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Modeling and Simulation of Physiological Systems Using Labview

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Abstract : *Modeling is needed in the area of medicine to understand the operation of functional systems of the human body. This paper describes modeling and simulation of respiratory system, lung mechanics and breathing of normal and congestive heart failure persons. Modeling of the respiratory system is helpful in finding out the diseases related to lungs. The characteristics of lung mechanics of normal and diseased persons can be evaluated through modeling. The steady state and cheyne stokes breathing models provide required controlled ventilation to the patients. In addition, they can also be used to analyze the stability of normal persons and congestive heart failure persons at high altitudes. All the conceptual models of the above interacting processes with predictions are useful for the development of many improved medical diagnostic techniques.*

Keywords *- Cheyne Stokes Breathing Models, LabVIEW, Lung Mechanics, and Respiratory System*

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I. INTRODUCTION

In human body, the primary function of a respiration system is inhalation of oxygen and exhalation of carbon-dioxide. The respiratory system consists of an upper respiratory tract formed by the nose, mouth, the pharynx, larynx and trachea. With each breath the upper tract leads the air into the lower respiratory tract and the lungs. In the lungs the erythrocytes in the bloodstream captures the oxygen content in the air and distributes it throughout the body. Simultaneously, the carbon dioxide that has been accumulated in the blood due to cellular metabolism is expelled on expiration [1], [2].

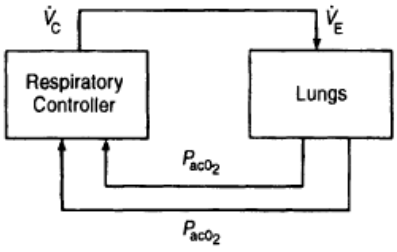
In general, the respiratory control system is modeled as a closed loop feedback/feed forward regulator [3]. Such a model is very useful for diagnosis and prediction of chronic diseases. The model and simulation are also used for controlling the operation of intensive care ventilators and special ventilators in chronic care units. Lung mechanics plays effective role in ventilator support. The lung mechanics are divided in two broad categories i.e., analysis of lung mechanical features with and without airflow. In [4], the flow of air in the way airfreight and in the lungs in normal conditions and its validation in the model implementation is observed.

II. MATHEMATICAL MODELING OF PHYSIOLOGICAL SYSTEMS

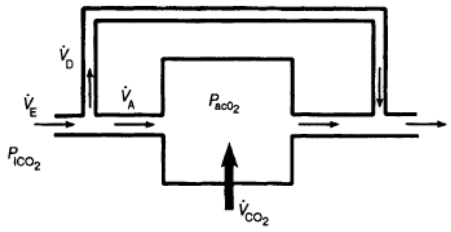
1. Modeling Of Respiratory System

A. Modeling of Gas Exchanger

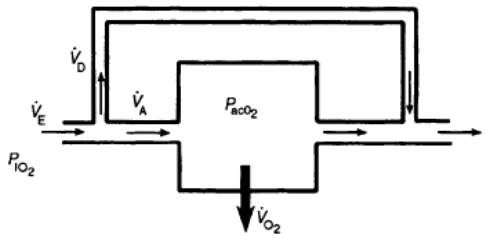
Models of Steady-state chemical regulation of ventilation and gas exchanger are shown in fig.1. The gas exchange occurring in the lungs is modeled using CO_2 and O_2 mass balance equations. Let V_E is the total ventilation, V_A is alveolar ventilation, V_D is dead space ventilation, F_{ICO2} and F_{ACO2} are air entering and leaving the alveoli [5], [6].



(a) Steady-state model of the chemical regulation of ventilation.



(b) Model of steady-state CO_2 exchange in the lungs.



(c) Model of steady-state O_2 exchange in the lungs.

