

A1: Simulation of Respiratory Mechanics

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1 Introduction

The human respiratory system can be modeled using advanced simulation software, incorporating key pulmonary parameters. An open-source attempt at such a model is available in a MATLAB Simulink project published by [1]. This report explores the behavior of three major pulmonary conditions, simulated through the aforementioned Simulink model. Below is an overview of the respiratory model.

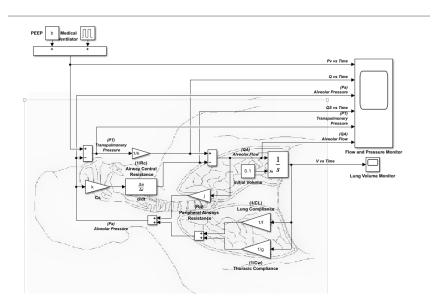


Figure 1: Overview of the model

Here are the important parameters used in this simulatuion listed.

- BPM (Breaths Per Minute): Number of breaths delivered by a ventilator (or taken spontaneously) per minute.
- PEEP (Positive End-Expiratory Pressure): Pressure maintained in the lungs at the end of expiration to prevent alveolar collapse.
- Peak Pressure (PIP): Maximum pressure reached during inspiration (reflects airway resistance + lung compliance).
- I:E ratio:The ratio of time spent in inspiration (I) to expiration (E) during a single breathing cycle.
- Respiratory Minute Volume (MV): Total air moved per minute (Tidal Volume × Respiratory Rate).
- Lung Compliance (C_L): Ease of lung expansion (Δ Volume/ Δ Pressure).
- Thoracic Compliance (C_Th): Chest wall expandability.
- Central Airway Resistance (R_aw): Opposition to airflow in large airways (trachea, bronchi).
- Peripheral Airway Resistance: Resistance in small airways (bronchioles).

• Airway Tissue Compliance: Ability of airways to resist collapse during expiration.

2 Normal Person

In a normal healthy individual, key respiratory parameters fall within the following physiological ranges:

- BPM (Breaths Per Minute): 12–20 breaths/min (15)
- PEEP (Positive End-Expiratory Pressure): 0 cmH₂O (normal spontaneous breathing)
- Peak Inspiratory Pressure (PIP): 8–12 cmH₂O (10)
- Respiratory Minute Volume (MV): 4.8–12 L/min (typically 7.5 L/min)
- I:E Ratio: 1:1 (inspiration:expiration)
- Lung Compliance (C_L): 0.1 L/cmH₂O
- Thoracic Compliance (C_{Th}): 0.1 L/cmH₂O
- Central Airway Resistance (R_{aw}): 3 cmH₂O/L/s
- Peripheral Airway Resistance: 0.05 cmH₂O/L/s
- Airway Tissue Compliance: Normal (resists collapse during expiration) :0.005 cmH₂O/L/s

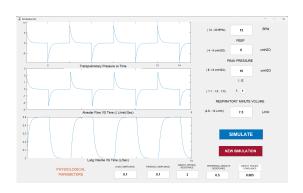


Figure 2: variation of flow,lung volume ,pressure

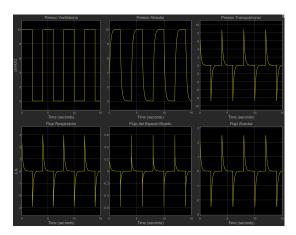


Figure 3: variation of flow,lung volume ,pressure

3 Restrictive Pulmonary Disease

People with restrictive lung disease can't fully fill their lungs with air. Their lungs are restricted from fully expanding.

Restrictive lung disease most often results from a condition that causes stiffness in the lungs themselves. In other cases, stiffness of the chest wall, weak muscles, or damaged nerves may restrict how much your lungs can expand.

3.1 Acute Respiratory Distress Syndrome (ARDS)

Acute respiratory distress syndrome (ARDS) occurs when lung swelling causes fluid to build up in the tiny elastic air sacs in the lungs. These air sacs, called alveoli, have a protective membrane, but lung swelling damages that membrane. The fluid leaking into the air sacs keeps the lungs from filling with enough air. This means less oxygen reaches the bloodstream, so the body's organs don't get the oxygen they need to work properly.

The following list shows how the physiological parameters change for a patient with ARDS with respect to a normal patient.

