# Mathematical Simulation of Pulmonary Haemorrhage Cardiovascular Model

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***Abstract:*** *A novel hybrid model combining lumped and distributed parameters has been developed to explore how pulmonary hemorrhage affects lung mechanics. Drawing on the principles of an electrical circuit analogy, the model integrates resistors and capacitors to represent lung dynamics. Built on a Simulink-based framework, it features user-selectable parameters and an intuitive graphical interface, enabling the simulation of pathological conditions with an emphasis on pulmonary hemorrhage. The study specifically examines variations in lung compliance by modeling the pulmonary system's response to pressure-controlled ventilation. Findings reveal that even slight changes in compliance can markedly impact air volume, underscoring the potential risks to patients with pulmonary hemorrhage. These results highlight the critical need for precise ventilator adjustments to mitigate adverse outcomes. This innovative use of the Windkessel model not only aligns closely with experimental data but also provides valuable insights into pulmonary ventilation mechanics, offering a robust tool for managing respiratory and cardiac pathologies.*

**Keywords:** *Pulmonary hemorrhage, Ventilation, Mathematical modeling.*

## 1. Introduction

Hemorrhage, often triggered by injuries or surgical procedures, leads to blood loss that, if severe, can result in exsanguination. Hemorrhagic shock sets off complex neuroendocrine responses that disrupt the function of vital organs such as the brain, kidneys, heart, lungs, and gastrointestinal system. Prompt intervention, including immediate treatment and blood transfusion, is crucial to improve patient outcomes [1]. Understanding hemorrhagic shock in humans poses significant challenges due to the diversity of physiological responses. Animal models, particularly mice, have proven to be effective in replicating clinical scenarios, offering advantages like cost-efficiency and the ability to utilize genetic modifications. Despite this, accurately monitoring and interpreting physiological changes after severe bleeding remains challenging, often contributing to variability in experimental results [2, 3].

Previous hemorrhage models have struggled to provide accurate simulations of hemodynamics, emphasizing the need for improved mathematical approaches. To address this gap, a novel model was developed to integrate multiple functional systems of the human body while incorporating the cardiovascular effects of hemorrhage and hypothermia. This model employs mathematical tools such as differential equations and the Euler method for concurrent solution generation [4]. In the context of pulmonary hemorrhage, two mechanisms were prioritized for simulation: autoregulatory and nervous system responses, with a specific focus on the baroreceptor reflex. The model employs compartmental analysis to replicate both the immediate and prolonged effects of blood loss, capturing the compensatory and de-compensatory phases associated with long-term hemorrhage [5].

The Windkessel model stands out for its dependability, ethical compatibility, and simplicity, making it a valuable tool in cardiovascular research. It has played a pivotal role in advancing the understanding of pathological components related to cardiovascular disease, forming the foundation for numerous subsequent models. Widely recognized as the first lumped-parameter arterial model based on hemodynamic principles, it remains the gold standard for systemic circulation analysis. Its ability to simulate an elastic reservoir, facilitated by the aortic valve, allows researchers and clinicians to evaluate arterial compliance and precisely represent afterload in clinical practice [6, 7].

While the Windkessel model has predominantly been applied to systemic circulation, its use in pulmonary circulation remains limited. The following sections explore its potential application in the context of pulmonary distress and hemorrhage. Given its flexibility, simplicity, and user-friendly nature, this model holds significant promise for advancing the understanding of respiratory dynamics in pulmonary hemorrhage. By leveraging mathematical modeling and Simulink-based simulations, this approach aims to enhance diagnostic accuracy, refine therapeutic strategies, and ultimately improve patient care.

## Materials and Methods :

This study investigates the effects of pulmonary hemorrhage on lung ventilation through the development of a mathematical lung model. Using Simulink simulation software, a circuit diagram was created based on the Windkessel model, offering an intuitive framework for analysis. The model incorporates adjustable controls that enable detailed observation of changes in both ventilation and hemodynamics across varying degrees of pulmonary hemorrhage. Furthermore, this work examines the role of the bronchi in influencing pulmonary ventilation, providing a comprehensive evaluation of respiratory function under diverse pathological conditions. These findings offer valuable insights into respiratory mechanics, with implications for understanding and managing pulmonary hemorrhage.