Fig.1 Models of Steady-state chemical regulation of ventilation and gas exchanger

$$V_A = V_E - V_D \tag{1}$$

and the CO_2 mass balance:

$$V_{CO_2} = kV_A\left(F_{ACO_2} - F_{ICO_2}\right) \tag{2}$$

Now the constant ' k ' can be is expressed as

$$k = \frac{P_b - 47}{863} \tag{3}$$

The volumetric fractions F_{ACO_2} , F_{ICO_2} can be represented as partial pressures using Daltons law

$$P_{ICO_2} = F_{ICO_2}\left(P_b - 47\right) \tag{4}$$

$$P_{ACO_2} = F_{ACO_2}\left(P_b - 47\right) \tag{5}$$

From (2), (3), (4), (5) we get

$$P_{ACO_2} = P_{ICO_2} + \frac{863V_c}{V_a} \tag{6}$$

Similarly developing mass balance equation for O_2

$$P_{AO_2} = P_{IO_2} - \frac{863V_c}{V_a} \tag{7}$$

Here P_{AO2} is partial pressure of alveolar o_2 and P_{IO2} is partial pressure of inhaled o_2 content and P_{ACO2} is partial pressure of alveolar co_2 and P_{ICO2} is partial pressure of inhaled co_2 content. The relation between P_{ACO2} and V_A is hyperbolic and this is referred as metabolic hyperbola

B. Modeling of Respiratory Controller

The chemo receptors are involved in the controller part of the system. The lower brain neuronal circuits are involved in the respiratory rhythm generation. A strong interaction between CO_2 and O_2 is involved in the controller operation. The ventilator controller output V_c is represented as sum of the O_2 independent term and a term in which there is a multiplicative interaction between hypoxia and hypercapnia.

$$V_c = \begin{cases} \left(1.46 + \dfrac{32}{P_{aO_2} - 38.6}\right)\left(P_{aCO_2} - 37\right) & P_{aCO_2} > 37 \\ 0 & P_{aCO_2} \le 37 \end{cases} \tag{8}$$

2. MODELING OF LUNGS MECHANICS

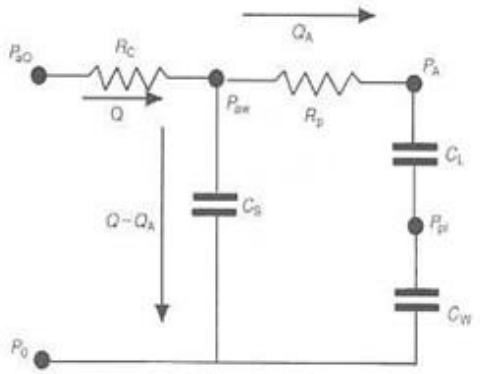


Fig.2 Linear model of lung mechanics

The lung mechanics linear model in electrical view point shown in fig.2. The airways are divided into the central airways and the peripheral airways, with resistances R_C and R_P respectively. An expansion of the chest-wall cavity is represented by the series connection of the lung ($C_{L)\ and}$ chest wall (C_W) compliances. However, a small fraction of the volume of air enters the respiratory system is shunted away from the alveoli because of compliance of the central airways and gas compressibility. 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 or a stiffening of the lungs or chest-wall. The decease effect is represented as shunt compliance, C_S as shown in fig.4[7], [8], [9] .The pressures at different points in lung model are P_{ao}(at the airway opening), P_{aw} (in the central airways), P_A (at the alveoli), P_{P1} (at the pleural space) and P_0(ambient pressure set to zero).Suppose the volume flow rate of air entering the respiratory system is Q. Applying, Kirchhoff's First law to the closed circuit, the following modeling equations are derived[10].

$$R_P Q_A + \left(\frac{1}{C_L} + \frac{1}{C_W}\right)\int Q_A dt = \frac{1}{C_S}\int (Q - Q_A)\,dt \tag{9}$$

