

MODELING, SIMULATION AND ANALYSIS OF LUNG MECHANICS USING LABVIEW

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ABSTRACT

The modeling is performed in order to know the behavior of a system. Modeling is needed in the area of medicine to understand the operation of functional systems of the human body. The model construction and the simulation within engineering are effectively recognized. The purpose of this work is to evaluate the characteristics of lung mechanics of normal person and diseased person and to show how computational and engineering basic tools can help in the biomedical studies.

KEYWORDS

Simulation, Modeling, Lung mechanics, Respiratory system, LABVIEW.

1. INTRODUCTION

The respiratory control system is a nonlinear, multi output, delayed-feedback dynamic system which is perturbed constantly by physiologic and pathologic disturbances. Lung mechanics plays an important role in providing adequate ventilatory support with a minimum of adverse effects. The lung mechanics are considered in two broad categories i.e., study of lung mechanical properties with airflow and without airflow. This paper shows the analysis and construction of the Lung mechanics of respiratory system which allows you to observe the flow of air in the way airfreight and in the lungs in normal conditions and its validation in the model implementation.

2. CONCEPTS

2.1 Respiratory Anatomy

The respiratory system as shown in fig 1, consists of a upper respiratory tract or via driving air formed by the nose, mouth, the pharynx, larynx and trachea; with each breath the upper tract leads the air into the interior and exterior of the ducts and structures that are the respiratory tract and the lungs. The main channels and structures in the lower respiratory tract are the trachea and, within the lungs, the bronchi, the bronchioles and the alveoli. In the depth of the lung, each bronchus is divided into bronchi, secondary and tertiary, which continue branch in airways smaller than are called bronchioles. They end up in air sacs that are called alveoli. [1][2][3].

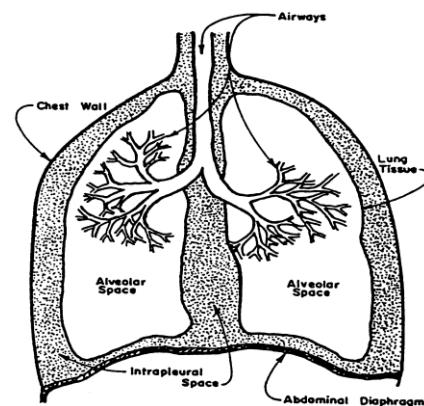


Fig 1 Schematic representation of respiratory system

2.2 Ventilation

The inspiration is an active process that requires muscle contraction of the diaphragm to increase the size of the chest cavity and make a stretch of the lungs, widening the alveoli and the ductwork alveolar and expanding the alveolar gas, which produces a reduction of the pressure inside the lungs less than the atmospheric pressure. Muscle contraction provides the force necessary for the air to be superior the elastic recoil of the lungs, the elastic recoil of the chest wall and the resistance to airflow through the airways and can reach the lungs [6] [7] [8] [9] [10].

Expiration is a passive process that does not require muscle work, just muscle relaxation, here, lungs regain their normal size and reduces the volume inside the chest cavity, causing the pressure inside increases inversely proportional to the volume, then the pressure difference is reversed in relation to the inspiration and the air is expelled from the lungs to try to equalize the pressures see Fig-2 [2] [6] [7].

2.3 Lung Mechanics

Lung mechanics has studied from a physical point of view, the behavior of the respiratory system when a flow of air inside, and is determined by the set of factors that characterize the movement of gas. As stated above, the effort which must be exerted to produce an inspiration must overcome the total resistance, which is the sum of the resistance offered by the airways and the capacitance of the lungs, see Fig 2 takes as reference that the airway resistance exists only in dynamic conditions, i.e. in the presence of airflow, whereas the elastic resistance offered by the compliance, there are both in static conditions (no air flow) and dynamic [6][7][8][9][10]. For airway resistance or simply resistance (R_c), means the relationship between pressure and flow is determined and directly proportional to the length and inversely proportional to the size of the airway. This accounts for 80% of lung resistance, expressed in $H_2O/l/second$ [2] [7] [8] [9].

