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The Impact of CO_2 and O_2 Levels on Respiratory Dynamics-A Simulation Based Study

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Abstract: - The lung model is implemented and simulated respiration mechanics, encompassing factors like airway resistance, lung compliance, and pleural pressure. Lung mechanics involve intricate interactions among various physiological components that aid respiration. Distributed parameter models, unlike lumped parameter models, consider spatial variations within the lung, offering a more detailed insight into respiratory mechanics by accounting for properties like resistance, compliance, and pressure. The chemical regulation of ventilation is crucial for maintaining optimal CO_2 and O_2 levels, involving complex feedback mechanisms mediated by chemoreceptors sensitive to changes in these chemical variables. This study aims to successfully implement a Simulink model that precisely replicates lung mechanisms in response to elevated CO_2 and decreased O_2 levels.

Keywords:Compliance, pleural space, Nonmonic Condition, Hypoxic Condition, body temperature pressure saturated, standard temperature pressure dry

I. INTRODUCTION

The human respiratory system is a sophisticated network of organs, tissues, and structures collaborating to enable gas exchange between the body and the external environment. The development of mathematical formulas to define the input-output properties of lung mechanics is presented in a linearized depiction of the lung mechanics model in figure 1[1].

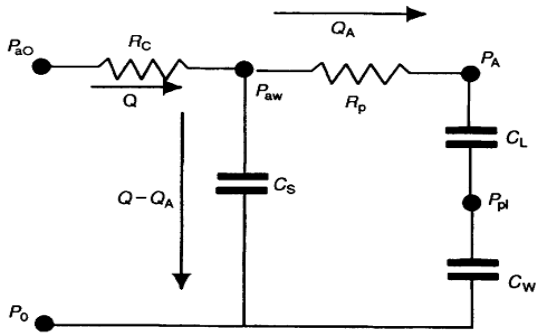

Fig. 1: Linear Model of Respiratory Mechanism

The airways are divided into two categories:

- The larger or central airways, with fluid mechanical resistances equal, $R_{c.}$
- The smaller or peripheral airways, with fluid mechanical resistances equal, $R_{P.}$

This is represented by the connection of the lung (C_l) and chest-wall (C_w) compliances in series. However, a small fraction of the volume of air that enters the respiratory system is shunted away from the alveoli as a result of the compliance of the central airways and gas compressibility.

- M C_L = Lung Compliance
- C_W = Chest-wall Compliance
- C_S = Shunt Compliance

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This shunted volume is very small under normal circumstances at regular breathing frequencies, but becomes progressively more substantial if disease leads to peripheral airway obstruction (i.e., increased R_p) or a stiffening of the lungs or chest-wall i.e., decreased C_L or C_W. The Pressure developed at the different points of the lung model are:

- P_{ao} at the airway opening pressure.
- P_{aw} in the central airways pressure.
- P_A in the alveoli
- P_{pi} in the pleural space (between the lung parenchyma and chest wall).
- P_o, the ambient pressure.

These pressures are referenced to Po, the ambient pressure, which we can set to zero. If the volume flow-rate of air entering the respiratory system is Q, then from Kirchhoff's Second Law (applied to the node Paw), if the flow delivered to the alveoli is QA' then the flow shunted away from the alveoli must be Q - Q_A.

Q = volume flow-rate of air entering the respiratory

Q_A= flow delivered to then alveoli

Q-Q_A = flow shunted away from the alveoli

Applying, Kirchhoff's First Law to the closed-circuit containing C_S, R_P, C_L, and C_w.

$$R_p Q_A + \left(\frac{1}{C_L} + \frac{1}{C_W}\right)\int Q_a\, dt = \frac{1}{C_S}\int (Q - Q_A)dt \quad \ldots\ldots (1)$$

Applying, Kirchhoff's First Law to the circuit containing C_S and R_C.