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

$$P_{ao} = R_c Q + \frac{1}{C_S}\int (Q - Q_A)\,dt \tag{10}$$

$$Q = P_{ao} - \frac{1}{C_S}\int (Q - Q_A)\,dt \tag{11}$$

$$Q = \frac{1}{R_c}\left(P_{ao} - R_P Q_A + \left(\frac{1}{C_L} + \frac{1}{C_W}\right)\int Q_A dt\right) \tag{12}$$

Writing equation at Paw by applying Kirchhoff's law

$$P_{aw} = \frac{1}{C_S}\int (Q - Q_A)\,dt \tag{13}$$

$$Q_A = Q - C_s \frac{dP_{aw}}{dt} \tag{14}$$

$$Q_A = Q - Q1 \qquad \left(Q1 = C_s \frac{dP_{aw}}{dt}\right) \tag{15}$$

3. MODELING OF CHEYNE –STOKES BREATHING

In chronic heart failure patient's diagnosis one of the important feature is periodic breathing. Periodic breathing is apnea separated clusters of breaths. Cheyne stokes respiration is an abnormal breathing pattern.

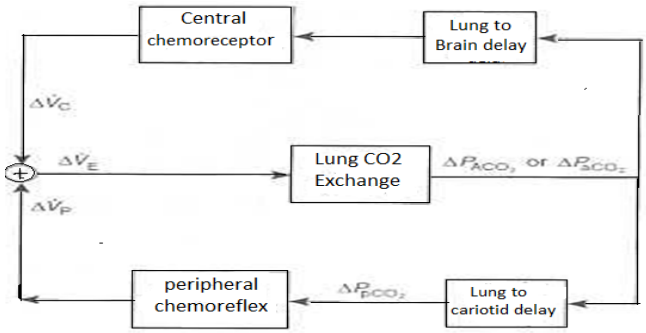


Fig.3: Respiratory control system including transportation delays

Two feedback loops, one from central chemo reflex and the other one from peripheral chemo reflex are considered for this model. First modeling of the system is developed without transport delays and finally added transport delays and taken the loop transfer function for stability analysis. A simplified schematic is shown in fig.3 which consists of delays taken to transport blood from the lungs and chemo receptors [11].

A.MODELING OF THE LUNGS

The dynamic equivalent of the gas exchange equation is given below

$$V_L\left(dP_{ACO_2}/dt\right)=\left(V_E-V_D\right)\left(P_{ICO_2}-P_{ACO_2}\right)+863Q\left(C_{VCO_2}-C_{aCO_2}\right) \quad (16)$$

Where

Q = Pulmonary blood flow

V_L = Effective CO_2 storage capacity of the lungs

C_{VCO2} = Concentrations in arterial and

CaCO = Mixed venous blood

$$V_L=\left(d\left(\Delta P_{ACO_2}\right)/dt\right)=\left(V_E-V_D\right)\Delta P_{ACO_2}+\left(P_{ICO_2}-P_{ACO_2}\right)\Delta V_E-863Q\Delta C_{aCO_2} \quad (17)$$

$$V_L\left(d\left(\Delta P_{ACO_2}\right)/dt\right)+\left(V_E-V_D+863QK_{CO_2}\right)\Delta P_{ACO_2}=\left(P_{ICO_2}-P_{ACO2}\right)\Delta V_E \quad (18)$$

Taking Laplace Transforms

$$H_L(s)=\left(\Delta P_{ACO_2}/\Delta V_E\right)=\left(-G_L/\left(T_Ls+1\right)\right) \quad (19)$$

Where

$$T_L=V_{lung}/\left(V_E-V_D+863QK_{CO_2}\right) \quad (20)$$

$$G_L=\left(P_{ACO_2}-P_{ICO_2}\right)/\left(V_EV_D+863QK_{CO_2}\right) \quad (21)$$