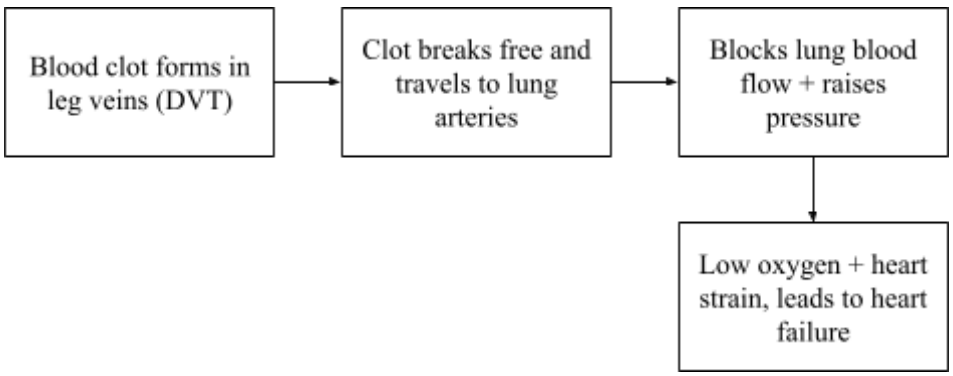



**Figure 1: Block diagram of the overall process**

### 2.1 Circuit Analogy

Figure 2 presents a circuit diagram that illustrates the impact of pulmonary hemorrhage and bronchial dynamics on pulmonary ventilation. This diagram adapts the Windkessel model, drawing a clear analogy between airflow in the lungs and electric current in a circuit, where pressure corresponds to electrical potential. Mechanical resistance is

defined as the ratio of pressure increase to airflow, mirroring the concept of electrical resistance. This analogy proves particularly valuable when analyzing airflow leakage caused by pulmonary hemorrhage, offering a practical and intuitive representation of the underlying physiological processes.

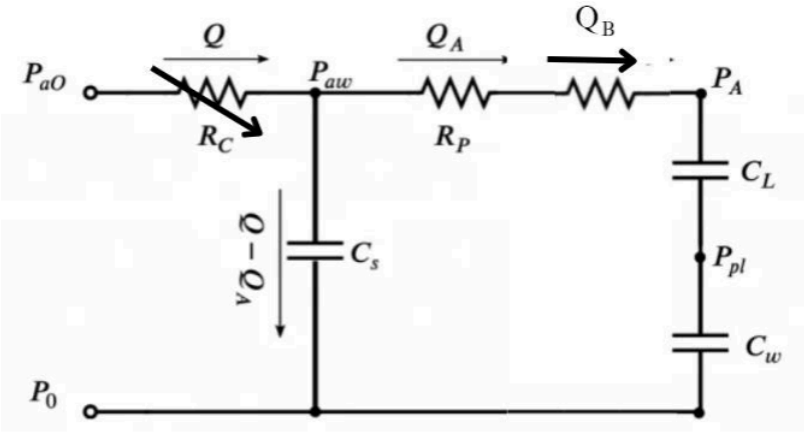



**Fig 2 Circuit diagram of pulmonary ventilation with bronchial effect and hemorrhage.**

The upper and lower airways contribute differently to overall resistance, represented by $R_C$ (upper airway resistance) and $R_P$ (lower airway resistance), respectively. Upper airway resistance ($R_C$) plays a crucial role in modulating the effects of pulmonary hemorrhage by restricting airflow, which can further exacerbate internal bleeding. These resistive components are arranged in series, connecting to air tanks that represent the compliance of the chest wall ($C_W$) and lungs ($C_L$). This series configuration ensures a uniform airflow distribution through both compartments.

In parallel with this arrangement lies a capacity that models the volume of air not involved in gas exchange. Under normal physiological conditions and at low respiratory frequencies, this volume is typically negligible.

Critical pressures within the system, such as alveolar pressure ($P_A$), pleural pressure ($P_L$), airway pressure ($P_{aw}$), and bronchial pressure ($P_B$), are meticulously monitored to assess respiratory function. The pressure at the airway opening ($P_{aO}$), encompassing the mouth and nasal cavities, serves as the entry point for the system, reflecting the interface between external and internal respiratory dynamics.