$$P_{ao} = R_C Q + \frac{1}{C_S}\int Q - Q_A\, dt \quad \ldots\ldots (2)$$

Differentiating Equation (1) and Equation (2) with respect to time, and subsequently reducing the two equations to one by eliminating QA' we obtain the equation relating P_{ao} to Q:

$$\frac{d^2P_{ao}}{dt^2} + \frac{1}{R_p C_T}\frac{dP_{ao}}{dt} = R_p\frac{d^2Q}{dt}\left(\frac{1}{C_L} + \frac{R_C}{R_P C_T}\right)\frac{dQ}{dt} + \frac{1}{R_p C_S}\left(\frac{1}{C_L} + \frac{1}{C_W}\right)Q \quad \ldots\ldots (3)$$

Where C_T is defined by,

$$C_T = \left(\frac{1}{C_L} + \frac{1}{C_W} + \frac{1}{C_S}\right)^{-1} \quad \ldots\ldots (4)$$

Different values are under normal condition and assume that patient has relatively normal mechanics and values of the different pulmonary parameters are:

- R_c = 1cm$H_2O_s L^{-1}$
- R_P = 0.5 cm $H_2O_s L^{-1}$
- C_L = 0.2 L cmH_2O^{-1}
- C_W = 0.2 L cmH_2O^{-1}
- C_S = 0.005 L cmH_2O^{-1}

Substituting the above parameter values into this differential equation and taking its Laplace transform yields, after some rearrangement of terms, the following expression:

$$\frac{Q(s)}{P_{ao}(s)} = \frac{S^2 + 420S}{S^2 + 620S + 4000} \quad \ldots\ldots (5)$$

Consideration for model at Different Frequencies:

-For 15 breaths/minute: ventilator generates a sinusoidal p_{ao} of the amplitude to 2.5 cm H_2O (peak to peak swing in p_{ao} will 5 cm H_2O) and frequency is set at 15 breaths/minute is 0.25 H_z.

-For 60 breaths/minute: frequency is set at 60 breaths/minute is 1 Hz.

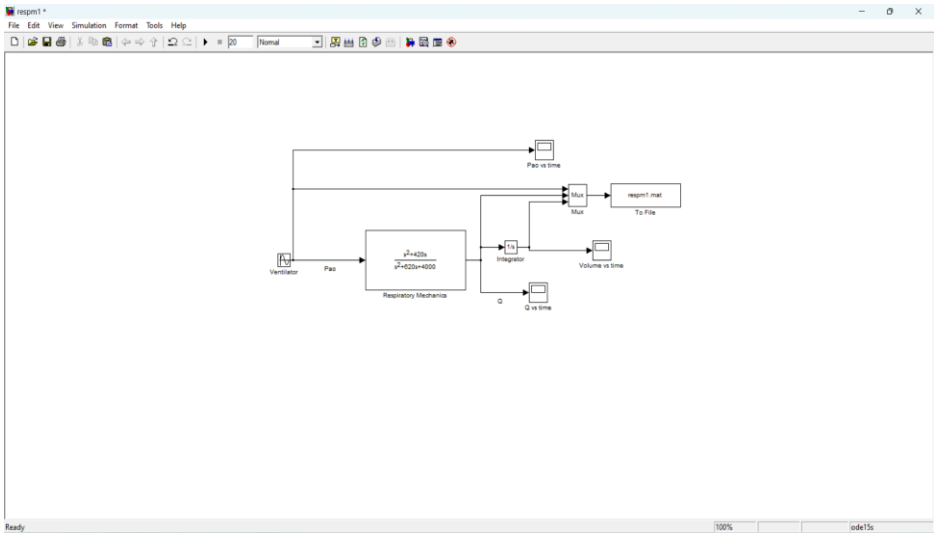


Fig. 2: Simulink model of lung mechanism

II. LITERATURE REVIEW

The intricate and organized structure of the lung offers a challenging yet rewarding research setting that necessitates a thorough comprehension of lung functionality as well as advanced computational resources to more effectively analyze lung biology in various health conditions. Utilizing computational modeling can enhance our insight into the correlation between structure and function within the lung [2].

Machine learning enables rapid simulations for a variety of purposes. In terms of lung operation, studies have identified a consistent nonlinear relationship between specific structural factors and chest wall rigidity. Researchers have noted significant alterations in breathing rate and tissue permeability [3].