The capacitance or dispensability is the relationship between the volume administered in an insufflations and the pressure that has generated in the lungs to the introduce, i.e. the resistance to the lung exercises to air flow to the distended. Therefore, the greater the dispensability, the greater the volume delivered per unit of pressure and with a dispensability diminished, for the same pressure gradient the volume of gas delivered will be much less. As is

variable that depends on volume and pressure, is expressed in $ml/cm H_2O$ [4][5][8][9][10].

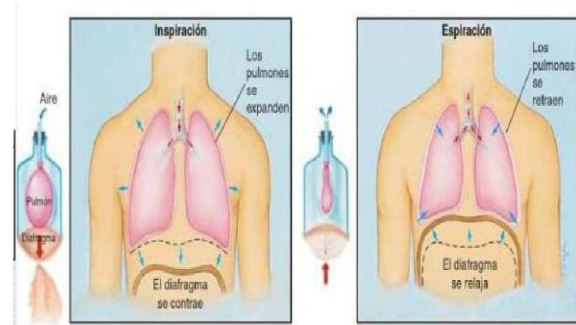


Fig 2 Process of inspiration and expiration of air

2.4 Pathologies of Respiratory System

There are several pathologies that can alter the resistance and capacitance, are classified in obstructive, if interfere in the resistance of the air, such as bronchial asthma, chronic bronchitis, edema of glottis, among others; and restrictive if alter the capacitance, are the ARDS, pulmonary edema, pneumothorax, fibrosis pleural, etc [6][7][9].

2.5 Pathologies Obstructive

There are numerous pathologies respiratory obstructive, deepen in the chronic bronchitis, by recurrent be in our population. The chronic bronchitis is the long-standing inflammation of the bronchi, recognizes clinically as the production chronic of mucus, usually with cough, because the cells goblet increase, producing more mucus than normal, and the cells are insufficient to remove, is contaminated easily with germs and bacteria, giving way to infection, inflammation and narrowing of the channel bronchial, making it difficult, the passage of the air.

2.6 Restrictive conditions

As stated above, under normal conditions, the lung has a tissue elastic that allows you to return to its original size after of distended after an inspiration; but this normal function is disrupted in the emphysema, since much of the fabric has been lost and the lung becomes little elastic, so that it was distended with very easily, i.e. its capacitance rises, making the lung is not empty properly after a inspiration, the time of the difficulties increase and the peak flow is compromised. In the Syndrome of Distress Acute Respiratory, ARDS, pathology consisting of an alteration and acute severe of the structure and lung function secondary to an insult

acute inflammatory that causes pulmonary edema diffuse product of an increase of the permeability of pulmonary capillary. Their clinical features distinctive include a deterioration of the oxygenation, a reduction in the capacitance lung and lung capacity and residual pulmonary infiltrates bilateral contacts in the chest x-ray, appear hours or days after an insult direct lung or an insult systemic[6][7][9]

3. MODEL OF LUNG MECHANICS

Fig 3 shows the electro- mechanical equivalent of the respiratory system. [6][11][12]

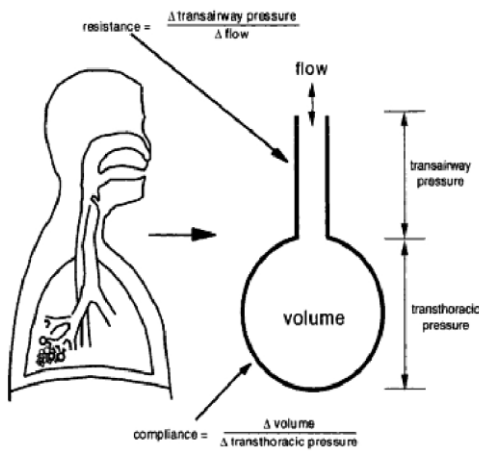


Fig 3 Model of respiratory system

This model has a similar electric considers:

- The opposition of the ducts or airways by where circulates the air to reach the alveoli lung, as an electrical resistance, R , given in $\text{cmH}_2\text{O/L/s}$ or kPa/L/s and is described by the “(1)”.

$$R = \frac{\Delta P}{\Delta F} \dots \dots \dots (1)$$

- The lung, property that allows the lengthening or relaxation of the lung with a pressure, also called capacitance or compliance is considered as a condenser, C , and is given in $\text{L/cmH}_2\text{O}$ or L/KPa or L/KPa , their behavior mathematics is shown in “(2)”.

$$\text{Capacitance} = \frac{\Delta \text{volume}}{\Delta p} \dots \dots \dots (2)$$

The linearised description of lung mechanics is shown in Fig-4[13]. The airways are divided into two categories: the larger or central airways and the

smaller or peripheral airways, with fluid mechanical resistances equal to R_C and R_P respectively. Air that enters the alveoli also produces an expansion of the chest-wall cavity by the same volume. 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 enters the respiratory system is shunted away from the alveoli as a result of compliance of the central airways and gas compressibility. This shunted volume is very small under normal circumstances at regular breathing

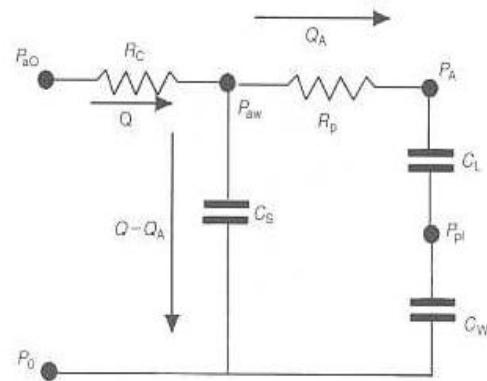


Fig 4 Linear model of lung mechanics

frequencies, but becomes progressively more substantial if disease leads to peripheral airway obstruction or a stiffening of the lungs or chest-wall. We account for this effect by placing a shunt compliance, C_s , in parallel with C_L and C_W . The pressures developed at the different of this lung model are

- P_{ao} at the airway opening
- P_{aw} in the central airways
- P_A the alveoli and
- P_{pi} pleural space.

These pressures are referenced to P_0 , the ambient pressure, which we can set to zero[13]. Suppose the volume flow rate of air entering the respiratory system is Q . Then the objective is to derive a mathematical relationship between P_{ao} and Q . From Kirchhoff’s second law (applied to the node P_{aw}), if the flow delivered to the alveoli is Q_A , then the flow shunted away from the alveoli must be $Q - Q_A$. Applying, Kirchhoff’s First law to the closed circuit containing C_s , R_p , C_L , and C_w we have

$$R_P Q_A + \left(\frac{1}{C_L} + \frac{1}{C_W} \right) \int Q_A dt = \frac{1}{C_S} \int (Q - Q_A) dt \dots \dots \dots (3)$$

Applying Kirchoff's first Law to the circuit containing R_c and C_s , we have

$$P_{ao} = R_c Q + \frac{1}{C_s} \int (Q - Q_A) dt \dots \dots \dots (4)$$

$$Q = P_{ao} - \frac{1}{C_s} \int (Q - Q_A) dt$$

$$Q = \frac{1}{R_c} (P_{ao} - R_p Q_A + (\frac{1}{C_L} + \frac{1}{C_W}) \int Q_A dt) \dots \dots \dots (5)$$

Writing equation at P_{aw} by applying Kirchoff's law

$$P_{aw} = \frac{1}{C_s} \int (Q - Q_A) dt \dots \dots \dots (6)$$

$$Q_A = Q - C_s \frac{dP_{aw}}{dt} \dots \dots \dots (7)$$

$$Q_A = Q - Q1 \quad (Q1 = C_s \frac{dP_{aw}}{dt})$$

4. RESULTS

LabVIEW is a graphical programming environment used by millions of engineers and scientists to develop sophisticated measurement, test, and control systems using intuitive graphical icons and wires that resemble a flowchart.