- Lung Compliance (C_{ℓ}) : Decreases significantly due to alveolar flooding, surfactant loss, and increased stiffness of lung tissue, which reduces the lungs' ability to expand.
- Thoracic Compliance (C_{th}) : Decreases as a result of reduced lung compliance, since total thoracic compliance is influenced by both lung and chest wall mechanics.
- Central Airway Resistance (R_{av}): May increase slightly due to airway inflammation, edema, or mucus obstruction, although this is less prominent in ARDS.
- Peripheral Airway Resistance: Slightly increases because of narrowing or collapse of distal airways due to inflammation and alveolar damage.
- Airway Tissue Compliance: Decreases due to stiffening and loss of elasticity in the lung parenchyma caused by diffuse alveolar injury and edema.

3.2 Simulation Results

- **BPM** 15 breaths/min
- **PEEP**: $0 \text{ cmH}_2\text{O}$
- Peak Inspiratory Pressure : 10 cmH₂O
- Respiratory Minute Volume (MV): 2 L/min (typically 7.5 L/min)
- I:E Ratio: 1:1
- Lung Compliance (C_L): 0.02 L/cmH₂O
- Thoracic Compliance (C_{Th}): 0.04 L/cmH₂O
- Central Airway Resistance (R_{aw}): 3.1 cmH₂O/L/s
- Peripheral Airway Resistance: 0.6 cmH₂O/L/s

• Airway Tissue Compliance: 0.002 cmH₂O/L/s

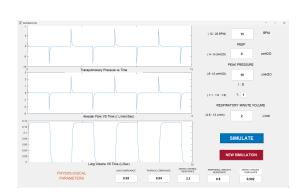


Figure 4: variation of flow,lung volume ,pressure

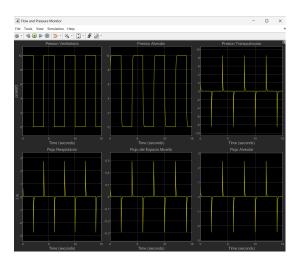


Figure 5: variation of flow,lung volume ,pressure

4 Obstructive Pulmonary Disease

People with obstructive lung disease have shortness of breath because it's hard for them to exhale all the air from their lungs. Because of damage to the lungs or narrowing of the airways inside the lungs, the exhaled air comes out more slowly than it should. At the end of a full exhalation, too much air may linger in the lungs.

4.1 Asthma

Asthma is a condition in which your airways narrow and swell and may produce extra mucus. This can make breathing difficult and trigger coughing, a whistling sound (wheezing) when you breathe out and shortness of breath.

For some people, asthma is a minor nuisance. For others, it can be a major problem that interferes with daily activities and may lead to a life-threatening asthma attack.

The following list shows how the physiological parameters change for a patient with Asthma with respect to a normal patient.

- Lung Compliance (C_{ℓ}) : Typically normal or slightly increased due to air trapping and hyperinflation. The lungs may appear more compliant as they retain more air, especially during acute exacerbations.
- Thoracic Compliance (C_{th}) : May be slightly increased due to lung hyperinflation, which pushes the chest wall outward and alters its mechanical properties.
- Central Airway Resistance (R_{av}): Increases significantly due to bronchoconstriction, inflammation, mucus production, and thickening of the airway walls, all of which narrow the central airways.

- Peripheral Airway Resistance: Increases significantly due to inflammation, edema, bronchoconstriction, and mucus buildup, leading to narrowing or collapse of peripheral airways.
- Airway Tissue Compliance: May be normal or slightly decreased in chronic asthma if there is airway remodeling, which can lead to fibrosis and stiffening of the airway tissues. In acute asthma, tissue compliance remains fairly normal.

4.2 Simulation Results

• **BPM** 15 breaths/min

• **PEEP**: $0 \text{ cmH}_2\text{O}$

• Peak Inspiratory Pressure: 10 cmH₂O

• Respiratory Minute Volume (MV): 10.98 L/min (typically 7.5 L/min)

• I:E Ratio: 1:1

• Lung Compliance (C_L): 0.1 L/cmH₂O

• Thoracic Compliance (C_{Th}): 0.3 L/cmH₂O

• Central Airway Resistance (R_{aw}): 6 cmH₂O/L/s

• Peripheral Airway Resistance: 1.1 cmH₂O/L/s

• Airway Tissue Compliance: 0.005 cmH₂O/L/s

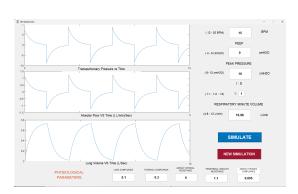


Figure 6: variation of flow,lung volume ,pressure

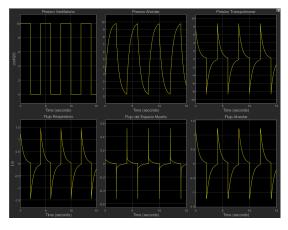


Figure 7: variation of flow,ung volume ,pressure

5 Comparison of Minute Ventilation

Minute Ventilation (MV) is a key respiratory parameter that represents the total volume of air moved in and out of the lungs per minute. It is calculated as:

 $MV = Tidal Volume (V_T) \times Respiratory Rate (RR)$

5.1 Normal Individual

- Typical MV: ~7.5 L/min
- Characteristics: Balanced ventilation with optimal gas exchange
- Simulation Result: Approximately 7.5 L/min (as expected for normal physiology)

5.2 ARDS (Restrictive Pattern)

- Simulated MV: 2 L/min (severely reduced)
- Reason for Reduction:
 - Decreased lung compliance (0.02 vs 0.1 L/cmH₂O in normal)
 - Higher work of breathing due to stiff lungs
 - Reduced tidal volumes to prevent barotrauma
- Clinical Implication: This severe hypoventilation would lead to significant hypoxemia and respiratory acidosis without mechanical ventilation support

5.3 Asthma (Obstructive Pattern)

- Simulated MV: 10.98 L/min (increased)
- Reason for Increase:
 - Airway resistance increased (central: 6 vs 3 cm $H_2O/L/s$; peripheral: 1.1 vs 0.05 cm $H_2O/L/s$)
 - Compensatory tachypnea (though rate was kept at 15 in simulation)
 - Air trapping leading to increased work of breathing
 - Potential for dynamic hyperinflation
- Clinical Implication: Increased work of breathing can lead to respiratory muscle fatigue

5.4 Key Observations

- Restrictive Disease Impact:
 - Marked reduction in MV despite normal respiratory drive
 - Reflects the difficulty in lung expansion

- Clinically would require positive pressure ventilation with PEEP

• Obstructive Disease Impact:

- Increased MV due to compensatory mechanisms
- Higher energy expenditure for ventilation
- Risk of air trapping and auto-PEEP development

• Pattern Differences:

- Restrictive: Low volumes, rapid shallow breathing pattern
- Obstructive: Prolonged expiration, potential for incomplete emptying

5.5 Clinical Relevance

Understanding these ventilation patterns helps in:

- Diagnosing the type of pulmonary disorder
- Guiding mechanical ventilation strategies
- Monitoring disease progression and treatment response
- Setting appropriate ventilator parameters (e.g., higher PEEP for ARDS, lower respiratory rates for asthma)

5.6 Ventilator parameter changes to achieve normal Minute Volume

5.6.1 ARDS

We can reach the Minute Volume of normal value of 7.5 L/min by changing the following Ventilator parameters.

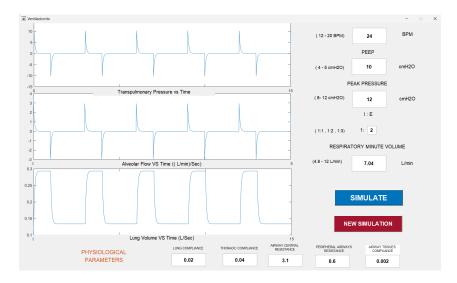


Figure 8: After parameter changes

• **BPM**: 24 breaths/min

• **PEEP**: 10 cmH₂O (normal spontaneous breathing)

• Peak Inspiratory Pressure : 12 cmH₂O (10)

• Respiratory Minute Volume (MV): 7.04 L/min (typically 7.5 L/min)

• I:E Ratio: 1:2

5.6.2 Asthma

We can reach the Minute Volume of normal value of 7.5 L/min by changing the following Ventilator parameters.

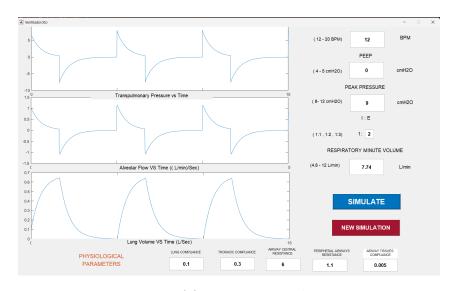


Figure 9: After parameter changes

• **BPM**: 12 breaths/min

• **PEEP**: 0 cmH₂O (normal spontaneous breathing)

• Peak Inspiratory Pressure: 9 cmH₂O (10)

• Respiratory Minute Volume (MV): 7.74 L/min (typically 7.5 L/min)

• I:E Ratio: 1:2

The simulation effectively demonstrates these fundamental differences in respiratory mechanics between restrictive and obstructive pulmonary diseases compared to normal lung function.