### 2.2 System Response

The circuit in Fig 2 can be described by the following equations:

$$P_{aO} = Q_{RC} + C_S \int Q - Q_A - Q_B \tag{1}$$

$$C_S \int (Q - Q_A - Q_B) = -(Q_A + Q_B)R_P - (C_L + C_w) \int Q_A - Q_B \tag{2}$$

The transfer function is:

$$H(s) = \frac{Q(s)}{P_{aO}(s)} = \frac{s^2 + s\frac{1}{R_P}\left(\frac{1}{C_S} + \frac{1}{C_{eq}}\right)}{s^2(R_C) + s\left(\frac{R_C + R_P + \frac{R_C C_S}{C_{eq}}}{C_S R_P}\right) + \frac{1}{C_{eq} C_S R_P}} \tag{3}$$

The series capacitance is expressed as:

$$\frac{1}{C_{eq}} = \frac{1}{C_L} + \frac{1}{C_W} \quad (4)$$

The following parameters were given for modeling in Simulink

**Table 1. Simulation values**

| PARAMETER | VALUE | UNIT |
|---|---|---|
| RC | 0- 0.1 | Cm H2O s/L |
| RP | 0.5 | Cm H2O s/L |
| CL | 0.2 | Cm H2O s/L |
| CW | 0.2 | L/cm H2O |
| CS | 0.005 | L/cm H2O |
| RB | 2.5 | Cm H2O s/L |

The system has been updated to incorporate an analysis of bronchial resistance following the division of the peripheral pathway into bronchial paths. This enhancement enables a more detailed examination of bronchial parameters, which play a pivotal role in shaping overall lung functionality. The inclusion of bronchial resistance analysis is particularly important, as both the bronchial and peripheral pathways are highly susceptible to the effects of pulmonary hemorrhage. By focusing on these areas, the updated system provides a more comprehensive understanding of their impact on lung performance, offering critical insights into the underlying mechanisms of respiratory dysfunction.

### 2.3 Effect of Bronchial and Alveolar Pressures During Inhalation and Exhalation

In this model of respiratory mechanics, bronchial and alveolar pressures exhibit distinct behaviors during the phases of inhalation and exhalation. At sea level, bronchial pressure is approximately 760 mmHg but slightly increases during inhalation, reaching about 761 mmHg. In contrast, alveolar pressure during inhalation decreases to around 758 mmHg, falling below atmospheric pressure.

During exhalation, this dynamic reverses. Bronchial pressure drops below alveolar pressure, sometimes reaching as low as 757 mmHg, while alveolar pressure rises closer to atmospheric levels, measuring approximately 759 mmHg. Notably, during inhalation, bronchial pressure exceeds alveolar pressure, but this relationship inverts during exhalation. This pressure differential plays a critical role in driving pulmonary ventilation, highlighting the intricate balance required for effective respiratory mechanics.

### 2.4 Impact of Pulmonary Haemorrhage on Ventilation

Pulmonary hemorrhage, characterized by bleeding within the lungs, significantly disrupts ventilation and overall respiratory function. The severity and extent of the hemorrhage determine its specific effects. One of the primary concerns is reduced oxygenation, as blood in the alveoli diminishes the surface area available for gas exchange. This leads to lower oxygen uptake and increased carbon dioxide retention, compromising pulmonary gas exchange.

Additionally, the accumulation of blood within lung tissues decreases lung compliance, making it harder for the lungs to expand during inhalation. This reduced compliance elevates the effort required for breathing, further exacerbating respiratory challenges. Blood within the airways can also partially or completely obstruct airflow, increasing airway resistance and impeding normal ventilation. These disruptions result in respiratory distress and reduced ventilation efficiency. Given the variability in pulmonary hemorrhage effects, a stage-dependent analysis is essential to fully understand its impact on respiratory mechanics.

### 2.5 Modeling Lung Ventilation with Pulmonary Haemorrhage

Simulink software provides a robust platform for simulating lung ventilation under conditions of pulmonary hemorrhage by converting mathematical equations into functional blocks. This approach simplifies the analysis of the lung model across varying degrees of hemorrhage. The developed model, illustrated in Fig. 4, includes multiple gain blocks with parameters detailed in Table 1. A control knob allows for adjustments to the severity of pulmonary hemorrhage, with settings ranging from low to high, each corresponding to changes in ventilation. As hemorrhage severity increases, the air output volume decreases accordingly.

The model's input is monitored using Simulink's Scope, labeled "PaO vs. Time," which processes a square wave input to simulate the inhalation and exhalation phases of the respiratory cycle. This square wave simulation effectively captures the dynamic response of the lungs to varying levels of haemorrhage, offering a powerful tool for analyzing the interplay between pulmonary ventilation and haemorrhage severity.