B.TRANSPORT DELAYS

It is assumed that pulmonary end-capillary blood returning to the heart will take some time (T_p) to arrive at the peripheral chemoreceptor's and a longer time (T_c>T_p). Thus,

$$\Delta P_{pCO2}(t)=\Delta P_{ACO_2}\left(t-T_p\right) \quad (22)$$

$$\Delta P_{cCO2}(t)=\Delta P_{ACO_2}\left(t-T_c\right) \quad (23)$$

Applying Laplace to the Above Equations,

We get, $$\Delta P_{pCO_2}(s)=e^{-sTp}\Delta P_{ACO_2}(s) \quad (24)$$

$$\Delta P_{cCO_2}(s)=e^{-sTp}\Delta P_{ACO_2}(s) \quad (25)$$

Dynamic response of peripheral and central chemo receptors are

$$T_p\left(dV_p/dt\right)+V_P=G_p\left[P_{pCO_2}-I_p\right] \quad (26)$$

$$T_c\left(dV_c/dt\right)+V_c=G_c\left[P_{cCO_2}-I_c\right] \quad (27)$$

The loop transfer functions of Lung mechanics with transportation delays are

$$H_{Lp}(s)=\left(\Delta V_p(s)/\Delta V_E(s)\right)=\left(G_{lung}G_p.e^{-sTp}\right)/\left(T_Ls+1\right)\left(T_ps+1\right) \quad (28)$$

$$H_{Lc}(s)=\left(\Delta V_c(s)/\Delta V_E(s)\right)=\left(G_{lung}G_p.e^{-sTp}\right)/\left(T_Ls+1\right)\ \left(T_cs+1\right) \quad (29)$$

Overall Frequency Response of the Loop Transfer Function is

$$H_L(\omega)=(\Delta V_p(\omega)+\Delta V_c(\omega))/(\Delta V_E(\omega)) \quad (30)$$

$$H_L(\omega)=(G_L/(1+j\omega T_L))((G_p.e^{-j\omega Tp}/1+j\omega T_p)+(G_c.e^{-j\omega Tp}/1+j\omega T_c) \quad (31)$$

The Stability analysis of lung mechanics for normal and congestive heart failure persons are tested by applying Nyquist stability Criterion.

III. SIMULATION RESULTS OF PHYSIOLOGICAL SYSTEMS

The modeling equations of respiratory system, lung mechanics, breathing systems are simulated using LabVIEW.

A. RESPIRATORY SYSTEM

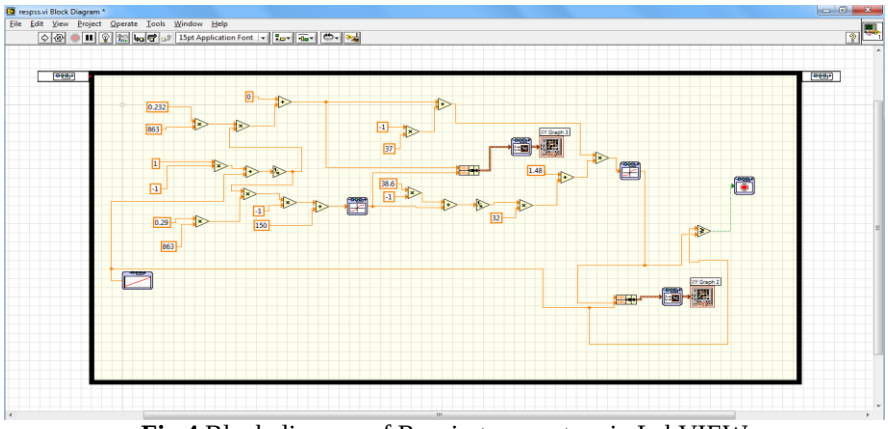


Fig.4 Block diagram of Respiratory system in LabVIEW

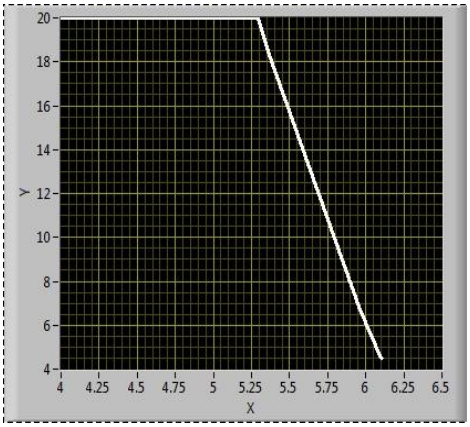


Fig.5 V_E in vs $V_E$out in case (1)