This paper introduces a standard respiratory simulation model using LabVIEW and Simulink. By integrating Matlab in LabVIEW, users can concurrently manage basic hardware and execute intricate algorithms. The combination of Simulink and Real-Time Workshop functionalities with LabVIEW, along with the Simulation Interface Toolkit, allows for the integration of the simulation model into LabVIEW. The paper introduces an effective implemented model for ventilation and control, which integrates the benefits of both methods for control objectives [6].

III. DURING DISTRIBUTION PARAMETER

A lumped parameter model simplifies a specific property of a system by consolidating it into a single element. For example, in lung mechanics, the total resistance of the central airways is often represented by a single value, R_c. However, the central airways are actually made up of the trachea and several branches, each with its own fluid mechanical resistance [9].

To more accurately represent the spatial distribution of system properties, a distributed-parameter model is typically used. This model employs one or more partial differential equations (PDEs) with time and spatial dimensions as independent variables, providing a detailed representation based on equations (1) and (2).

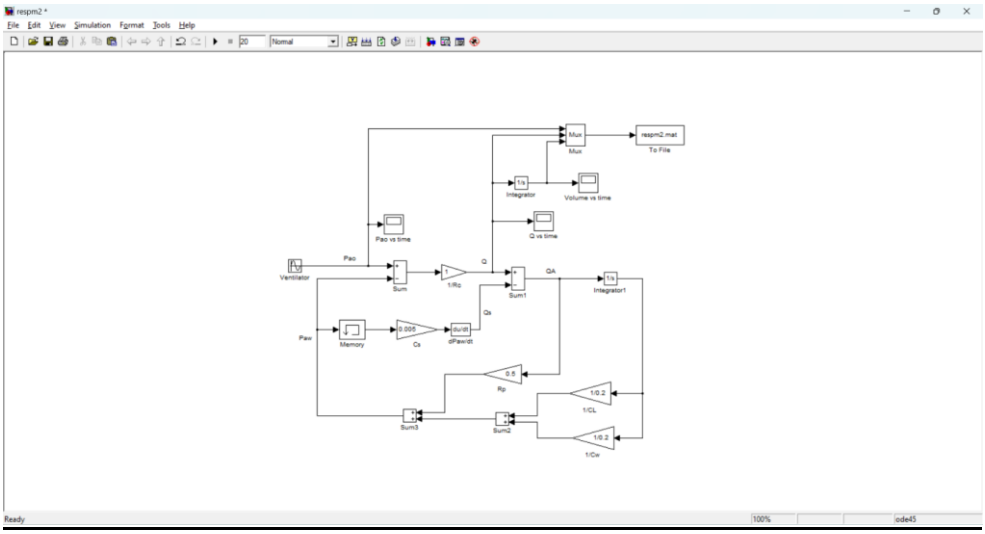


Fig. 3: Simulink model of lung mechanism during distributed parameter

Values for Different Condition

NORMAL:

$R_C = 1 cmH_2O_sL^{-1}$

$R_P = 0.5\ cm\ H_2O_sL^{-1}$

$C_L = 0.2\ L\ cmH_2O^{-1}$

$C_W = 0.2\ L\ cmH_2O^{-1}$

$C_S = 0.005\ L\ cmH_2O^{-1}$

ASTHAMA:

$R_C = 1 cmH_2O_sL^{-1}$

$R_P = 5\ cm\ H_2O_sL^{-1}$

$C_L = 0.2\ L\ cmH_2O^{-1}$

$C_W = 0.2\ L\ cmH_2O^{-1}$

$C_S = 0.005\ L\ cmH_2O^{-1}$

PULMONARY FIBROSIS:

$R_C = 1 cmH_2O_sL^{-1}$

$R_P = 0.5\ cm\ H_2O_sL^{-1}$

$C_L = 0.02\ L\ cmH_2O^{-1}$

$C_W = 0.2\ L\ cmH_2O^{-1}$

$C_S = 0.005\ L\ cmH_2O^{-1}$

EMPHYSEMA:

$R_C = 1 cmH_2O_sL^{-1}$

$R_P = 7.5\ cm\ H_2O_sL^{-1}$

$C_L = 0.4\ L\ cmH_2O^{-1}$

$C_W = 0.2\ L\ cmH_2O^{-1}$

$C_S = 0.005\ L\ cmH_2O^{-1}$

IV. CHEMICAL REGULATION OF VENTILATION

Breathing is one of the most vital processes occurring continuously in the body. The normal breathing rate of an adult human being is 15-20 breaths/min. This system gives the best examples of negative feedback system (gas exchange characteristic of lung). In nonmonic condition, breathing is exclusively controlled by the level of CO_2 in arterial blood. A rise in $PaCO_2$ by 1 mmHg from its normal level of approximately 40 mmHg may increase the ventilatory output by a third of its resting level.