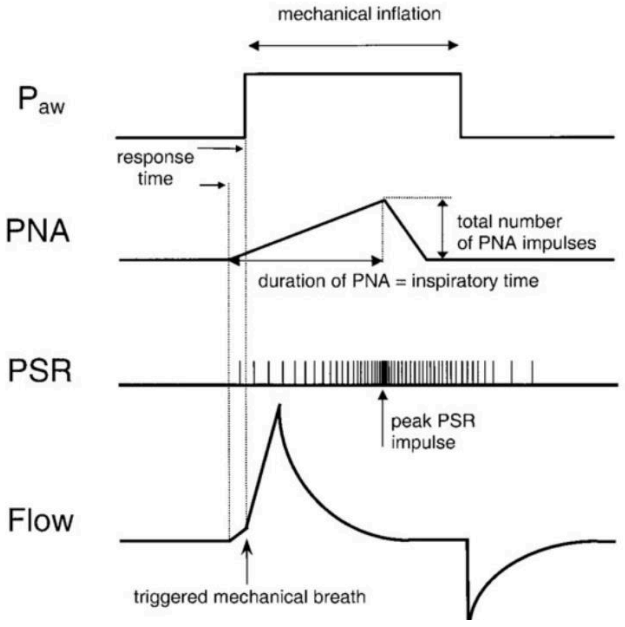



**Figure 3: Square wave input to replicate the inhalation process during ventilation.**

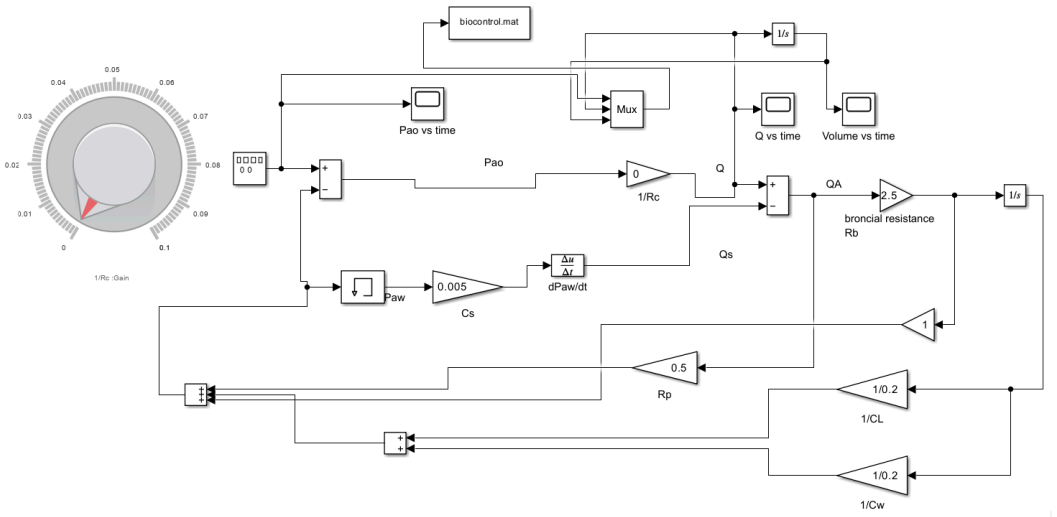
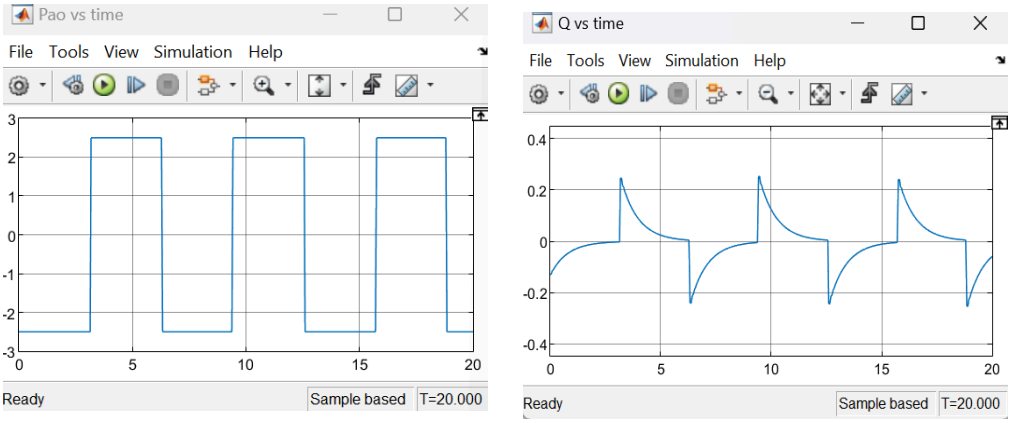



**Figure 4. Simulink model of pulmonary ventilation with hemorrhage**

## 3. RESULTS AND ANALYSIS

A square waveform input with a defined peak-to-peak amplitude is applied to the lung model to simulate the effects of pulmonary hemorrhage, with the system's response carefully measured. This waveform is designed to replicate the physiological processes of inspiration and expiration, making it a valuable tool for analyzing hemodynamic changes.