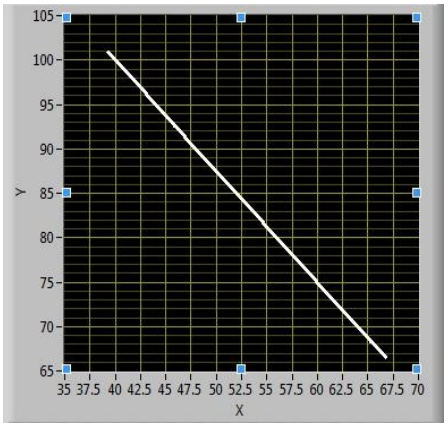


Fig.6 P_{aco2} vs P_{ao2} in case (1)

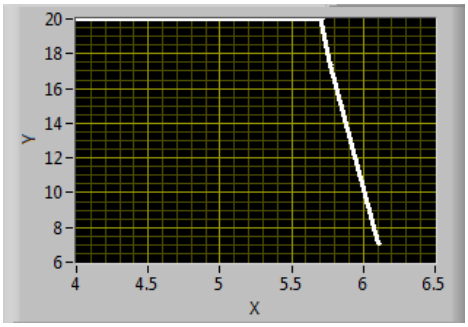


Fig.7 V_E in vs $V_E$out in case (2)

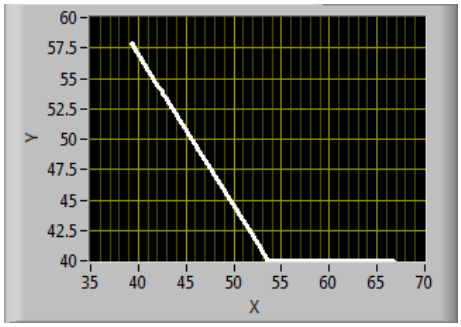


Fig.8 P_{aco2} vs P_{ao2} in case (2)

Case1: The person is at ground level

In case1, P_{IO2} = 150mm of Hg i.e. 21% of room air and P_{ICO2} = 0. The partial pressures of O2 and CO2 are affected by value of V_E. At low value of V_E, the P_{AO2} is 65 mm of Hg and P_{ACO2} is 67 mm of Hg. The simulation is terminated when V_E=6 L/min then P_{ACO2}=40 mm of Hg and P_{AO2}=100 mm of Hg. The values are tabulated in table 1 and table 2. Based on V_E value P_{ACO2} and P_{AO2} are affected. The controller output at normal sea level shown in fig.5. and fig.6.

Case 2: The person is at high altitude (8500ft)

In case2, P_{IO2} = 107mm of Hg i.e.15% of room air, while P_{ICO2} == 0 [10]. Initially P_{AO2} is 40 mm of Hg and P_{ACO2} is 67mm of Hg at low value of V_E. PAO2, is not allowed to fall below 40, due to the saturation effect. The simulation is terminated when V_E=6.1 L/min then P_{ACO2}=39 mm of Hg and P_{AO2}=58.2 mm of Hg. The output of a controller in high altitude (8500 ft) is shown in fig.7 and fig.8. The P_{ACO2} and P_{AO2} for V_E case 1 and case 2 are tabulated in table 3 and table 4.