To observe the behavior of the equations obtained in the study of Lung mechanics have been applied to two case studies, the first for a subject in normal conditions and the second for a subject under pathological conditions, the assumed values for each variable are taken from the references and construct the block diagram of governing equations 5 & 7 in LABVIEW

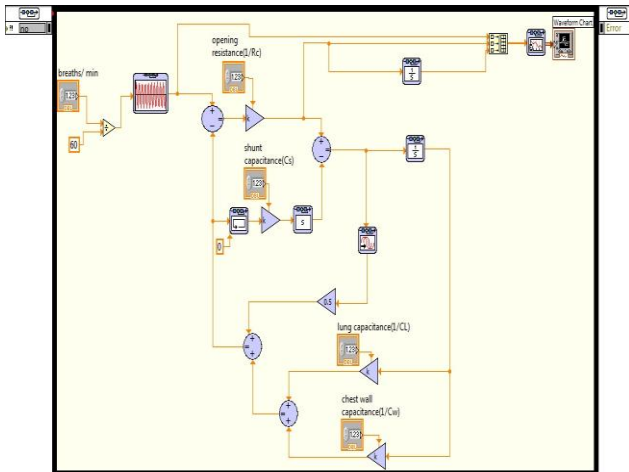
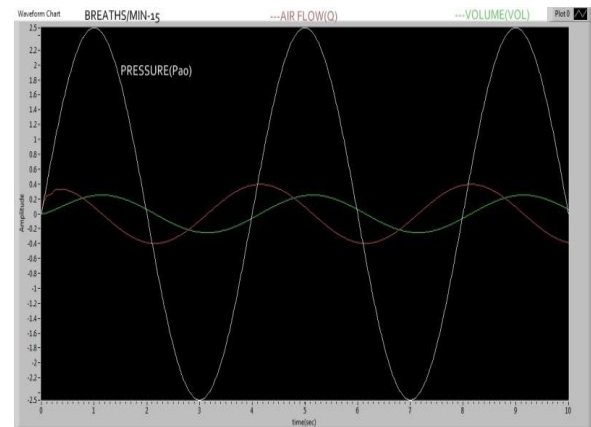


Fig 5 LAB VIEW model of lung mechanics

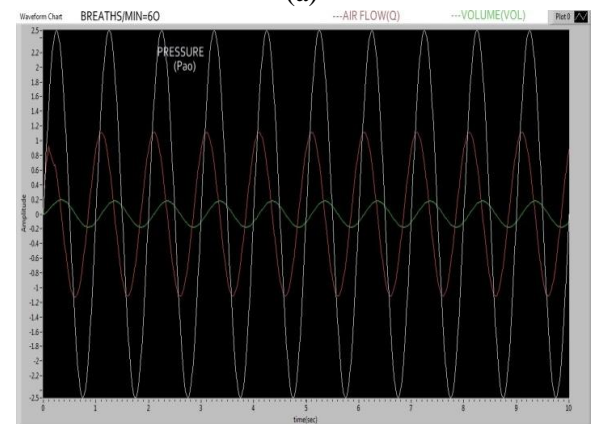
A Model of The Mechanics Applied in Subject Normal

The values for the pressure, the compliance and resistance, according to a study done in healthy adults [15], are established within the following ranges:

- OPENING RESISTANCE ($R_c=1\text{CM of H}_2\text{O SL}^{-1}$)
- PERIPHERAL RESISTANCE ($R_p=0.5\text{CM of H}_2\text{O SL}^{-1}$)
- SHUNTCAPACITACNE ($C_s=0.005\text{LCM of H}_2\text{O}^{-1}$)
- LUNG CAPACITACNE ($C_L=0.2\text{LCM of H}_2\text{O}^{-1}$)
- CHEST WALL CAPACITANCE ($C_W=0.2\text{LCM of H}_2\text{O}^{-1}$)



(a)



(b)

- PRESSURE (Pao)
- VOLUME
- AIRFLOW (Q)

Fig 6 Simulation results of lung mechanics model
(a) Predicted dynamics of airflow(Q) and volume(Vol) in response to sinusoidal forcing Pao (amplitude = 2.5 cm of H2O) at 15 breaths/min.(b) Predicted dynamics of airflow(Q) and volume(Vol) in response to sinusoidal forcing Pao (amplitude = 2.5 cm of H2O) at 60 breaths/min.