As shown in Fig. 5, the flow quickly returns to zero during the expiratory phase, resulting in a plateau phase in the volume. This is followed by the inspiratory phase, where the flow curve exhibits a symmetrical but negative shape, peaking at the same amplitude as during expiration. During this phase, the volume increases gradually, accurately reflecting the dynamics of inspiration. Together, these graphs provide a clear representation of both inspiration and expiration, capturing the essential features of respiratory cycles under the influence of pulmonary hemorrhage.




**a)** **b)**

**Figure 4: a) Input square wave, b) Output Wave form**

The pulmonary hemorrhage lung model was analyzed using a Simulink framework featuring two nodes. Outputs such as "flow (Q) vs. time" and "volume vs. time" were evaluated to assess the model's behavior. The analysis revealed that altering frequency did not significantly affect the flow or volume curves influenced by pulmonary hemorrhage, bronchial effects, and alveolar dynamics. However, a higher frequency led to a shorter plateau phase in the volume, with this reduction closely tied to the extent of pulmonary hemorrhage.

Further exploration of hemorrhage effects on lung volume was conducted using a control knob in Simulink. Adjusting the knob setting from 0.1 to 0.01 reduced the percentage of hemorrhage, which in turn influenced the model's output parameters. While the resulting waveforms provide valuable insights into respiratory mechanics, their application in clinical settings is challenging due to the need to maintain consistent positive pressure gradients, avoiding negative pressures.

In the Simulink model, although the amplitude remains constant, the rise and fall times of the waveforms decrease as frequency increases. At different stages of pulmonary hemorrhage, these rise and fall times increase, reflecting changes in respiratory dynamics.

As shown in Figures 6 and 7, variations in "Q vs. time" and "volume vs. time" are strongly linked to changes in pulmonary hemorrhage. Notably, a decrease in upper airway resistance ($Rc$) correlates with an increased likelihood of pulmonary hemorrhage. This finding indicates that reduced resistance in the respiratory circuit may amplify hemodynamic disturbances and internal bleeding, exacerbating the risk of pulmonary hemorrhage.