Table.1 P_{ACO2} vs P_{AO2} in case 1

P_{ACO2} in mm of Hg	P_{AO2} in mm of Hg
67	65
60	70
50	80
40	100

Table.2 V_E in vs V_E out in case 1

V_E in	V_E out
5.1	20
5.4	15
5.8	10
6	6

Table.3 P_{AO2} vs P_{ACO2} in case 2

P_{AO2} in mm of Hg	P_{ACO2} in mm of Hg
40	67
40	60
45	52
58.2	39

Table.4 V_E in vs V_E out in case 2

V_E in	V_E out
5.1	20
5.4	15
5.8	10
6.1	6.1

B. LUNG MECHANICS

The model of lung mechanics is implemented in LabVIEW as shown in fig.9. The front panel of LabVIEW displays the simulation results for three cases is shown in fig.10 to fig.12

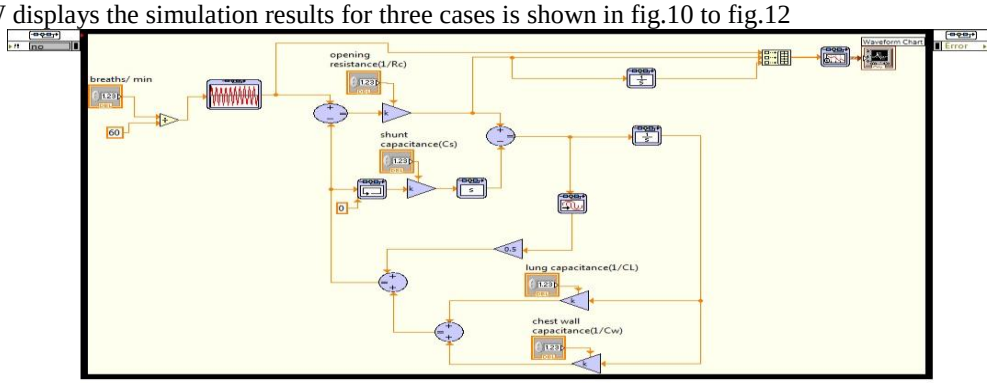
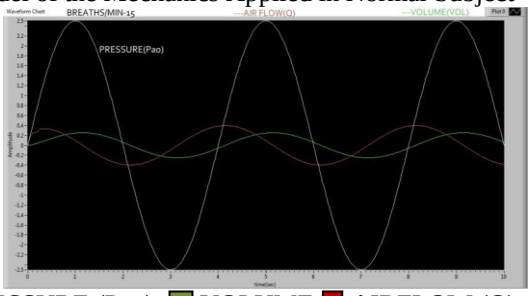
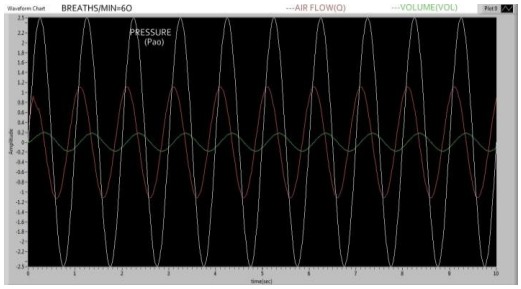


Fig.9 Block diagram of Lung Mechanics in LabVIEW

A Model of the Mechanics Applied in Normal Subject





☐ PRESSURE (Pao) ■ VOLUME ■ AIRFLOW (Q)

Fig.10(a) **Fig.10(b)**

Fig.10 Simulation results of lung mechanics model

Fig.10. and table 5 demonstrate the lung mechanics of normal person. A normal person at rest takes 15breaths/min. The peak-to-peak change in volume is 0.5Lt, while peak Q is 0.4lit/sec. When the breathing frequency increases to four-fold i.e. to 60breaths/min with amplitude kept unchanged peak Q rises to 1.2Lt/sec, while volume is decreased to 0.4Lt. So, if the breaths/min increases the airflow increases and volume decreases as height increases. This clearly shown in fig.10 and the values are tabulated in table 5. Predicted dynamics of airflow (Q) and volume (Vol) in response to sinusoidal forcing Pao (amplitude = 2.5 cm of H2O) at 15 breaths/min. and at 60 breaths/min are illustrated in fig.10a and fig.10b respectively.