Table 1 volume and airflow values at different breaths/min

Fig-6 and Table-1 demonstrates the lung mechanics of normal person for normal breathing at rest approximately 15breaths/min. The peak-to-peak

Breaths/Min	Volume(vol) Peak to peak(Lit)	Airflow(Q) (LitSec ⁻¹)
12	0.5	0.32
15	0.5	0.4
20	0.48	0.5
60	0.4	1.2
90	0.32	1.4
120	0.2	1.55
150	0.16	1.75
180	0.14	1.95

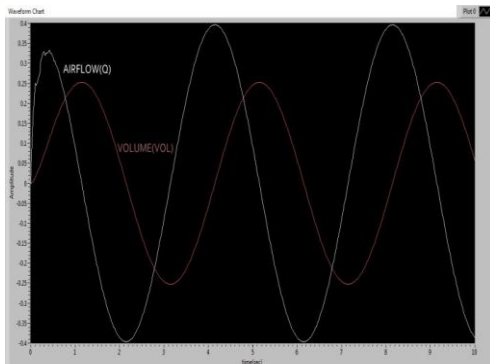
change in volume is 0.5lit, while peak Q is 0.4lit/sec. When the breathing frequency increases to four fold to 60breaths/min with amplitude kept unchanged peak Q clearly rises to 1.2lit/sec, while volume is decreased to 0.4lit. The airflow depends on the intake of pressure, at normal condition pressure inside the lungs is less than atmospheric pressure. As the person goes up(altitude increases) where the atmospheric pressure more than pressure inside the lungs, volume will be less(pressure is inversely proportional to volume) makes the person to take more air. So if the breaths/min increases the airflow increases and volume decreases as height increases. This clearly shown in Fig-6(a) &(b) and the values of Table-1.

B. Model of the Mechanics Applied in Subject with diseased condition

(i)Pathology obstruction

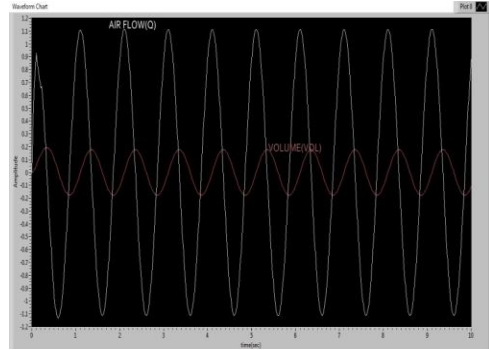
Rc=1

BREATHS/MIN=15



(a)

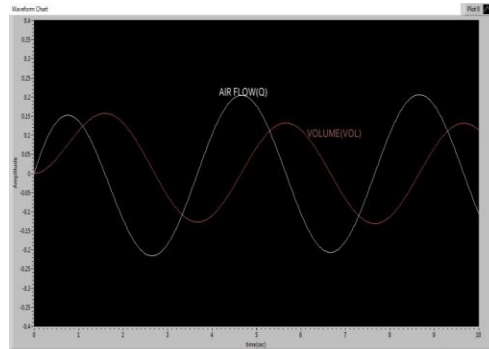
BREATHS/MIN=60



(b)

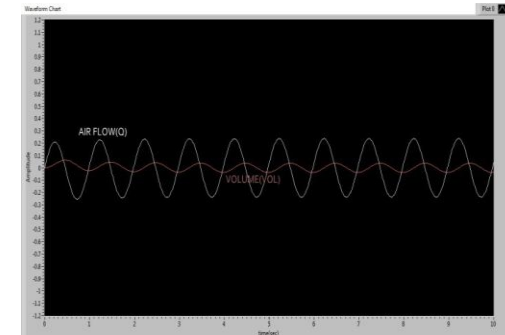
Rc=10

BREATHS/MIN=15



(c)