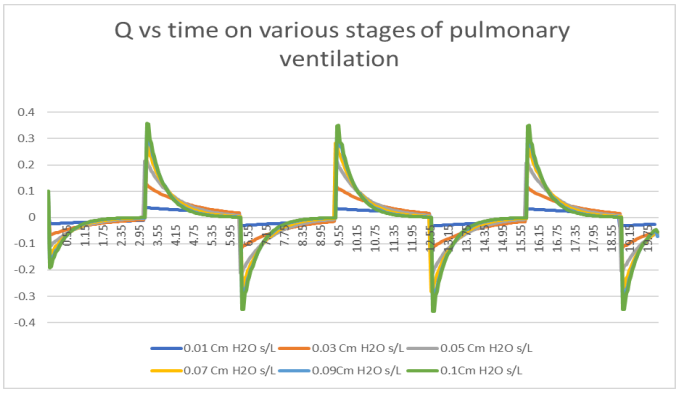



**Figure 7: Graph of Q vs time on various stages of pulmonary ventilation**

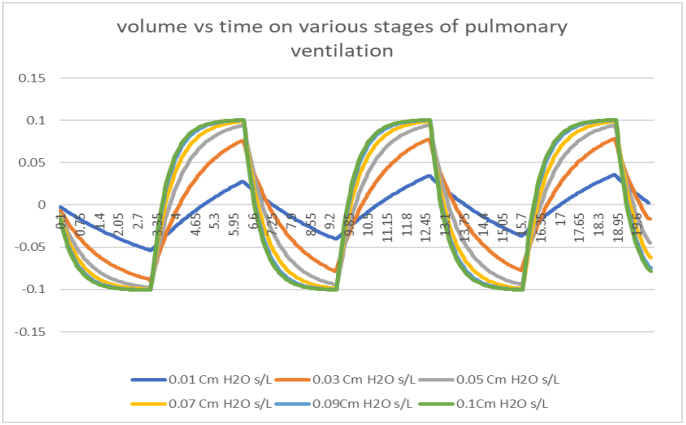



**Figure 8: Graph of volume vs time on various stages of pulmonary ventilation**

## 4. DISCUSSIONS

Strong correlations were observed when the pulmonary hemorrhage model's outputs were compared to clinical data from hemorrhagic cases, particularly in lung compliance and airway resistance changes. These findings validated the model, demonstrating its ability to simulate physiological responses with high fidelity. By incorporating dynamic parameter adjustments, the model facilitates real-time simulations that closely resemble clinical scenarios. Its accuracy was further confirmed through statistical analysis using RMSE and correlation metrics, underscoring its potential as a clinical decision-support tool for managing pulmonary hemorrhage and optimizing ventilator settings.

Looking ahead, integrating the model into real-time ICU monitoring systems could enhance patient-specific treatment strategies. Achieving this goal will require additional validation through prospective clinical trials. Built on an electrical analogy and dynamic simulations using Simulink, the model aligns with established mathematical frameworks validated in clinical settings. Its reliance on clinical data for parameter calibration strengthens its foundation in real-world applications, ensuring relevance and accuracy. Dynamically adapting to variations in hemorrhage severity, the model has been verified against clinical observations, making it a valuable tool for both research and clinical practice.

The Windkessel-based approach employed by this pulmonary hemorrhage model mirrors techniques used in notable cardiovascular research, such as those by Tawhai et al. (2011). Its ability to dynamically adjust to different levels of haemorrhage replicates physiological responses observed in clinical settings, offering significant potential for refining diagnostic and treatment strategies.

Validation of the model followed Tammy J. Doherty's established method, which integrates clinical and experimental data to ensure the accuracy of mathematical simulations. This approach involves comparative analyses that match model predictions with observed physiological changes in clinical studies, such as blood volume, flow rates, and pressures influenced by vascular resistance and compliance. By fine-tuning the model’s parameters to more closely align with real-world physiological reactions, this validation process enhances the model's specificity and sensitivity for predicting human responses in clinical settings.

The validation process involved comparing outputs, such as changes in resistance and compliance, to clinical data collected through Doherty's framework. Simulated results were benchmarked against documented clinical effects of pulmonary hemorrhage, ensuring that the model accurately captures dynamic physiological responses. Doherty's methodology provided a robust standard for assessing the simulation’s fidelity and clinical relevance.

This study also builds on prior findings by focusing on bronchial effects in pulmonary ventilation—an area often overlooked. Unlike other models that emphasize variables such as dynamic compliance in volume-controlled ventilation or Positive End Expiratory Pressure (PEEP), this approach incorporates distinct lung ($CL$) and chest wall ($CW$) compliance parameters. This distinction enables more precise measurements and a deeper understanding of the interplay between hemorrhage and ventilation mechanics. By modeling pulmonary hemorrhage at varying severities, the study provides clearer insights into how hemorrhage-related changes in compliance influence ventilation.

In contrast, previous research has concentrated on developing mathematical models to mimic hemorrhagic transformation in cerebrovascular conditions, particularly in stroke. These models assess the mechanical and physiological impacts of such events, focusing on factors like blood leakage fraction, hematoma volume, and capillary compression due to elevated intracranial pressure. Using a 2D vasculature framework, this body of work offers a novel perspective on the effects of hemorrhagic transformation on brain tissue, complementing the pulmonary focus of this study.

## 5. CONCLUSION

The findings underscore the critical role of monitoring ventilation parameters in accurately evaluating the impact of pulmonary hemorrhage, as well as the bronchial and alveolar effects on respiratory function. Variations in circuit parameters, particularly upper airway resistance ($Rc$), significantly influence ventilator behavior and the volume of air required for effective ventilation.

Notably, increasing the settings on the control knobs is used to simulate hemorrhage across different lung capacities which can result in dangerously high input volumes. This analysis provides valuable insights into the effects of haemorrhage on ventilation at various stages, highlighting the importance of addressing hemorrhage in respiratory management strategies.

Additionally, bronchial and alveolar dynamics play a pivotal role in the overall mechanics of ventilation. A deeper understanding of these variables is essential for developing accurate lung models and comprehensively assessing the interplay between hemorrhage, bronchial, and alveolar effects on ventilation. These insights are crucial for advancing respiratory management and optimizing treatment approaches in clinical practice.

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