Gas Exchanger:

CO_2 production rate to be $\dot{V_{CO2}}$, this is the rate at which CO_2 is delivered to the lungs from the blood that is perfusing the pulmonary circulation$\dot{V_A}$. The alveolar ventilation represents that portion of the total ventilation, $\dot{V_E}$ that actually participates in the gas exchange process. Part of $\dot{V_E}$ is "wasted" on ventilating the non-gas-exchanging airways in the lungs; this flow is known as "Dead Space Ventilation", $\dot{V_D}$.

$$\dot{V_A} = \dot{V_E} + \dot{V_D} \quad \ldots (6)$$

The ventilatory flow rates are generally measured in BTPS (Body Temperature Pressure Saturated) units, while the CO_2 metabolic production rate is usually expressed in STPD (Standard Temperature Pressure dry, i.e., at 273K and 760 nun Hg) units. The constant k allows volumes and flows measured in BTPS units to be converted into STPD units. This conversion is achieved by equation:

$$\frac{V_{STPD}760}{273} = \frac{V_{BTPS}(P_B - 47)}{310} \quad \ldots (5)$$

On rearranging the equation, we get:

$$K = \frac{V_{STPD}}{V_{BTPS}} = \frac{(P_B - 47)}{863} \quad \ldots (6)$$

The volumetric fractions can be converted into their corresponding partial pressures

$$P_{ICO2} = F_{ICO2}(P_B - 47) \quad \ldots (7)$$

$$P_{ACO2} = F_{ACO2}(P_B - 47) \quad \ldots (8)$$

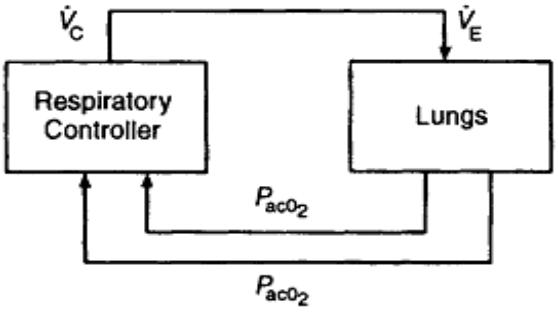
So finally, we can write the equation as

$$P_{ACO2} = P_{ICO2} = \frac{863\dot{V}_{CO2}}{\dot{V}_A} \quad \ldots (9)$$

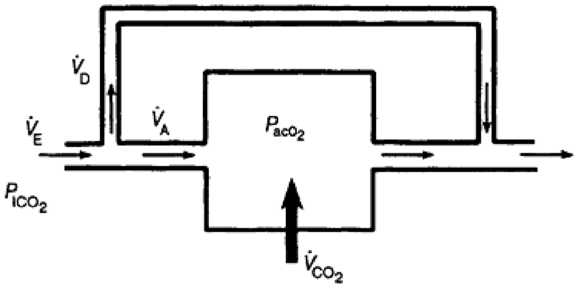
Similarly, we can write for oxygen partial pressure as

$$P_{AO2} = P_{IO2} = \frac{863\dot{V}_{O2}}{\dot{V}_A} \quad \ldots (10)$$

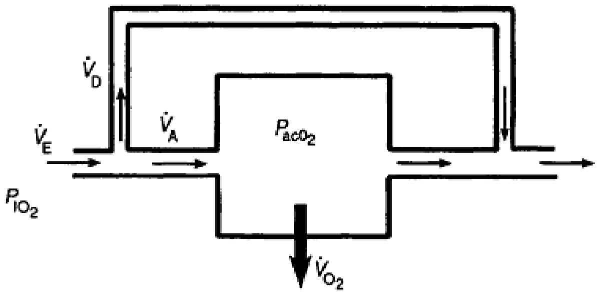
The negative sign in above Equation accounts for the fact that O2 is removed from the lungs by the perfusing blood and, therefore, the alveolar O2 content (P_{ACO2}) will always be lower than the inhaled O_2.