BREATHS/MIN=60



(d)

■ VOLUME (VOL)
□ AIRFLOW (Q)

Fig 7 Simulation results of lung mechanics model for (C_L, C_w=0.2) .The predicted dynamics of airflow(Q) and Volume for pathological obstructive when (a) Rc=1, 15breaths/min(b) Rc=1, 60breaths/min (c) Rc=10, 15breaths/min (d) Rc=10, 60breaths/min

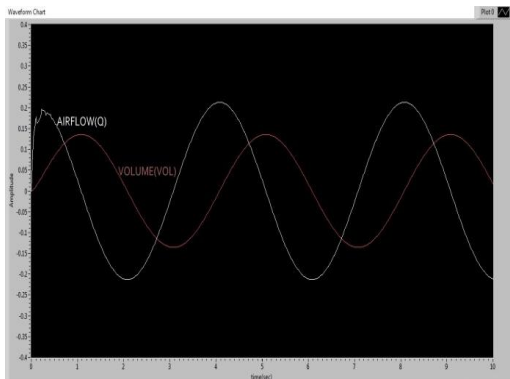
Table 2 Volume and Air flow at different values of R_c when breaths/min equal to (i) 15 (ii) 60

Resistance (R_c) (CM of $H_2O SL^{-1}$)	Volume(VOL)Peak-Peak (Lit)		Air Flow(Q) (Lit/Sec ⁻¹)	
	breaths/min=15	breaths/min=60	breaths/min=15	breaths/min=60
1	0.5	0.4	0.4	1.2
2	0.48	0.27	0.38	0.84
4	0.44	0.16	0.33	0.54
10	0.32	0.08	0.21	0.24
20	0.2	0.05	0.1	0.125
40	0.12	0.03	0.06	0.06

Fig 7 and Table 2 demonstrates the lung mechanics of pathology obstructive, under normal condition $R_c=1$ and 15breaths/min the peak to peak volume is 0.5lit and airflow is 0.4lit/sec. if R_c is increased to 10 due to obstruction a the peak to peak volume is decreased to 0.08 and airflow is 0.21 lit/sec. When a patient have chronic bronchitis, according to the state of art done, the excess mucus produced by the cells makes it closer and obstructs the airway, that is, it increases the value of the resistance of the airways and therefore the air flow and volume diminishes, which is valid in fig- 7. In the Table 2 shows a decrease of volume when R_c is increased indicating the existence of a high difficulty in breathing. Even the if the person tries to take more breaths the airflow is increased but the volume is diminished.

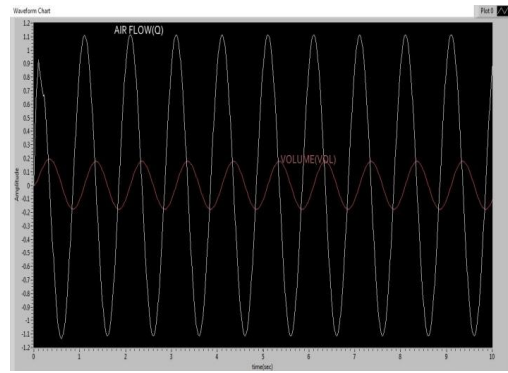
(ii)Pathology restrictive

$C_L, C_w=0.1$
BREATHS/MIN=15



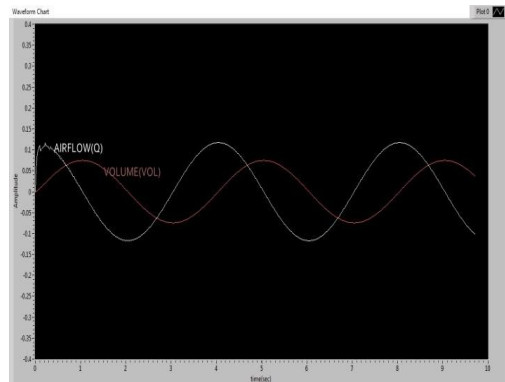
(a)