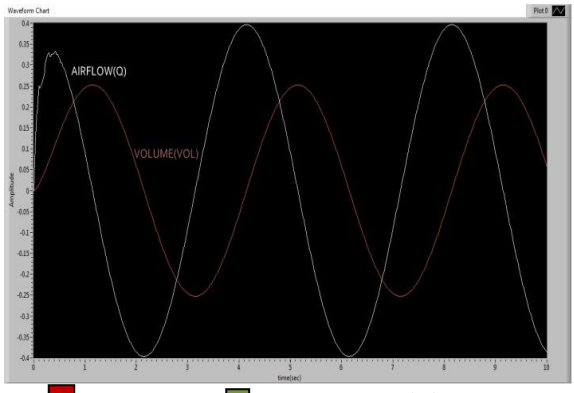
Table.5 Volume and airflow values at different breaths/min

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

B. Model of the Mechanics Applied in Pathological Subject

Pathology obstruction

BREATHS/MIN=15



VOLUME AIRFLOW (Q)

Fig. 11(a)

BREATHS/MIN=60

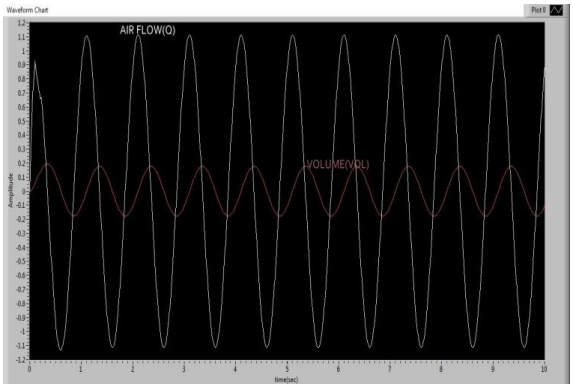


Fig. 11(b)

BREATHS/MIN=15

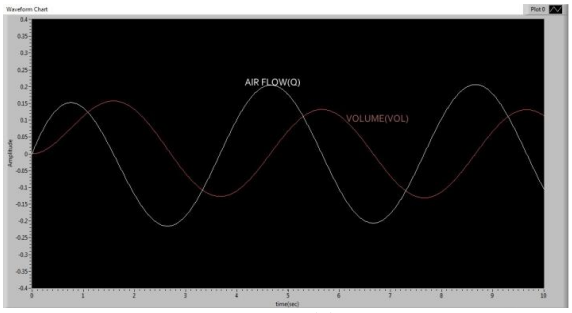


Fig. 11(c)

BREATHS/MIN=60

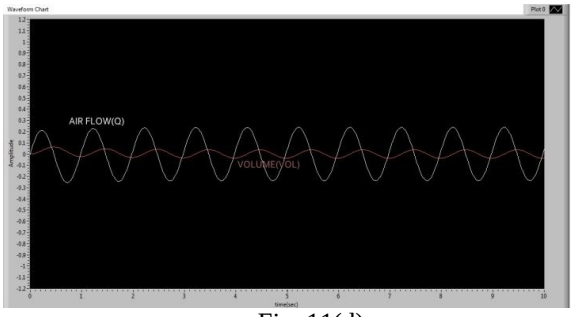


Fig. 11(d)

Fig.11 Simulation results of lung mechanics model

Fig.11 and table 6 demonstrates the lung mechanics of pathology obstructive patient.

In normal case Rc=1 and breathing frequency=15breaths/min. The peak to peak volume is 0.5lit and airflow is 0.4lit/sec. If Rc is increased to 10, due to obstruction the peak to peak volume is decreased to 0.08 and airflow is 0.21 lit/sec. When a patient has chronic bronchitis, 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 diminish as shown in fig.11. and table 6. Even the if the person tries to take more breaths the airflow is increased but the volume is diminished.

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 are illustrated through fig.11(a) to 11(d).