Steady state model of chemical ventilation



Model of steady state CO_2 exchange



Model of steady state O_2 exchange in lungs

Assumptions made for the model

-The body temperature to be 37°C or 310 K

-Saturated water vapor partial pressure of 47 mmHg

-Alveolar partial pressures are completely equilibrated with the corresponding arterial blood gas partial pressures, i.e.,

$$P_{aCO2} = P_{ACO2} \& P_{aO2} = P_{AO2} \quad \ldots \ldots (11)$$

Respiratory Controller –

The controller part of the system includes the chemoreceptors, the neuronal circuits in the lower brain involved in the generation of the respiratory rhythm as well as the neural drive to breathe, and the respiratory muscles.

$$\dot{V}_C = \left(1.46 + \frac{32}{P_{aO2} - 38.6}\right)(P_{aCO2} - 37), \qquad P_{aCO2} > 37$$

$$\dot{V}_C = 0 \qquad , \qquad P_{aCO2} \leq 37$$

Above expression becomes progressively less valid as Pan approaches the P_{AO2} asymptotic value of 38.6, in which case $\dot{V}_C$ would become infinitely large. A Simulink model of chemical regulation of ventilation is shown in figure 4,[9].

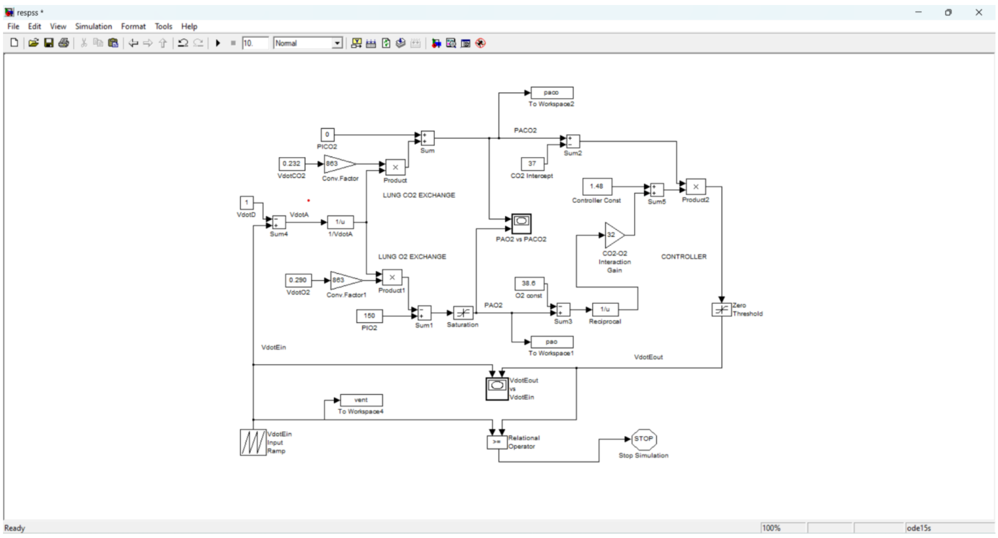


Fig. 4. Simulink modle of chemical regulation of ventilation

V. Result

Various cases of the chemical ventilation models are simulation. It is as under.

Case- I-Nonmonic Condition

Take the value of P_{ICO2} as 150 mm Hg (i.e. 21% room air) P_{ICO2} is set equal to zero. The simulation is terminated when the value of $\dot{V_C}$ becomes equal to $\dot{V_E}$.

Case- II Hypoxic Condition

Subject inhale gas mixture containing only 15% O_2, P_{IO2} is equal to 107 mm Hg P_{ICO2} is left at zero. Graphs are plot graphs between P_{ACO2} and P_{AO2} for both conditions, $\dot{V_E}$. and $\dot{V_C}$ for both condition and steady state values of $\dot{V_C}$,P_{AO2} and P_{ACO2} for both conditions.