BREATHS/MIN=60



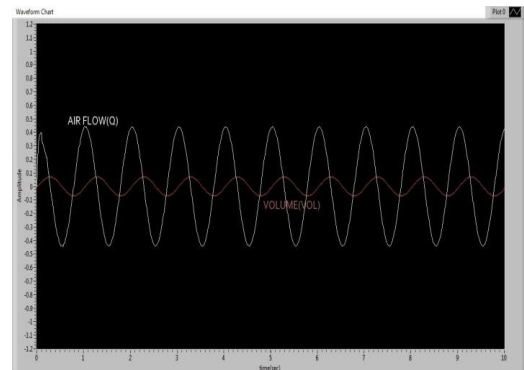
(b)

$C_L, C_w=0.05$
BREATHS/MIN=15



(c)

BREATHS/MIN=60



(d)

█ VOLUME (VOL)
 AIRFLOW (Q)

Fig 8 Simulation results of lung mechanics model for $R_c= 1$. The predicted dynamics of airflow(Q) and Volume for pathological obstructive when (a) $C_L, C_w=0.2, 15$ breaths/min (b) $C_L, C_w=0.2, 60$ breaths/min (c) $C_L, C_w=0.05, 15$ breaths/min. (d) $C_L, C_w=0.05, 60$ breaths/min

Table 3 Volume and Air flow at different values of C_L, C_w when breaths/min equal to (i) 15 (ii) 60

Lung Capacitance (C_L) (LCM of H_2O^{-1})	Chest Wall Capacitance (C_w) (LCM of H_2O^{-1})	Volume(VOL) Peak-Peak (Lit)		Air Flow(Q) (Lit/Sec ⁻¹)	
		breath s/min =15	breath s/min =60	breath s/min =15	breath s/min =60
0.2	0.2	0.5	0.4	0.4	1.2
0.1	0.1	0.27	0.22	0.215	0.72
0.05	0.05	0.14	0.14	0.11	0.44
0.025	0.025	0.08	0.08	0.07	0.26

Fig 8 and table 3 demonstrates the lung mechanics of pathology restrictive, under normal condition $C_L, C_w=0.2$ and 15breaths/min the peak to peak volume is 0.5lit and airflow is 0.4lit/sec. if capacitance is decreased to 0.05the volume is decreased to 0.14lit and airflow is 0.14lit/sec . In the pulmonary emphysema, the capacitance is compromised increased drastically their value, which produces both decrease in the breath flow as in the pressure of the lung, due to the decline of elasticity in the same, and is reflected in the time of exhalation that now takes more than twice as long as inspiration. These symptoms can validate in forms of wave of Fig-8, because by increasing the capacitance the volume decreases. Even though the breaths/min the volume remains unchanged.

5. CONCLUSION

The results obtained in carrying out the simulation of the Lung mechanics model which was obtained, both with normal conditions and with pathological conditions, show that the data and insights in the literature are validated, it has been able to verify that the change in clinical parameters breaths entry, resistance of airways, capacitance depending on the pathology submitted, make the behavior of the system will be affected.

At high frequencies (60breaths/min), the lung mechanics is dominated by resistive effect at such high frequencies. At this point Airflow (Q) has become more in phase with P_{ao} (pressure) while volume displays significant lag. At low frequencies (15breaths/min), the lung mechanics is dominated by capacitance effect at such low frequencies. volume waveform is more in phase with P_{ao} (pressure); the

airflow (Q) shows a substantial phase lead relative to P_{ao} .

The changes in Airflow (Q) and Volume with frequency demonstrate the phenomenon pulmonologists refer to as frequency dependence of pulmonary resistance and capacitance i.e., the lungs appear stiffer and less resistive as frequency increases from resting breathing.

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