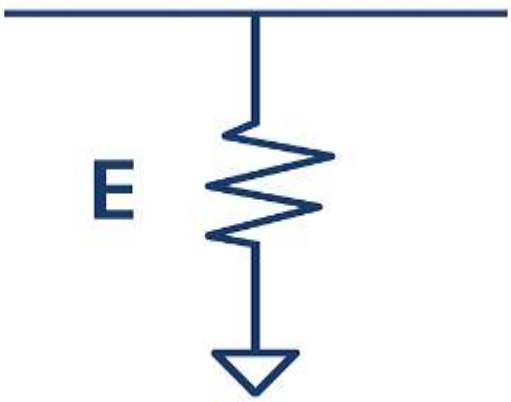

Figure 7: Spring. Represents elasticity (E).

Dashpot: it represents viscosity (η), encompassing resistance to airflow and energy dissipation in the form of heat (Figure 8). This resistance includes both airway resistance and the intrinsic viscous resistance of lung tissue. The dashpot obeys Newton's Law, with its constitutive equation involving the first-order derivative indicating that stress is proportional to the strain rate [31] It is the resistance to deformation that depends on the speed at which the force is applied. When a piston is driven into a cylinder filled with oil, the resistance encountered is directly proportional to the speed at which the piston is moved. In the context of mechanical ventilation, this principle is analogous to the relationship between airway resistance and the viscosity of lung tissue, where the rate of airflow significantly influences the mechanical load experienced by the respiratory system. [32]

The combination of these two elements gives rise to viscoelasticity, a material property characterized by both solid-like (elastic) and liquid-like (viscous) behavior. As previously discussed, the lung is fundamentally a viscoelastic organ. [32]

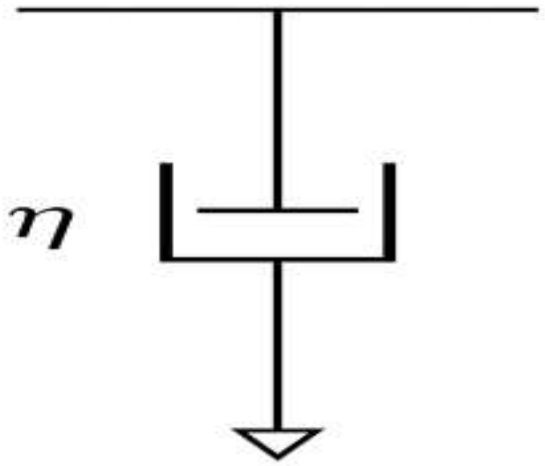

Figure 8: Dashpot. Represents viscosity (n)

Hooke's law: describes the elasticity of a solid where stress is directly proportional to strain, a behavior that is independent of the strain rate. It is analogous to an ideal mechanical spring. Purely elastic materials store and release all strain energy without loss. [32]

Newton's law: governs viscosity, where stress is proportional to the strain rate, and is represented by an ideal dashpot. Purely viscous fluids continuously dissipate mechanical energy in the form of heat. [32]

Young's modulus (lungs specific elastance): derived from Hooke's law. It is a constant that defines the linear relationship

between stress and strain. A lung with a high Young's modulus is stiffer (as in pulmonary fibrosis). A lung with a low Young's modulus is more distensible (as in emphysema). This is a simplistic approach that cannot capture the inherent complexity of biological tissues, which exhibit dynamic and nonlinear behavior on multiple time scales. In ARDS, this value is surprisingly constant in adults (13.5 ± 4.1 cmH_2O), regardless of the cause of ARDS, VT, or PEEP used. [1,32]

In our subsequent publications, we will present a comprehensive analysis of rheological models designed to characterize pulmonary viscoelasticity, beginning with integer-order approaches and progressing to fractional order frameworks.

Conclusions

Understanding lung ventilation from a rheological perspective provides a more comprehensive framework for interpreting respiratory mechanics and the risks associated with mechanical ventilation. The lung is a viscoelastic organ, exhibiting both elastic and viscous properties, which means its response to mechanical forces depends on the magnitude, speed, and frequency of those forces. This duality explains phenomena such as hysteresis, creep, and stress relaxation, all of which are crucial for understanding lung behavior during ventilation.

Traditional linear models are insufficient to capture the complex, nonlinear, and time-dependent behavior of lung tissue. Key concepts such as elasticity, viscosity, plasticity, resilience, anisotropy, stress, strain, and strain rate are essential for evaluating the mechanical limits of the lung and for designing safer ventilation strategies.

Ultimately, the application of rheological theory to lung mechanics emphasizes the importance of not only the amount of energy delivered to the lung but also the rate and manner in which it is applied. Staying within the elastic limits of lung tissue and minimizing excessive stress, strain, and strain rate are critical to preventing irreversible damage and improving patient outcomes during mechanical ventilation.

References

1. Medina A, Del Villar Guerra P, Valle Ortiz JR, et al. Rheological theory applied to mechanical ventilation in acute respiratory distress syndrome: A new paradigm for understanding and preventing ventilator-induced lung injury. J Clin Med 2025; 14(18):6544.

2. Cipulli F, Vasques F, Duscio E, et al. Atelectrauma or volutrauma: the dilemma. J Thorac Dis 2018; 10(3):1258–1264.

3. Donoso FA, Cruces RP. Daño pulmonar inducido por ventilación mecánica y estrategia ventilatoria convencional protectora. Rev Chil Pediatr 2007; 78 (3): 241-252

4. Castaño J, Giraldo MA, Montoya Y, et al. Electropneumatic system for the simulation of the pulmonary viscoelastic effect in a mechanical ventilation scenario. Sci Rep 2023; 13(1):21275.