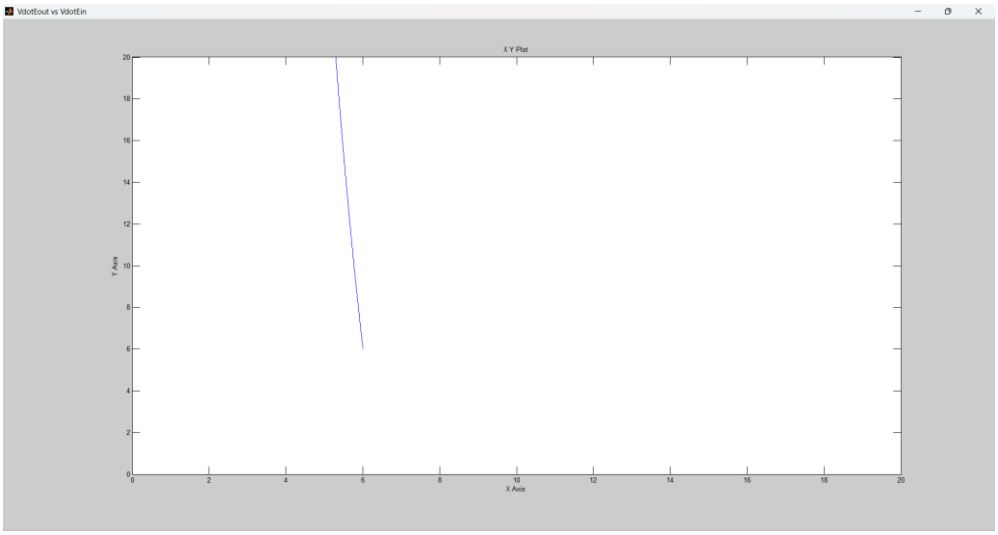


Fig. 5. Graph of chemical regulation of ventilation output vs input

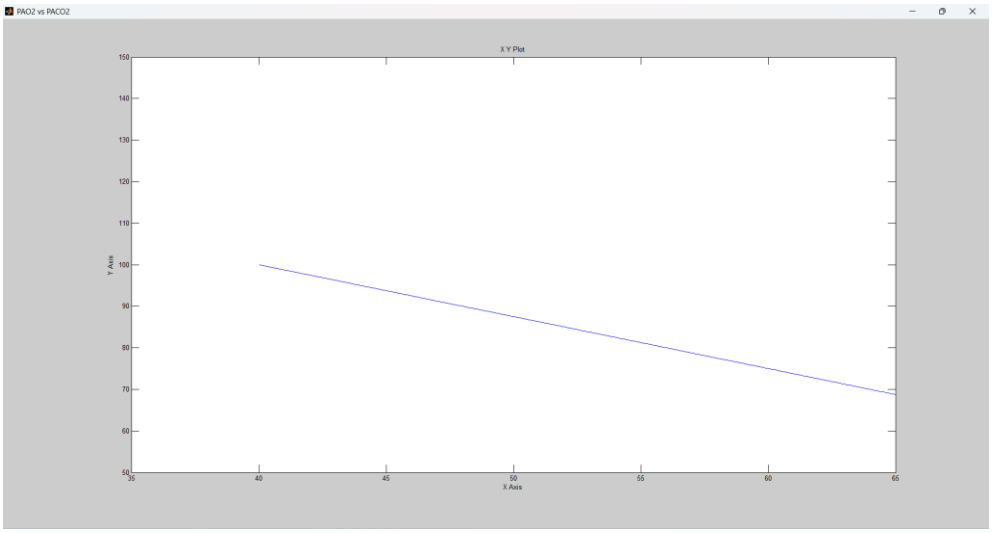


Fig. 6. Graph of P_{AO2} Vs P_{ACO2}

VI. CONCLUSION

The simulation findings offer information on how lung volumes, airflow rates, and pressure fluctuations operate in various parts of the respiratory system. The model effectively mirrors the natural behavior of the lungs, showcasing spatial differences during different breathing conditions. Additionally, the simulation results show how the respiratory system reacts dynamically to chemical triggers.

The model implementation validates physiological reactions, like heightened ventilation in the presence of heightened CO_2 and lowered O_2 levels. This confirms the observed decrease in inhaled oxygen levels.

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