Table.6 Volume and Air flow at variable R_c when breaths/min equal to (i) 15 (ii) 60

Resistance (R_c) (CM of H_2O SL^{-1})	Volume (Vol) Peak-Peak (Lt)		Air flow (Q) (Lt/sec)	
	Breaths/Min=15	Breaths/Min=60	Breaths/Min=15	Breaths/Min=60
1	0.50	0.40	0.40	1.20
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	0.20	0.05	0.10	0.12
40	0.12	0.03	0.06	0.06

C. CHEYNE- STOKES BREATHING MODEL

Cheyne- stokes breathing model is implemented based on steady state value of P_{ACO2} obtained from steady state model and the parameters of respiratory system (shown in table 7) for normal and congestive heart failure persons.

Table.7 Parameter values used in Cheyne-stokes breathing Model.

Parameter	Value
V_L	2.5L
K_{CO2}	$0.0065mmHg^{-1}$
G_p	$0.02Ls^{-1}mmHg$
G_c	$0.04Ls^{-1}mmHg^{-1}$
T_p	20sec
T_c	120sec
V_E	$0.12Ls^{-1}$
V_D	$0.03Ls^{-1}$
T_p	6.1sec

Nyquist plots for normal and congestive heart failure persons at high altitudes based on Cheyne-stokes breathing model are shown in figures (12) and (13).

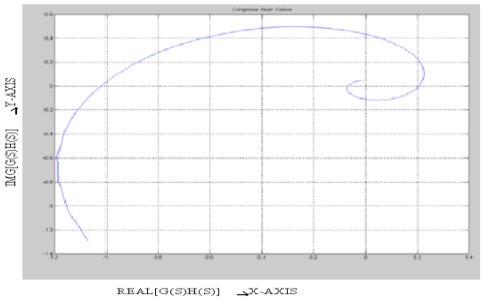


Fig.12 Normal Person

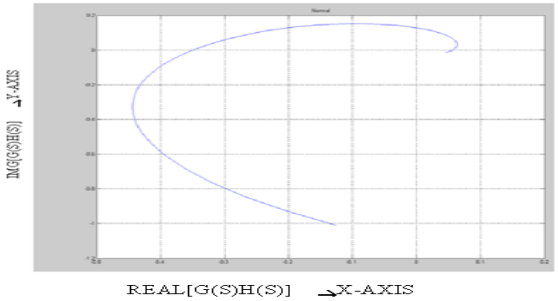


Fig.13 Congestive heart failure Person

The Nyquist plot shown in figures 12 and 13 represent a bandwidth of 0.01 to 0.1 Hz i.e. inter breath periodicities of cycle duration 10 Sec to 100 Sec. In the case of normal person, shown in fig.12, respiratory system is stable with $G_{C\,=}$ 0.34 which occurred at f=0.0295 Hz and having a periodicity of 34 Sec. In the case of congestive heart failure person, the respiratory system is less stable and enters unstable region if the frequency oscillations are more than critical frequency and the loop gain is 1.02.These oscillations are consistent throughout the total cycle duration for normal and congestive heart failure case.

IV. CONCLUSION

The mathematical modeling of physiological systems i.e. respiratory system, lung mechanics and breathing of normal and congestive heart failure persons are developed and simulated using LabVIEW. The simulation results of respiratory system are used to design respiratory controller which controls the inhalation of oxygen of the person based on location and reduce the wastage of oxygen compared to manual control. This can be further extended to medical application such as designing an automatic ventilator. Lung mechanics models with normal and pathological conditions are simulated and observed that lung mechanics is dominated by resistive effect at high frequencies (60 breaths/min.) and by capacitance effect at low frequencies (15breaths/min.).

Cheyne-stokes breathing model demonstrates the stability of the respiratory system for normal and congestive heart failure persons at high altitudes. The stability analysis gives prior information about the critical breathing frequencies of patients which is helpful in various diagnoses related to respiratory system. In summary, these simulation models can contribute in the current research on physiological systems.

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