5. Tuza FA d´Alegria, De Sá PM, Castro HA, et al. Combined forced oscillation and fractional-order modeling in patients with work-related asthma: a case–control study analyzing respiratory biomechanics and diagnostic accuracy. Biomed Eng OnLine 2020; 19(1):93.

6. Zhang W, Capilnasiu A, Sommer G, et al. An efficient and accurate method for modeling nonlinear fractional viscoelastic biomaterials. Comput Methods Appl Mech Eng 2020; 362:112834.

7. Ganzert S, Möller K, Steinmann D, et al. Pressure-dependent stress relaxation in acute respiratory distress syndrome and healthy lungs: an investigation based on a viscoelastic model. Crit Care 2009; 13(6):R199.

8. Li Z, Pei Y, Wang Y, et al. An enhanced respiratory mechanics model based on double-exponential and fractional calculus. Front Physiol 2023; 14:1273645.

9. Modesto I Alapont V, Aguar Carrascosa M, Medina Villanueva A. Stress, strain y potencia mecánica. ¿Es la ingeniería de materiales la respuesta para prevenir la lesión inducida por el ventilador? Med Intensiva 2019; 43(3):165-175.

10. Suki B, Stamenović D, Hubmayr R. Lung parenchymal mechanics. Compr Physiol 2011; 1(3):1317-5131.

11. Faffe DS, Zin WA. Lung parenchymal mechanics in health and disease. Physiol Rev 2009; 89(3):759-775.

12. Sarabia-Vallejos MA, Zuñiga M, Hurtado DE. The role of three-dimensionality and alveolar pressure in the distribution and amplification of alveolar stresses. Sci Rep 2019; 9(1):8783.

13. Dai Z, Peng Y, Mansy HA, et al. A model of lung parenchyma stress relaxation using fractional viscoelasticity. Med Eng Phys 2015; 37(8):752–758.

14. Jóźwiak B, Orczykowska M, Dziubiński M. Fractional generalizations of maxwell and kelvin-voigt models for biopolymer characterization. PLOS ONE 2015; 10(11):e0143090.

15. D'Angelo E, Calderini E, Tavola M, et al. Standard and viscoelastic mechanical properties of respiratory system compartments in dogs: Effect of volume, posture, and shape. Respir Physiol Neurobiol 2019; 261:31-39.

16. Suki B, Bates JHT. Lung tissue mechanics as an emergent phenomenon. J Appl Physiol Bethesda Md 1985 2011; 110(4):1111-1118.

17. Nam S, Lee J, Brownfield DG, et al. Viscoplasticity enables mechanical remodeling of matrix by cells. Biophys J 2016; 111(10):2296-2308.

18. Anssari-Benam A, Screen HRC, Bucchi A. Insights into the micromechanics of stress-relaxation and creep behaviours in the aortic valve. J Mech Behav Biomed Mater 2019; 93:230-245.

19. Lin CY, Chen YC, Lin CH, et al. Constitutive equations for analyzing stress relaxation and creep of viscoelastic materials based on standard linear solid model derived with finite loading rate. Polymers 2022; 14(10):2124.

20. Daoud E, Franck C. Alveolar mechanics: A new concept in respiratory monitoring. J Mech Vent 2022; 3(4):178-188.

21. Santini A, Mauri T, Dalla Corte F, et al. Effects of inspiratory flow on lung stress, pendelluft, and ventilation heterogeneity in ARDS: a physiological study. Crit Care 2019; 23(1):369.

22. Albaiceta GM. Curvas presión-volumen en la lesión pulmonar aguda. Med Intensiva 2009; 33(5):243-250.

23. Demory D, Arnal JM, Wysocki M, et al. Recruitability of the lung estimated by the pressure volume curve hysteresis in ARDS patients. Intensive Care Med 2008; 34(11):2019-2025.

24. Sattari S, Mariano CA, Vittalbabu S, et al. Introducing a custom-designed volume-pressure machine for novel measurements of whole lung organ viscoelasticity and direct comparisons between positive- and negative-pressure ventilation. Front Bioeng Biotechnol 2020; 8:578762.

25. Modesto I Alapont V. Rheological theory of ergotrauma. CHEST Crit Care 2025; 3(4):100190.

26. Fajardo-Campoverdi A, López-Fernández Y, Vivanco P, et al. Lung Mechanotransduction, the minuet of Biophysics (Part 1). J Mech Vent 2025; 6(3):108-119.

27. Pincus SM, Goldberger AL. Physiological time-series analysis: what does regularity quantify? Am J Physiol-Heart Circ Physiol 1994; 266(4):H1643-656.

28. Tonelli R, Rizzoni R, Grasso S, et al. Stress–strain curve and elastic behavior of the fibrotic lung with usual interstitial pneumonia pattern during protective mechanical ventilation. Sci Rep 2024; 14(1):13158.

29. Thebaud C, Fresnel E, Arrouch S, et al. Impact de la ventilation mécanique invasive dans l'exacerbation aiguë de la pneumopathie interstitielle diffuse : étude sur banc d'essai. Rev Mal Respir Actual 2025; 17(1):38-39.

30. Tang BT, Pickard SS, Chan FP, et al. Wall shear stress is decreased in the pulmonary arteries of patients with pulmonary arterial hypertension: An image-based, computational fluid dynamics study. Pulm Circ 2012; 2(4):470-476.

31. Bonfanti A, Kaplan JL, Charras G, et al. Fractional viscoelastic models for power-law materials. Soft Matter 2020; 16(26):6002-6020.

32. Fajardo-Campoverdi A, López-Fernández Y, Vivanco P, Ibarra-Estrada M, González A, Medina A, et al. Lung Mechanotransduction, the minuet of Biophysics (Part 2). J Mech Vent 2025; 6(3):122-130.